This is a digital copy of a book that was preserved for generations on library shelves before it was carefully scanned by Google as part of a project to make the world's books discoverable online.

It has survived long enough for the copyright to expire and the book to enter the public domain. A public domain book is one that was never subject to copyright or whose legal copyright term has expired. Whether a book is in the public domain may vary country to country. Public domain books are our gateways to the past, representing a wealth of history, culture and knowledge that's often difficult to discover.

Marks, notations and other marginalia present in the original volume will appear in this file - a reminder of this book's long journey from the publisher to a library and finally to you.

Usage guidelines

Google is proud to partner with libraries to digitize public domain materials and make them widely accessible. Public domain books belong to the public and we are merely their custodians. Nevertheless, this work is expensive, so in order to keep providing this resource, we have taken steps to prevent abuse by commercial parties, including placing technical restrictions on automated querying.

We also ask that you:

+ *Make non-commercial use of the files* We designed Google Book Search for use by individuals, and we request that you use these files for personal, non-commercial purposes.

+ *Refrain from automated querying* Do not send automated queries of any sort to Google's system: If you are conducting research on machine translation, optical character recognition or other areas where access to a large amount of text is helpful, please contact us. We encourage the use of public domain materials for these purposes and may be able to help.

+ *Maintain attribution* The Google "watermark" you see on each file is essential for informing people about this project and helping them find additional materials through Google Book Search. Please do not remove it.

+ *Keep it legal* Whatever your use, remember that you are responsible for ensuring that what you are doing is legal. Do not assume that just because we believe a book is in the public domain for users in the United States, that the work is also in the public domain for users in other countries. Whether a book is still in copyright varies from country to country, and we can't offer guidance on whether any specific use of any specific book is allowed. Please do not assume that a book's appearance in Google Book Search means it can be used in any manner anywhere in the world. Copyright infringement liability can be quite severe.

About Google Book Search

Google's mission is to organize the world's information and to make it universally accessible and useful. Google Book Search helps readers discover the world's books while helping authors and publishers reach new audiences. You can search through the full text of this book on the web at `http://books.google.com/`

LANE MEDICAL LIBRARY STANFORD
L73 .L652s 1904 STOR
Medical diagnosis : special diagnosis of

LANE

MEDICAL LIBRARY

GIFT OF DR.J.L.GAMBLE.

James L. Gamble
Sept 2. 1909

MEDICAL DIAGNOSIS

MEDICAL DIAGNOSIS

SPECIAL DIAGNOSIS OF INTERNAL MEDICINE

A HANDBOOK FOR PHYSICIANS AND STUDENTS

BY

DR. WILHELM v. LEUBE

PROFESSOR OF MEDICINE, AND PHYSICIAN-IN-CHIEF TO THE JULIUS HOSPITAL AT WÜRZBURG

AUTHORIZED TRANSLATION FROM
THE SIXTH GERMAN EDITION

EDITED, WITH ANNOTATIONS, BY

JULIUS L. SALINGER, M.D.

LATE ASSISTANT PROFESSOR OF CLINICAL MEDICINE IN THE JEFFERSON MEDICAL COLLEGE, AND PHYSICIAN TO THE PHILADELPHIA HOSPITAL

WITH FIVE COLOURED PLATES AND SEVENTY-FOUR ILLUSTRATIONS IN THE TEXT

LANE LIBRARY

NEW YORK AND LONDON
D. APPLETON AND COMPANY
1904

COPYRIGHT, 1904, BY
D. APPLETON AND COMPANY

PRINTED AT THE APPLETON PRESS
NEW YORK, U. S. A.

LANE LIBRARY

L73
L652s
1904

AUTHOR'S PREFACE

ABOUT a year ago Dr. Salinger asked my permission to translate my Special Diagnosis of Internal Diseases into English. I have gladly complied with this request because I hope thus to make my work accessible to greater numbers of English-speaking colleagues than has been possible so far. I would be very glad, indeed, if the book in the English translation will find as numerous friends and patrons in America and England as has been the case in Germany, and if it contributes to stimulate the pleasure in the development of our science and the appreciation of diagnostic art.

DR. LEUBE.

WÜRZBURG.

44048

EDITOR'S PREFACE

WHEN a student of medicine in Germany, the Diagnosis of Leube always impressed itself upon the editor as a plain, practical work which described disease and the various differential points in such a lucid manner that it has always been his wish to give to the English-reading profession the advantages of this storehouse of bedside knowledge. The fact that a medical work has appeared in its sixth edition in the course of twelve years is sufficient evidence of its value. The main features of this text-book consist in the explicit differential diagnoses. If it is true that rational treatment depends upon the accuracy of diagnosis, the fundamental requirements of the therapist will be diagnosis in its broadest sense; not only the ability to recognise the true nature of the affection, but the knowledge of its course, complications and eventual termination. The author has been signally successful in presenting some of the most difficult problems of the diagnosis of Internal Diseases in a clear, concise manner, never attempting the impossible.

The work has been brought thoroughly up to date, but it has been necessary occasionally to add some few descriptions, etc.; these have been inclosed in brackets so as to distinguish them from the original text.

J. L. S.

PHILADELPHIA.

INTRODUCTION

THE object of "diagnosis" is the recognition of disease, combined with a careful consideration of the reasons which in a special case appear to be in favour of the existence of a given malady; it also has for its purpose the determination of the particular variety of the disease. Whereas in the dawn of practical medicine the therapeutic aspect was cultivated alone, and the choice of the particular remedy was influenced either by tradition, or was arbitrarily or intuitively administered, in the course of time, only gradually at first, the necessity for diagnosis awakened. The theoretical study of disease became more and more a necessity, because the empiric therapist could not fail to observe that he owed all his skill and expert knowledge of disease to bedside experience. Soon it was no longer the natural sense of self-preservation alone, nor mere sympathy for an unfortunate, suffering fellow-being—humanity in its broadest sense—which caused some to devote themselves to the actual study of disease. It was rather an incentive to certain persons gifted in a special direction, whether priests, who in ancient times were almost the only practitioners of medicine, or others, to observe, to study, to analyze more objectively the natural course of disease. The observation of a great number of patients naturally showed that a certain category presented analogies in the picture of the affection which were absent in other groups; that some symptoms indicated a favourable, others an unfavourable course; that certain remedies were of advantage in some pathological conditions, but without effect, and even harmful, in others. Thus the first important step was taken, not only towards the establishment of an individual medical profession, but furthermore towards the development of *scientific* medicine as a whole and medical *diagnosis*.

The rapid and positive perception of a morbid process implies an exact knowledge of disease in general, and a complete insight into the probable outcome in a given case; this is gradually acquired by the study of special pathology and by bedside experience, gained by one physician slowly, by another more rapidly, not only by the extent of his practice, but, above all, by his acuteness and conscientiousness of observation, and by his ability to differentiate between the essential and the non-essential at the bedside, and the correct application of his findings to his former experiences. The latter faculty, diagnosis in its restricted sense, may to a great extent be acquired by faithful study, but to a certain degree it is the result of an innate talent, which, however, even in its most developed form, may go astray if not supported by profound knowledge and a conscientiously perfected technique.

The only correct method to arrive at an opinion regarding the variety of an affection in a given case is the method applied by naturalists, which has come in vogue since the time of Francis Bacon, the application of *induction*—that is, to determine the individual morbid phenomena and the derivation of the diagnosis from the *concrete* facts. This analytic method in diagnosis is to be preferred to any other, especially to the *deductive* method, as it has the advantage of greater certainty. It is true, and this will be seen later, that, at times, synthetic conclusions are indispensable in arriving at a diagnosis. Thus it is occasionally of advantage, if it is impossible to establish the entire symptom-complex, to select but a few symptoms which are easily recognised, by their aid gain a general aspect of the condition, and then more systematically to investigate and examine in detail. This deductive method, however, should only be resorted to exceptionally, and then with the greatest care; the firm foundation from which the diagnosis must issue is, and remains, in my experience, the *analytic, inductive* method.

The analysis of morbid phenomena, upon which the correct interpretation of the component elements of the affection depends, must be thoroughly appreciated by the examining physician at the bedside; he must have studied and frequently practised the various methods of examination. Only after he has completely mastered the technique of these various methods will he be able in a given case to establish for himself a solid basis upon which it will be possible to build the special diagnosis. To mention but one instance, not every one, no matter how carefully he scrutinizes his patient, will be able at once to observe in what respect even the external appearance deviates from that of health; to differentiate the essential from the unessential it is necessary that the eye of the physician should be clinically trained. These fundamental principles have been extensively formulated in our time by clinical instruction. Accordingly, the range of examination by clinical methods has steadily grown, and, by means of this, accuracy in diagnosis has been enormously advanced. The refinements of diagnosis went hand in hand with every appreciable improvement and extension of the physical and chemical methods of examination.

It is not my purpose to discuss these various methods of examination individually. Both a knowledge and a practical experience of the same I take for granted; the object of this book is the application of their results in arriving at a diagnosis in a special case. But, before entering upon the subject of special diagnosis, a general sketch of the routine of examination of a patient at the bedside will be given, such as should precede every diagnosis.

The first duty of the physician is to obtain a reliable history (anamnesis) of the patient. To be complete without involving too great a loss of time, a definite plan should be observed, which, in my experience, should contain the following data:

1. Age, occupation, residence, family history.

2. Previous illness; beginning of the present attack, interruption or continuation of the same, the cause ascribed by the patient.

3. Symptoms of the affection, condition of the various organs and their function.

***a. Thoracic Organs.*—Dyspnœa, cough, expectoration, chest pains, cardiac palpitation.**

b. Abdominal Organs.—Tympanites, abdominal pain, condition of the appetite, eructation, nausea, vomiting, appearance of the vomited material, jaundice; condition and appearance of the stool (mucus, blood), hæmorrhoids; urination (quantity, appearance).

c. Nervous System.—Headache, vertigo, sleep, weakness, pains in the extremities, etc.

d. Condition of strength, loss of weight, fever.

e. In the Female.—Menstrual condition, the preceding puerperia.

History-taking, according to this formula, requires but little time, and is quite sufficient in all cases to proceed at once to an examination of the patient with the data at hand. Should the history show certain organs to be the seat of the affection, the anamnesis must be more complete. Do not allow yourself to be detained too long with the story of the patient, but ask to have your questions answered as nearly as possible in the order indicated above. The result thus obtained is much more satisfactory as regards the diagnosis than the patient's most detailed account of his symptoms.

The physical examination, which should then follow, is also best accomplished according to a fixed scheme. With but few exceptions (peritonitis), *all* organs are to be inspected according to the methods of physical diagnosis, at least the first time. Only in this manner may gross errors in diagnosis be avoided; refinements of diagnosis always require repeated extensive examinations of the various organs.

The routine is as follows: Determine the structure of the body, its nutrition, the colour of the skin and mucous membranes, the presence of eruptions, œdema, and glandular enlargement, the possible presence of jaundice. Inspection of the oral cavity and pharynx is absolutely necessary in every febrile condition (especially in children).

Inspection of the *naked* chest. Diagnoses based upon examination of the lungs with a completely or partly covered thorax are absolutely unreliable. *Inspection* of the chest and thoracic movements; *percussion* of the apices; it is best for the physician to stand behind the back of the patient; in bedridden patients, who are still able to sit up, the patient should be made to turn in such a manner that the posterior part of his chest is as much as possible in front of the examining physician; with patients able to be about, they are made to sit upon a chair, the back of which is turned to one side. Percussion of the remainder of the thorax; test for displacement of the border of the lungs in inspiration and for the disappearance of an eventual dulness at the base with change of position. Auscultation: It is best to have the patient take short and deep breaths; should there be impairment of resonance, absent breath sounds or bronchial breathing, bronchophony and vocal fremitus are to be tested.

Examination of the *heart*: Inspection of the præcordial area, especially in regard to the position and character of the apex beat, palpation of the cardiac area with the flat of the hand, demonstrating the character of the apex beat, the presence of a possible thrill or friction, percussion of the borders of the heart, auscultation of the heart and great vessels, if possible as well in the recumbent as in the erect position of the patient, on account of the fact that murmurs are occasionally more pronounced in one position than in the other; examination of the pulse.

Examination of the *abdomen*: Percussion of the *liver*; always attempting palpation of the lower border, the latter can generally be defined even with a soft consistence of the liver, bounded by the intestine. Palpation of the *spleen*; the physician stands to the right of the patient, makes him assume the right half-lateral position and have him firmly rest the head; then, holding with your right hand, which is placed *flat* upon the abdomen, below the left costal arch, and with the left hand, placed in the left axillary line, grasp the inferior sternal ribs, pressing them towards the right hand, which is palpating the spleen. This organ, if enlarged, can be felt, without exception, on deep inspiration; this result is much more reliable than that obtained by percussion, which procedure, however, must always follow to make the examination more thorough.

Inspection of the *gastric region* and the remaining parts of the *abdomen*: Evidence of circumscribed tenderness and possible tumours by palpation; percussion of the gas-

tric area, with the patient in the sitting posture or, still better, while standing, to determine for the present the position of the lower border of the stomach from the boundaries of dulness, by comparison with the results of percussion obtained with the patient in the recumbent position. Should changes in the size of the stomach be noted by this method, this must be followed by the introduction of the stomach tube, allowing the water to flow in and out. Inspection, percussion and palpation of the intestinal region, observation of eventual intestinal movements, palpation of fæcal masses or possibility of tumours, comparison of the results of percussion of lateral dulness on change of position, with consideration of the question of the presence or absence of fluid in the peritoneal sac.

Analysis of the *urine* for albumin and sugar should always be made, even if there is not the slightest suspicion of a renal or constitutional disease. The examination often yields surprising results, gives a certain direction to the diagnosis and, on the other hand, takes but little more than a minute's time. I regard the neglect of an analysis of the urine as a grave error in the systematic examination of patients.

If there is the slightest suspicion of an affection of the nervous system, the urine analysis is to be preceded by an examination for disturbances of motility, sensibility, reflexes, etc.; upon changes of the voice an examination of the larynx is to be made; on disturbances of the fæcal function and presence of hæmorrhoids, the digital examination of the rectum; on anomalies of menstruation, etc., the examination of the uterine appendages.

The examination, as indicated above, should be carried out in *every* patient, no matter what affection may be indicated by the history. If this rule be not followed, and the examination is limited to a symptom or to an organ that may have appeared prominent in the history, the diagnosis is frequently faulty, as the examination of the remaining parts of the body is involuntarily more or less neglected. The diagnostician is very apt to follow an incorrect train of thought, and is led to an erroneous interpretation of certain symptoms on account of his preconceived opinion [snap diagnosis].

If the diagnosis is to be entirely unprejudiced, and to be determined as objectively as possible and the probable ailment brought out by the history not exclusively dwelt upon, the method previously mentioned should be adhered to in general.

All the morbid phenomena which have been obtained by means of the various methods of examination are now collected, and, if necessary, a renewed investigation of the organ in which pathological manifestations have been noted is undertaken, so that the result may be controlled in various directions. The more exact details of examination are now brought into action, such as the microscopic examination of the sputum, of the urinary sediment, of the dejecta, of the blood, the exact analysis of vomited material, of the urine, the action of the blood-vessels, the temperature, the condition of the sphygmographic pulse curve, the measure of both halves of the thorax and their excursus, the ophthalmoscopic examination of the eye ground, examination of the ear with the speculum, of the larynx and trachea with the laryngoscope, of the reaction of the nerves by means of the electric current, the various disturbances of sensation and vaso-motor action and reflexes, possible disturbances of speech, and others.

Finally, resting upon the completed basis of the results obtained by examination, the *intellectual act of working all the pathological phenomena into a diagnosis, the moulding of the morbid form into a definite affec-*

tion begins. To do this, it is necessary at first to separate the essential from the non-essential, unimportant symptoms, to consider which symptoms in the pathological picture are to be regarded as primary, and which may be taken as consequences of them. This not only requires a knowledge of pathology as a whole, of the symptom-complex recognised by clinical and anatomical experience as characteristic of a certain disease, but, above all, a knowledge of physiology and the faculty to comprehend fully the *course* of the pathological process and correctly to estimate the resulting interlacings of separate processes in the special organism which has become diseased, and therefore acts under changed conditions. If, by this means, the physician has succeeded in understanding the interrelations of the various morbid phenomena in a special case, the decision of the question must follow to which variety of disease, according to the various forms of maladies as differentiated in the course of time, do the objective findings conform most. Only in rare instances are the latter so manifest that the placing of the affection in question into one of the traditional, closely circumscribed categories of disease may be done without any further thought. On the contrary, in most instances it is necessary, in order to arrive at a positive diagnosis, to refer for comparison to those pathological conditions in which the symptoms are similar to the affection which probably presents itself. In some cases this method of diagnosis so dominates the situation that it forms the chief method of arriving at a diagnosis. Under these circumstances we speak specially of *differential diagnosis*, although, as previously stated, "differential diagnosis" plays a certain part in every diagnosis, even if not particularly noted by the diagnostician.

After the diagnosis has reached the point that the affection has been recognised and a distinct name given to it, it is necessary to go into details regarding the anomalies or deviations from the *ordinary* type to see whether they are consistent with the diagnosis, or whether they may be explained by the individuality of the patient or are due to accompanying conditions. It is especially essential to consider carefully the complications, as to whether they may be looked upon as usual or unusual in the case in question; it is further to be observed to what extent the complications are able to modify the clinical picture, and, a point to which I call particular attention, whether they are to be looked upon as separate processes accompanying the principal affection or are independent of the latter. If this point is not sufficiently taken into consideration, we are very apt to distort the clinical picture. There can be no doubt that, in the *majority* of instances, only *one* primary affection is present, and that the symptoms which do not correspond to the usual clinical syndrome can be made to fit into the groundwork of the affection in question. It should be a chief consideration, therefore, whenever possible, to make the *attempt*, starting from the primary affection, of which the diagnosis has been made, to arrange all symptoms in their genetic connection. But we should not go further in this respect than is consistent with cool, objective reasoning; any forced effort to bring together all symptoms under *one* aspect is certain to avenge itself most emphatically. Supported by a long experience,

I believe this to be one of the most dangerous points upon which occasionally a diagnosis, arrived at after the most careful consideration, shatters and in this manner becomes a "misdiagnosis" (*Fehldiagnose*).

An example may illustrate this:

A woman, forty-seven years old, was admitted to my clinic suffering from marked swelling of the abdomen. On examination an extensive dulness and fluctuation was noted, as well as a hard, resisting mass within the area of dulness. For reasons not to be defined here, the diagnosis of cystosarcoma of the ovaries was made. It was decided not to operate. Success was questionable principally on account of a glandular mass the size of a walnut in the axilla, which was considered metastatic and rendered the malignant character of the tumour all the more probable inasmuch as the swollen gland was partly hard to the touch. The autopsy showed that the latter conclusion was based on an *erroneous* presumption. It is true, the tumour was ovarian, which in this case was difficult to diagnosticate, in accordance with the clinical evidence. It was a cysto-adenoma colloides ovarii utriusque; *but the swollen gland had nothing whatever to do with it, it was not of a metastatic, but of a caseous-tuberculous character,* an accidental secondary finding which gave rise to wrong conclusions as to the character and the practicability of operation.

I remember another case in my practice in which there was quite an argument between the ophthalmologist and myself whether there was an affection of the brain or of the spinal marrow present. It was certainly correct in principle that I would not change the diagnosis of an affection of the spinal marrow as long as the patient lived, because the symptoms could easily be ascribed to such an affection; and yet, this obstinacy in adhering to the diagnosis was not correct, the ophthalmoscopical examination rather favouring the presence of a cerebral tumour. The autopsy proved that both diagnoses were correct, that tumours *both* in the spinal marrow *and* in the brain caused the symptoms.

Another rule, which I successfully employ in my diagnoses, is always to select as my final opinion the *most usual* probability, if two possibilities present themselves in a doubtful case in which the chances are about equal. It is also well not to make too finely precise anatomical diagnoses, for instance in pleural exudates and pulmonary infiltrations. Do not attempt to determine their extent to the centimetre. Under such circumstances it is possible that the autopsy will often give you a disagreeable surprise, simply because demands have been made upon physical diagnosis which this art cannot fulfil. For a positive diagnosis in quite rare cases the physical, chemical, and bacteriologico-microscopical examinations alone are not sufficient; it will become necessary to introduce experiment as a diagnostic auxiliary, for instance, the employment of bacteriological cultures, the inoculation of the latter or of pathological material upon animals.

If, after a careful consideration of the results of the examination and of the course of the disease, the diagnosis has been decided upon, *it is not well, in general, readily to change the diagnosis* if some symptom or other, which makes its appearance later on, seems to be more in accordance with another view than the one decided upon. This gives rise to vacillation and hesitation in diagnosis, and, in my experience, such diagnoses, as a rule, are wrong which have been substituted by reason of symptoms occurring shortly before death, in place of the original well-considered one. At any rate, I would advise only to change the diagnosis if there exists a really urgent reason for doing so. The physician who is not very conscientious regarding a change of diagnosis, easily falls into the habit of making only

provisional diagnoses, and to postpone the thorough examination and consideration of the case until later; in this way he will never be able to understand the seriousness and certainty in diagnosis.

Should the patient die, we ought, before the autopsy takes place, again call to our mind the entire pathological picture of the disease with its symptoms, the result of the examination, and the various phases of its course, and again look into the reasons which determined us to make this or the other diagnosis. Now to alter our opinion is not admissible after what has been said, and this is always a sign that the fullest attention and the proper care has not been paid to the diagnosis while the patient was still alive. But the review of the case before the necropsy has *this* advantage, that the shortcomings of the diagnosis are rendered clear, or that one or the other fault in diagnosis is brought to our notice and avoided in the future; that we are better prepared for the result of the autopsy, and less surprised if it should not conform to the diagnosis. The clinical diagnosis should be handed to the pathologist in writing before the autopsy takes place. Whenever possible, the physician who made the diagnosis should not perform the autopsy. A purely objective result, which can be used for self-criticism, can be obtained only in this manner. It is always left to the diagnostician, if some point or other is not in accordance with his diagnosis, to ask the pathologist for an explanation of the findings of the autopsy in certain directions.

It is scarcely necessary to allude to what the diagnostician may learn from carefully conducted autopsies. There is no doubt that a diagnosis may prove itself to be right or wrong according to the occurrence of symptoms and signs, or from the unquestionable effect of certain therapeutic measures. Thus it may occur that, in a case in which the often difficult differential diagnosis between enteric fever and acute miliary tuberculosis is for a long time undecided, the favourable course of the malady decides us in favour of enteric fever; that, in a doubtful case of pleurisy, an exploratory puncture demonstrates the presence of an exudate, etc. The principal correction of the diagnosis, however, is and will always be the autopsy. I do not hesitate to admit that I owe a great deal of my diagnostic skill to pathological anatomy.

The comparison of the anatomical diagnosis (which is also to be given in writing by the pathologist) with the clinical diagnosis proves to be of especial value. Here every point is to be subjected to a rigid criticism; it should be taken into consideration how eventual errors in the diagnosis could have been avoided, the beginning, course, and termination of the disease in the *epicrisis* should be again reflected upon before the case is disposed of, which, with other similar cases, should be used as a criterion in the diagnoses of the future.

If the clinical diagnosis corresponds in the *majority* of cases to the anatomical—and every physician ought to perfect his diagnostic skill up to this point—then we feel satisfactorily conscious that also in the majority of those cases in which there has been no autopsy, the diagnosis will have been correct. If less serious as well as severer cases are treated with the same amount of care in their examination, I do not fear that the discre-

tion of the physician suffers any loss from this point of view. For even the most expert diagnostician, reflecting upon the results obtained by the autopsy, will continually feel that his diagnostic knowledge is here and there deficient and needs improvement. On the other hand, however, the conscientious aim in this direction, the more minute perfection of diagnosis, gives the physician the firmness and calmness required at the bedside, and becomes a fruitful source of lasting, genuine satisfaction. If this already is found in the search for truth, it acquires its proper sanction in the thought that, with the increase in certainty in diagnosis, the ability to help our suffering fellow-beings materially increases. Then the ancient dictum *qui bene dignoscit, bene curat* will remain true as long as scientific medicine exists.

CONTENTS

	PAGE
INTRODUCTION	v-xii
DIAGNOSIS OF HEART DISEASES	1
Preliminary Remarks	1
DIAGNOSIS OF INDIVIDUAL HEART AFFECTIONS	6
Diseases of the Endocardium	6
Acute Endocarditis	6
Chronic Endocarditis—Valvular Disease	11
Mitral Insufficiency	12
Stenosis of the Mitral Valve	15
Insufficiency of the Aortic Valves	19
Stenosis of the Aortic Orifice	26
Insufficiency of the Valves of the Pulmonary Artery	29
Stenosis of the Pulmonary Orifice	29
Insufficiency of the Tricuspid Valves	30
Stenosis of the Tricuspid Orifice	33
Combined Valvular Defects	33
Malformations of the Heart and the Large Vessels	36
Persistency of the Ductus Botalli	37
Persistency of the Aortic Isthmus	38
Diseases of the Pericardium	39
Pericarditis	39
Sequels of Pericarditis, Adhesive Pericarditis, Calcareous Mediastino-pericarditis	45
Pneumopericardium	47
Affections of the Myocardium	48
Myocarditis	48
The Fatty Heart, Adipose Degeneration of the Musculature of the Heart	51
Spontaneous Rupture of the Heart	53
Hypertrophy of the Heart	53
The Neuroses of the Heart	57
Nervous Palpitation of the Heart	57
Angina Pectoris—Stenocardia	57
Cardiac Asthma	58
DIAGNOSIS OF DISEASES OF THE LARGE VESSELS	62
Atheroma of the Arteries, Arteriosclerosis	62
Aortic Aneurysm	64
DIAGNOSIS OF DISEASES OF THE RESPIRATORY ORGANS	68
Diseases of the Larynx	68
Acute Laryngitis—Acute Laryngeal Catarrh	68
Special Forms of Acute Laryngitis	68
Chronic Laryngitis	70

PAGE

DIAGNOSIS OF DISEASES OF THE RESPIRATORY ORGANS (*Continued*)

Diphtheritic Laryngitis—Croup of the Larynx 70
Laryngeal Œdema; Œdema of the Glottis 72
Perichondritis 72
Ulcers of the Larynx 73
Cicatrization in the Larynx—Stenosis of the Larynx 77
New Growths in the Larynx 78
Foreign Bodies in the Larynx 80
Neuroses of the Larynx 81
Sensory Neuroses 81
Disturbances of Motility of the Larynx 82
Spasms of the Muscles of the Larynx 82
Laryngospasm 82
Paralysis of the Laryngeal Nerves and Muscles 83
Motor Paralysis in the Region of the Superior Laryngeal Nerve . 83
Paralysis in the Region of the Recurrent Nerve 84
Complete Paralysis of the Recurrens 84
Isolated Paralysis of the Individual Muscles supplied by the Inferior Laryngeal Nerve 85
Paralysis of the Glottis-closing Muscles—Paralysis of the Adductors 85
Paralysis of the Lateral Crico-arytænoid 86
Paralysis of the (Internal) Thyreo-arytænoid 86
Paralysis of the Transverse Arytænoid (Interarytænoid) . . 87
Paralysis of the Abductors of the Glottis 87
Affections of the Trachea and the Bronchi 90
Catarrh of the Bronchi; Bronchitis 90
Putrid Bronchitis 91
Capillary Bronchitis 92
Fibrinous Bronchitis 94
Bronchiostenosis 95
Perforation of the Bronchial Wall 98
Bronchiectasis 99
Bronchial Asthma 103
Diseases of the Pulmonary Tissue 106
Atelectasis of the Lungs 106
Congestion of the Lungs 108
Pulmonary Emphysema 110
Œdema of the Lungs 117
Inflammatory Alveolar Infiltration—Pneumonia 121
Fibrinous-Croupous Pneumonia 121
Broncho-pneumonia—Catarrhal Pneumonia 126
Interstitial Pneumonia 128
Caseous Pneumonia, Chronic Pulmonary Tuberculosis, Phthisis pulmonum 130
I. Diagnosis of the First Stage of Pulmonary Tuberculosis . . . 131
II. Diagnosis of the Second Stage of Pulmonary Tuberculosis . . 134
III. Diagnosis of the Third Stage of Pulmonary Tuberculosis . . . 135
Embolism of the Pulmonary Artery, Hæmorrhagic Infarct . . . 139
Pulmonary Abscess 141
Gangrene of the Lungs 143
Syphilis of the Lungs 145
Neoplasms of the Lungs 145

PAGE

DIAGNOSIS OF DISEASES OF THE RESPIRATORY ORGANS (*Continued*)
Actinomycosis of the Lungs 146
Diseases of the Mediastinum 146
Mediastinal Tumours 146
Diseases of the Pleura 153
Pleuritis 153
Pleuritis exsudativa (Pleurisy with Effusion) 155
Pneumothorax 162
Neoplasms of the Pleura 168
DIAGNOSIS OF AFFECTIONS OF THE ABDOMINAL ORGANS 169
DIAGNOSIS OF AFFECTIONS OF THE LIVER 170
Introduction 170
Acute Yellow Atrophy of the Liver 176
Hepatic Cirrhosis—Atrophic Interstitial Hepatitis—Laennec's Atrophy of the Liver 180
Connective-Tissue Hyperplasia of the Liver—Hypertrophic Cirrhosis—Elephantiasis of the Liver 185
Syphilitic Affections of the Liver—Syphilis of the Liver 187
Suppurative Hepatitis—Abscess of the Liver 189
Hyperæmia of the Liver 192
Fatty Liver 194
Amyloid Degeneration of the Liver—Amyloid Liver 195
Cancer of the Liver 196
Echinococcus of the Liver 204
Perihepatitis 205
Changes in Form and Position of the Normal Liver 206
Diseases of the Bile Ducts 207
Stenosis or Occlusion of the Biliary Passages—Jaundice 207
Acute Febrile Infectious Jaundice—Weil's Disease 213
Cholelithiasis 214
Diseases of the Gall-bladder 217
Diseases of the Blood-vessels of the Liver 219
Pylethrombosis—Pylephlebitis 219
Aneurysms of the Hepatic Artery 220
Table for the Diagnosis of Diseases of the Liver 221
DIAGNOSIS OF DISEASES OF THE PANCREAS 222
Carcinoma of the Pancreas 223
Cysts of the Pancreas 225
Stones of the Pancreas—Pancreatic Stone Colic 225
DIAGNOSIS OF DISEASES OF THE SPLEEN 227
Hyperæmia of the Spleen—Hyperplasia of the Spleen 228
Embolism of the Spleen—Hæmorrhagic Infarct of the Spleen—Abscess of the Spleen 230
Splenitis 231
Rupture of the Spleen 231
Amyloid Spleen 231
Neoplasms of the Spleen—Syphilitic Changes of the Spleen . . . 232
Parasites of the Spleen 232
Perisplenitis 232
Changes of Form and Position of the Spleen—Floating Spleen . . . 232
DIAGNOSIS OF DISEASES OF THE DIGESTIVE TRACT 235
Diseases of the Oral Cavity 235
Scurvy of the Oral Cavity 237

PAGE

DIAGNOSIS OF DISEASES OF THE DIGESTIVE TRACT (*Continued*)

Noma, Gangrene of the Cheek, Cancrum Oris 237
Development of Fungi in the Mouth—Bacterial Diseases of the Mouth 238
Diseases of the Palate and the Pharynx 239
Angina—Pharyngitis 239
Diphtheria of the Palate and Pharynx 242
Tuberculosis of the Palate and Pharynx 244
Syphilis of the Palate and Pharynx 244
Diseases of the Œsophagus 245
Œsophagitis 245
Ulceration of the Œsophagus 245
Neoplasm of the Œsophagus 246
Stenosis of the Œsophagus 247
Dilatations of the Œsophagus 251
Rupture of Continuity of the Œsophagus 253
Neuroses of the Œsophagus 255
Diseases of the Stomach 255
Anatomico-Physiological Preliminary Observations 255
Diagnostic Preliminary Remarks 259
Catarrh of the Stomach—Gastritis in its Various Forms 264
Phlegmonous Gastritis—Interstitial Purulent Gastritis—Abscess of the Stomach 266
Toxic Gastritis 267
Mycotic Gastritis 267
Chronic Gastritis—Chronic Catarrh of the Stomach 268
Atrophy of the Mucous Membrane of the Stomach—Anadenia of the Stomach—Achylia gastrica 271
Ulcus ventriculi pepticum—Gastric Ulcer 273
Cancer of the Stomach—Carcinoma ventriculi 280
Dilatation of the Stomach—Gastrectasis 287
Changes of Form and Position of the Stomach 291
Gastroptosis 292
Vertical Position of the Stomach 292
Neuroses of the Stomach 292
Nervous Dyspepsia 294
Case I. Nervous Dyspepsia with Normal Acid Secretion . . . 295
Case II. Nervous Dyspepsia with Excessive Acid Production . 295
Case III. Nervous Dyspepsia with Subacidity respectively Anacidity 295
Sensory Neuroses 297
Gastralgia Gastrodynia—Spasm of the Stomach 297
Hyperæsthesia of the Mucous Membrane of the Stomach . . . 299
Disturbance of Hunger and Satiation 299
Neuroses of Secretion 300
Nervous Hyperchlorhydria 300
Nervous Gastrosuccorrhœa, Periodic Excessive Secretion of Gastric Juice 301
Continuous Gastrosuccorrhœa—Chronic Excessive Secretion of Gastric Juice—Continuous Secretion of Juice 302
Depressive Condition of the Secretory Nerves of the Stomach—Nervous Subacidity—Achylia gastrica nervosa 303
Motor Neuroses 304
Irritative Condition of the Gastric Motor Nerves 304
Hyperkinesis, Peristaltic Unrest of the Stomach 304
Cardiac Spasm 305

PAGE

DIAGNOSIS OF DISEASES OF THE DIGESTIVE TRACT (*Continued*)

Spasm of the Pylorus 306
Nervous Eructation and Vomiting 306
Motor Neuroses of a Depressive Character 307
Insufficiency or Incontinence of the Pylorus 307
Insufficiency of the Cardia 307
Diseases of the Intestines 308
Acute Inflammatory Processes of the Intestines 308
Acute Diffuse (Simple) Enteritis, Acute Intestinal Catarrh . . . 308
Inflammations proceeding from the Large Intestine in the Right Iliac Fossa—Typhlitis, Skolikoiditis (Nothnagel), "Appendicitis," Perityphlitis 311
Colitis, Proctitis 315
Chronic Intestinal Catarrh 316
Intestinal Atrophy (Atrophia intestinalis) 318
Ulcers of the Intestines 319
Cancer of the Intestine 325
Enterostenosis—Intestinal Contraction. Intussusception 330
Nervous Intestinal Affections 338
Motor Neuroses of the Intestine 339
Nervous Diarrhœa 339
Peristaltic Unrest of the Intestine (Tormina intestinorum) . . . 340
Enterospasm 340
Motor Neuroses of a Depressive Character 341
Sensory Neuroses of the Intestine 342
Enteralgia, Neuralgia mesenterica, "Nervous Colic" 342
Sensory Neuroses of a Depressive Character 344
Neuroses of Secretion of the Intestine 345
Helminthiasis—Intestinal Worms 345
DIAGNOSIS OF DISEASES OF THE URINARY ORGANS 346
Preliminary Remarks 346
Diseases of the Kidneys 348
Hyperæmia of the Kidneys—Congested Kidney 348
Cholera Kidney, Gestation Kidney 350
Diffuse Nephritis, Bright's Disease 351
Acute Nephritis—Acute Parenchymatous Nephritis, Acute Bright's Disease 351
Chronic Nephritis s. str., Subacute and Subchronic Nephritis, Chronic Parenchymatous Nephritis, Large White Kidney. Large Red ("chronic hæmorrhagic nephritis") 357
Secondary Atrophic Kidney 361
Primary, Genuine Atrophic Kidney ("chronic interstitial" nephritis); Sclerosis of the Kidney 362
Amyloid Degeneration of the Kidney—Fatty Kidney 364
Suppurative Nephritis, Septic and Pyæmic Nephritis, Pyelonephritis, Renal Abscess 367
Diagram of the Differential Diagnosis of Diffuse Affections of the Kidney 368-369
Paranephritis 372
Hæmorrhagic Infarct of the Kidney 373
Tuberculosis of the Kidney—Nephrophthisis 374
Tumours of the Kidney 376

PAGE

DIAGNOSIS OF DISEASES OF THE URINARY ORGANS (*Continued*)
Cystic Tumours of the Kidney—Renal Cysts—Hydronephrosis, Echinococcus . . . 380
Renal Cysts—Hydronephrosis . . . 381
Echinococcus of the Kidney . . . 384
Solid Tumours of the Kidney—Cancer of the Kidney—Sarcoma of the Kidney . . . 385
Nephrolithiasis—Formation of Concrement in the Kidneys and the Urinary Passages . . . 387
Anomalies of Form and Position of the Kidneys . . . 391
Movable Kidney, Wandering Kidney, Nephroptosis . . . 391
Diseases of the Urinary Bladder . . . 393
Catarrh of the Bladder . . . 393
Tuberculosis of the Bladder . . . 398
Neoplasms of the Bladder—Cancer of the Bladder . . . 399
Hæmorrhoids of the Bladder . . . 400
Nervous Disturbances of the Function of the Bladder . . . 400
Paralysis of the Musculature of the Bladder, "Bladder Paralysis," Cystoplegia . . . 401
Paralysis of the Detrusor . . . 401
Paralysis of the Sphincter . . . 401
Synchronous Paralysis of the Detrusor and Sphincter Vesicæ . . . 402
Spasm of the Bladder Musculature—Cystospasm . . . 402
Simultaneous Spasm of the Detrusor and Sphincter Vesicæ . . . 403
Disturbance of Sensibility of the Urinary Bladder . . . 403
DIAGNOSIS OF DISEASES OF ADRENAL BODIES . . . 405
Addison's Disease . . . 405
DIAGNOSIS OF DISEASES OF THE PERITONÆUM . . . 408
Peritonitis . . . 408
Acute Peritoneal Inflammation, Acute Peritonitis, Pneumo-Peritonitis . . . 408
Circumscribed Acute Peritonitis . . . 412
Perforative Peritonitis, Pneumo peritonitis . . . 413
Chronic Peritonitis . . . 414
Circumscribed Chronic Peritonitis . . . 414
Diffused Chronic Peritonitis . . . 415
Tuberculosis of the Peritonæum—Tuberculous Peritonitis . . . 417
Tumours of the Peritonæum . . . 419
Sarcoma; Cancer of the Peritonæum; Mesenteric Cyst . . . 419
Ascites . . . 421
DIAGNOSIS OF DISEASES OF THE NERVOUS SYSTEM . . . 425
Diseases of the Peripheral Nerves, Anatomical and Physiological . . . 426
Introduction . . . 426
Diagnostic Preliminary Remarks . . . 433
DIAGNOSIS OF DISEASES OF THE SENSORY NERVES . . . 435
Anæsthesia . . . 436
Anæsthesia of Smell, Anosmia . . . 437
Anæsthesia of Taste, Ageusia . . . 438
Cutaneous Anæsthesia . . . 442
Hyperæsthesia—Neuralgia . . . 445
Neuralgia of Individual Nerves . . . 451
Neuralgia of the Trigeminus Nerve, Prosopalgia . . . 451

PAGE

DIAGNOSIS OF DISEASES OF THE SENSORY NERVES (*Continued*)
Neuralgia of the Cervical Nerves 453
Intercostal Neuralgia 454
Neuralgia of the Lumbar Nerves 456
Neuralgia of the Sacral Nerves—Neuralgia of the Sciatic Plexus—Sciatica 458
DIAGNOSIS OF DISEASES OF THE MOTOR NERVES 465
Paralysis of the Peripheral Motor Nerves 465
Paralysis of the Motor Portion of the Fifth Nerve—Paralysis of the Muscles of Mastication 467
Paralysis of the Facial Nerve—Mimical Facial Paralysis 468
Paralysis of the Vagus 479
Paralysis of the Spinal Accessory 479
Paralysis of the Hypoglossal Nerve—Glossoplegia 481
Paralysis in the Course of the Cervical Nerves 483
Paralysis in the Course of the Terminal Branches of the Brachial Plexus 486
Paralysis in the Course of the Radial Nerve 486
Paralysis in the Course fo the Ulnar Nerve 489
Paralysis in the Course of the Median Nerve 490
Combined Paralysis of the Nerves of the Arm 491
Paralysis in the Course of the Dorsal Nerves 492
Paralysis in the Course of the Lumbar and Sacral Nerves . . . 492
Paralysis of the Crural Nerve 493
Paralysis of the Obturator Nerve 493
Paralysis of the Gluteal Nerves 493
Paralysis of the Sciatic Nerve 494
Spasm in the Course of the Motor Nerves 495
Spasm in the Course of the Cranial Nerves 496
Spasm in the Course of the Cervical Nerves 499
Spasm in the Course of the Lumbar and Sacral Nerves 502
Neuritis, Multiple Neuritis 503
DIAGNOSIS OF DISEASES OF THE SPINAL CORD 512
Anatomico-Physiological Introduction 513
Preliminary Diagnostic Remarks 525
Diseases of the Membranes of the Spinal Cord 529
Acute Spinal Meningitis 529
Chronic Spinal Meningitis 533
DIAGNOSIS OF DISEASES OF THE SPINAL CORD SUBSTANCE 536
Preliminary Remarks 536
Tabes Dorsalis [Locomotor Ataxia]—Gray Degeneration of the Posterior Columns—Chronic Posterior Leucomyelitis 537
Hereditary Ataxia—Family Ataxia—Friedreich's Disease . . . 549
Degeneration of the Motor Conduction Tract 551
Primary and Secondary Degenerations of the Lateral Columns . . 551
Spastic Spinal Paralysis 553
Amyotrophic Lateral Sclerosis 554
Anterior Poliomyelitis 558
Acute Anterior Poliomyelitis—Acute Anterior Infantile Poliomyelitis—Spinal (Essential) Infantile Paralysis 558
Acute Anterior Adult Poliomyelitis, Acute Amyotrophic Spinal Paralysis of Adults 561

PAGE

DIAGNOSIS OF DISEASES OF THE SPINAL CORD SUBSTANCE (*Continued*)

Subacute and Chronic Anterior Poliomyelitis 562
Progressive Spinal Muscular Atrophy: Poliomyelitis s. Polioartrophia Anterior Chronica Progressiva 563
Neural Muscular Atrophy (Peroneal Type of Progressive Muscular Atrophy) . 567
Dystrophia Muscularis Progressiva (Erb)—Juvenile Myopathic Muscular Atrophy—Pseudohypertrophy 568
Posterior Poliomyelitis 570
Syringomyelia—Cavity Formation in the Spinal Cord—Gliosis Spinalis . . 571
Diffuse Myelitis (Acute and Chronic Myelitis, Transverse Myelitis) . . . 573
Compression of the Spinal Cord, Spinal-Pressure Paralysis, Compression Myelitis 579
Unilateral Lesions of the Spinal Cord—Brown-Séquard's Spinal Paralysis . 582
Tumours of the Spinal Cord, Tumours of the Spinal Marrow and its Meninges 583
Acute Ascending Spinal Paralysis, Landry's Paralysis 586
Multiple Focal Affections of the Spinal Cord (and Brain)—Acute Disseminated Myelitis—Multiple Myelitis 588
Chronic Disseminated Myelitis, Sclerosis Cerebrospinalis Multiplex. Multiple Sclerosis of the Brain and Spinal Cord 589
Anæmia, Hyperæmia of the Spinal Cord, Spinal-Cord Hæmorrhage, Myelomalacia 594

DIAGNOSIS OF DISEASES OF THE MEDULLA OBLONGATA AND THE PONS . . 596
Anatomico-Physiological Introduction 596
Diagnostic Preliminary Remarks 606
Hæmorrhages of the Pons and of the Medulla Oblongata 610
Chronic Progressive Bulbar Paralysis—Duchenne's Disease 612
Pseudobulbar Paralysis—Bulbar Paralysis of Supranuclear Origin—Paralysis Glossopharyngolabialis Cerebralis et Cerebrobulbaris 615
Progressive Nuclear Ophthalmoplegia 616
Functional Asthenic Bulbar Paralysis—Bulbar Paralysis without Anatomical Lesions—Myasthenia Gravis Pseudoparalytica 616
Compression of the Pons and Medulla Oblongata 617

DIAGNOSIS OF DISEASES OF THE CEREBELLUM 619
Anatomico-Physiological Introduction 619
Clinico-Diagnostic Remarks 622

DIAGNOSIS OF DISEASES OF THE MIDDLE BRAIN (CEREBRAL PEDUNCLES AND CORPORA QUADRIGEMINA) 626
Anatomico-Physiological Preliminary Remarks 626
Clinico-Diagnostic Remarks 630
Affections of the Anterior Brain 634
Anatomico-Physiological Introduction 634
Clinico-Diagnostic Preliminary Remarks 644
Disturbances of Speech—Aphasia, Alexia, Agraphia 651

DIAGNOSIS OF THE VARIOUS FOCAL DISEASES OF THE ANTERIOR BRAIN . . 664
Cerebral Hæmorrhage—Sanguineous Apoplexy—Hæmorrhagia Cerebri . . 664
Embolism and Thrombosis of the Cerebral Arteries: Softening of the Brain—Encephalomalacia 676
Neoplasms of the Brain, Cerebral Tumour 680
Abscess of the Brain—Encephalitis Suppurativa 691
Sclerotic Encephalitis—Polioencephalitis—Non-suppurating Inflammation of the Substance of the Brain 696
Dementia Paralytica, Progressive Paralysis of the Insane 697

PAGE

DIAGNOSIS OF THE VARIOUS FOCAL DISEASES OF THE ANTERIOR BRAIN (*Continued*)

Diffuse Affections of the Brain. General Disturbances of Circulation—Hyperæmia and Anæmia of the Brain—Hyperdiæmorrhysis and Adiæmorrhysis Cerebri (Geigel) 699

Meningitis—Suppurative and Tuberculous Meningitis—Convexity Meningitis—Basilar Meningitis 702

Thrombosis and Phlebitis of the Sinuses of the Brain—Sinus Thrombosis . 714

Meningeal Hæmorrhages—Hæmatoma of the Dura Mater 718

Hydrocephalus (Hydrops Ventriculorum Cerebri—Hydrocephalus Internus et Externus s. Intermeningealis) 719

DIAGNOSIS OF FUNCTIONAL DISEASES OF THE BRAIN (SO-CALLED NEUROSES) . 722

Hysteria 722

Neurasthenia, Nervousness 731

Traumatic Neurosis 734

Epilepsy, Falling Sickness 736

Chorea (Chorea Minor, Sydenham's Chorea, St. Vitus's Dance) . . . 744

Athetosis 746

Paramyoclonus Multiplex—Myoclonia 747

Tetany 748

Shaking Palsy, Paralysis Agitans, Parkinson's Disease 751

Myotonia Congenita—Thomsen's Disease 753

So-called Angeioneuroses and Trophoneuroses 754

Progressive Facial Hemiatrophy and Hemihypertrophy 754

Erythromelalgia 755

Acute Angeioneurotic Œdema, Acute Circumscribed Cutaneous Œdema . 755

Intermittent Articular Dropsy 756

Symmetrical Gangrene, Raynaud's Disease 756

Acroparæsthesia 756

Acromegaly, Pachyacria 757

Arthropathic Hypertrophy 757

Myxœdema 758

Graves's Disease (Basedow's Disease, Exophthalmic Goître) . . . 759

DIAGNOSIS OF DISEASES OF THE MUSCLES 765

Acute and Chronic Muscular Rheumatism 765

Acute Multiple Myositis 766

Myositis Ossificans 770

Sunstroke and Heat Exhaustion 771

Diseases of the Blood and of Metabolism—Constitutional Diseases . . . 772

I. Diseases of the Blood 772

Anatomico-Physiological Introduction 772

Anæmia—Chlorosis—Severe ("Pernicious") Anæmia 776

Leucæmia 790

Leucocytosis 800

Pseudoleucæmia—Hodgkin's Disease—Lymphadenia 802

Leucanæmia 803

Hæmorrhagic Diathesis—Purpura Hæmorrhagica, Peliosis Rheumatica, Morbus Maculosus Werlhofii, Scurvy, Hæmophilia 807

Hæmoglobinæmia—Hæmoglobinuria 811

II. Diseases of Metabolism 817

Physiological Introduction 817

Diabetes Mellitus 823

PAGE

DIAGNOSIS OF DISEASES OF THE MUSCLES (*Continued*)

Diabetes Insipidus 837
Obesity—Adipositas Universalis 839
[Adiposis Dolorosa—Dercum's Disease] 844
Gout—Arthritis 844
Arthritis Deformans; Chronic Articular Rheumatism 849
Osteomalacia 850
Rhachitis; Rickets 852
Osteitis Deformans, Pseudo Rhachitis Senilis, Paget's Disease . . . 854

INFECTIOUS DISEASES 856
Preliminary Remarks; General Considerations 856

DIAGNOSIS OF INFECTIOUS DISEASES 862
Measles, Morbilli, Rubeola 863
Scarlet Fever, Scarlatina 870
Rötheln, Rubella, German Measles, French Measles, Epidemic Roseola . 879
Variola—Small-pox 880
Varicella—Chicken-pox 889
Erysipelas, Cryptogenetic Erysipelas—St. Anthony's Fire—The Rose . 891
Typhus Fever—Typhus Exanthematicus—Spotted Fever—Spotted Typhus . 896
Relapsing Fever, "Recurrens"—Typhus Recurrens 900
Enteric Fever—Typhoid Fever—Typhus Abdominalis 906
Digestive Organs 916
Respiratory Organs 917
Organs of Circulation 917
Urinary and Sexual Organs 918
Nervous System 918
Skin, Muscles, and Bones 918
Paratyphoid 927
Dysentery—Bloody Flux 928
Asiatic Cholera—Cholera Infectiosa 931
Mumps, Epidemic Parotitis 937
Diphtheria 940
Pertussis; Tussis Convulsiva, Whooping-Cough 946
Cerebro-spinal Fever—Epidemic Cerebro-spinal Meningitis . . . 949
Acute Miliary Tuberculosis, General Diffuse Tuberculosis 955
Cryptogenetic Septicopyæmia 963
Acute Articular Rheumatism—Acute Polyarthritis—Acute Rheumatic Fever . 970
Malaria—Intermittent Fever 976
Influenza, La Grippe 985
Tetanus—Lockjaw 988
Hydrophobia, Rabies, Lyssa Humana 993
Anthrax 994
Glanders—Malleus Humidus 997
Trichinosis 998
[Yellow Fever] 1001
[Dengue] 1002
[Malta Fever, Mediterranean Fever, Rock Fever, Neapolitan Fever, Undulant Fever] 1004
[Plague—Bubonic Plague—Pest] 1004
[Leprosy] 1005

DIAGNOSIS OF DISEASES OF THE HEART

PRELIMINARY REMARKS

THERE is no other category of affections in which physical diagnosis is so important as a means of arriving at a correct diagnosis as in affections of the heart. Its findings almost exclusively form the foundation from which the deviations from the normal condition of the organ can be correctly recognised in an anatomical as well as in a functional respect. Provided the original cause of the disturbance has been detected, the consequences that will result from the disorganization of the centre of circulation may be deduced with almost mathematical precision. The diagnosis of the various diseases of the heart is, therefore, not difficult, as a rule, provided the physician understands the details of the normal conditions of circulation and has learned to think out the effects of disturbances of the same.

Results of Disturbances of Circulation.—In all diseases of the heart, immaterial whether they relate to affections of the valves, of the myocardium, or to pericardial effusion, it sooner or later results that, with the disturbance of circulation, necessarily *the blood pressure falls in the arterial system, but in the veins* (which empty their blood with greater difficulty into the heart in these pathological conditions), *on the other hand, it is raised. The velocity of the blood current* in the capillary system, on this account, becomes slower, for it is dependent upon the extent of variation between arterial and venous pressure, thus the blood in the capillary system loses more oxygen to the tissues. This abnormal condition of the circulation, and alteration in the blood itself, manifests itself by two very striking symptoms: *Œdema* of the skin and *cyanosis* of the most peripheral parts of the body.

Phenomena of Engorgement.—Upon closer examination of such patients engorgement of the circulation may be noted in other phenomena. In the first place, congestion and uniform increase in the consistence of the *liver* are almost always plainly demonstrable (in the milder forms less by percussion than by palpation), as is also the presence of hydrothorax, which is easily demonstrated if the exact position of the lower border of the lungs is determined; also, by way of control, an examination is made with the upper part of the patient's body bent forward, at the same time observing any decrease of the vesicular murmur or alterations of the latter into bronchial respiration; hydropericardium also is frequently present. Less constant are the consequences of engorgement in the *kidneys*. It appears that a greater de-

gree of disturbance of circulation is required for a characteristic change in the function of the kidneys in conditions of stasis—i. e., to reduce the amount of urine and produce a moderate elimination of albumin. The sediment of the urine in the latter case shows numerous salts (usually uric acid), few hyaline casts, and no, or at the utmost few, red blood cells. Rarely, in my experience, is a chronic inflammation of the kidneys a sequel of such hyperæmia. It then takes the form of interstitial inflammation, which may occur after long-continued engorgement of the kidneys. If, therefore, upon examination of the urine of patients affected with cardiac disease, not only symptoms of an engorged kidney are found, but also those of parenchymatous nephritis—i. e., many red blood cells, muddy colour of the urine (which is independent of the admixture of blood, as can be demonstrated by chemical examination of the urine), epithelial and altered casts, etc.—then we are dealing no longer with simple engorgement of the kidney, but with a complication, inasmuch as either accidentally a parenchymatous nephritis has been added to the affection of the heart, or, as is usually the case, both affections owe their existence to the *same* cause (generally an infection). To this latter category belong also the relatively frequent combinations of aortic insufficiency and contracted kidney, in which the latter, as well as the valvular defect, is dependent upon atheroma. *Ascites* as a result of the engorgement is rare; still rarer, in contrast to the hyperæmia of the liver, is enlargement of the *spleen*, because the latter is only indirectly affected by engorgement of the liver.

Venous Pulse.—On the other hand, in the graver forms of engorgement the distended jugular veins are commonly found to *pulsate*. This visible pulsation of the jugular veins is always a symptom of an overfilled venous system. Under these circumstances the venous pulse is more pronounced, which is the result of circulatory disturbance in the various cardiac affections. It is occasionally noted in healthy individuals, but rarely to the same marked extent. The wave which dilates the veins of the neck, noted in persons in whom there is no cardiac disturbance, even with decided overdistention of the venous system, never produces a pulse but only a heaving—an "undulation." According to Mosso's, Gottwalt's, and Riegel's investigations, the normal as well as this more developed (normal) venous pulse is a "negative" pulse (as compared with the expansion of the artery), so that, while the artery dilates, the vein contracts, and vice versa. At the same time this cata (mono) crotic shank of the venous pulse curve is short, the anacrotic long-drawn out, and shows a visible depression (anadicrotic). The second part of the anadicrotic shank of the curve corresponds to the systole of the auricle (presystole), the catamonocrotic one to the systole of the heart (diastole of the auricle); the pulse, therefore, is *diastolic-presystolic* in time. It is characteristic of the venous pulse, in contrast to the arterial pulse, and also of the impulse conveyed from the carotid to the jugular vein, that it is but slowly distended; it is commonly noted to occur with an interval, besides the lower pressure in the pulsating jugular vein, in contrast to the pulsating carotid artery. With the diastolic-presystolic venous pulse it is always noted that, as the artery contracts, the vein dilates with a pause, which corresponds to the systole of the auricle, and, as the systole of the heart takes place, contraction occurs. The conditions are different, however, as shown in his time by Riegel, regarding the occurrence of *that* venous pulse which appears in tricuspid insufficiency. The venous pulse noted in this condition is markedly developed, no mere undulation; upon careful examination it proves to be *presystolic-systolic* in time—i. e., during the systole of the auricle the vein dilates, as with the other varieties of venous pulse, but no contraction occurs with the systole of the heart; on the contrary, another dilatation of the vein takes place that forces the blood into the auricle through the insufficient valve, a synchronous dilatation. During the diastole which then follows, quite in contrast to normal conditions, no dilatation, but a contraction of the vein occurs—that is, a *relatively slighter deflux* of the venous blood takes place, until the systole of the auricle once more dilates the vein. It is therefore noted that in the "genuine" venous pulse, due to tricuspid insufficiency, the gradual distention of the vein sets in with the presystole and lasts during the entire systole of the heart. This latter condition, the distention of the vein with the systole of the heart, is the pathological sign of insufficiency of the tricuspid valve, which will be further entered upon when discussing the diagnosis of that valvular defect.

Compensation.—It would be absolutely wrong to presume that these symptoms of engorgement occur at once to the extent described, as a consequence of disturbances of circulation in the clinical picture of cardiac affections. Clinical experience teaches, on the contrary, that in valvular insufficiency all symptoms of engorgement that are worth mentioning are absent for a long time, as a rule; in those conditions which run a chronic course they may not appear for many years. The cause of this is found in the fact that the heart is still able to meet the increased demands thrust upon it by the valvular affection, by a corresponding exertion thus more or less completely equalizing the defect.[1] This is accomplished in the onset of acute disturbances in such a manner that the heart muscle, stimulated by the abnormally strong emptying and filling of its cavities due to obstacles, makes use of its "reserve force" to "accommodate itself to these increased demands." In case of continued obstruction of the circulation, however, a *permanent* factor is required to overcome these functional difficulties. This is furnished by those muscular fibres of the heart which are most concerned with contraction and which bear the brunt in the circulatory disturbance, they gradually undergo changes of increased metabolism and *hypertrophy*.

With this the force of the heart has increased, a *permanent* condition has been created equalizing the circulatory obstruction—*compensation*—by means of which the heart is able not only to do justice to the increased demand during rest, but also to accommodate itself to extraordinary efforts (for instance, mounting stairs). This leads to the surmise that the hypertrophied heart also possesses a certain amount of reserve force which, however, according to our clinical experience, is less than that of the normal heart. Thus the hypertrophied heart, its capacity of accommodation being smaller, will tire the sooner and the easier—it then becomes "insufficient." The reserve force—which even with moderately increased demands gradually fails ("rupture of compensation") or is entirely lost ("loss of compensation")—no longer meets the requirements, the power of the heart is now but sufficient to furnish an amount of force necessary for the circulation in a state of complete rest. Under these circumstances—i. e., if the compensation in the course of disease becomes defective, or from the onset was deficient, passive dilatation of the heart occurs, and this unquestionably leads to the grave consequences of circulatory disturbance previously mentioned.

Deficient Compensation.—A reason for deficient compensation is the inability of the heart to hypertrophy, as sometimes occurs in old or in very debilitated individuals; furthermore, the intensity of the anatomical changes in the heart, which cause the circulatory obstruction or the occurrence of complications. Above all, however, compensation in diseases of the heart becomes deficient from the fact that the energy [*vis a tergo*] required for the transformation into active energy is too rapidly consumed in proportion to its accumulation, which is the case in continued excessive demands upon the activity of the heart. Thus the power of the hypertrophied heart weakens, even if it was originally sufficient. This occurs the more readily and the earlier in cases in which general nutritive disturbances exist, or in which the heart muscle does not show throughout a normal consistency, but is deficient as a result of fatty degeneration, myocardiac infiltrations, or softening as

[1] Compare the works of M. v. Frey and F. Martius, in which the principles of compensation are exhaustively and clearly discussed.

a result of arteriosclerosis, etc. Finally, in the case of considerable exudates into the pericardial sac the circulatory disturbances are pronounced from the onset, inasmuch as here the diastolic distention of the cavities of the heart is rendered very difficult, and with it the systolic filling of the arteries is decreased.

Diagnostically the knowledge of the process of compensation in disease of the heart and of the obstructions to complete compensation, just mentioned, is of the utmost importance in various directions. It is obvious, according to the principles explained, that there may exist even marked anatomical changes in the heart, especially in the valves, without producing cyanosis and œdema, while, on the other hand, the latter may appear even in apparently insignificant changes in the heart, as soon as compensation does not take place sufficiently at the onset or fails in the course of time. The standard by which to judge the degree of compensation present is given, in the first place, by the condition of the pulse, especially by the degree of tension in the arteries. A small, and generally at the same time frequent, soft, irregular pulse announces deficient compensation; necessarily the diagnosis of the condition of the pulse in this respect is obtained chiefly by *sphygmographic* examination, which clearly shows the softness of the pulse and the irregularities of the pulse waves. The sphygmograph also gives the most important evidence in cases of full compensation to support the diagnosis of the special variety of valvular affection, inasmuch—as will be further explained later on—as the pulse in the different affections of the heart presents a very characteristic picture, not rarely even being [almost] pathognomonic. According to the law that the structures of the heart situated behind the points of circulatory disturbance are subjected to greater pressure by the blood, and that the muscles hypertrophy according to the greater demand made upon them, we shall have to expect in the different cardiac diseases, according to the variety of the affection, either dilatation or hypertrophy, or both, affecting the entire organ or only parts of the same. It is evident, if the especial form of heart disease can be determined from other symptoms, that the appearance of the subsequent symptoms just alluded to and resulting logically from the anatomical changes in the heart, gives a most important support to the diagnosis and also corroborates it.

Accidental Murmurs.—Of the various subsequent manifestations, the changes in the boundary of the heart upon percussion, of the apex beat and of the auscultatory signs, they will be referred to later on in the discussion of the individual diseases of the heart. But before entering upon this subject we must first look into an auscultatory phenomenon which in general, although not a sign of affection of the heart, quite often plays a part in the diagnosis of heart disease in a differentio-diagnostical respect. I refer to the exceedingly frequent "*accidental*" (functional, anæmic) murmurs to which no anatomical changes in the heart are found to correspond at the post mortem.

Differential Diagnosis.—In contradistinction to endocardial murmurs they usually show a *lesser intensity*; this, however, in my experience, does not always hold good. Their pitch is *soft, blowing*, the time at which they are heard is the *systole* of the ventricle; I have *never* absolutely determined a diastolic accidental murmur except in a case in which a transmitted venous murmur could be heard at the base of the heart occurring during diastole (compare page 23). My advice, therefore, is to do away entirely with diastolic murmurs from the category of accidental murmurs,

at least for diagnostic purposes. The distinctive characteristics mentioned so far are evidently uncertain in a differentio-diagnostic respect; it is more important to know that accidental (functional) murmurs generally vary very much *as to their intensity.* Their direct diagnosis depends on (1) that *the area of cardiac dulness does not extend beyond the normal borders and the apex beat is found in its normal position*; (2) *that the second pulmonic sound is not accentuated*; *and* (3) *that the murmurs are heard only, or at least plainest, at the ostium pulmonale* (*pulmonary area*). The latter position is, in my opinion, connected with the origin of these murmurs in the initial portions of the large vessels at the base of the heart. They may partly be caused by the fact that in anæmic and similar conditions the tonicity of the vascular wall is deficient, and by this means a greater dilatation of the arterial wall is caused, isochronous with the systole—i. e., a temporary dilatation of the vascular wall, and with it the formation of a murmur. As to time, according to this conception, this would coincide with the "time of expulsion" of the systole (see p. 20, note 1), and it is to be surmised that the slight sound which is formed during the systolic closure may be heard for a brief period besides the murmur—i. e., it introduces the latter. In some cases this can actually be determined beyond any question of doubt. For those cases in which the murmur occupies the entire period of the systole, the explanation of R. Geigel is probably correct, according to which in anæmics the pressure in the aorta and pulmonary artery is abnormally low, and thus the difference between the latter and the *relatively* great pressure in the ventricle is more marked at the onset of the systole than under normal circumstances. This prevents the closure of the semilunar valves during the so-called closure period and also the formation of the systolic sound, and thus a systolic murmur results upon the entrance of the blood into the large vessels, in the region of "physiological stenosis."

In contrast to these "anæmic" murmurs, the heart may be proved to be enlarged by means of percussion in those cases in which there is a failure in the mitral valve to close, the second pulmonic sound is accentuated, and the murmur is heard almost exclusively at the apex. It is then a question whether the mitral insufficiency is of endocardiac or *functional, relative* origin (a symptom frequently observed in anæmics)—i. e., whether the muscular apparatus of the valves is insufficient although the valves are intact. The differential diagnosis of these two varieties of mitral insufficiency is always difficult; but it is not beyond the possibilities of diagnosis, and will be discussed in the chapter relating to the diagnosis of mitral insufficiency of endocardial origin (p. 15).

Muffled and Split Heart Sounds.—Besides anæmic murmurs mention may be made here of the "*muffled*" and "*split*" *heart sounds.* It is beyond doubt that they are not infrequent; *but I consider it a mistake to attach any value to them, or even draw diagnostic conclusions from them.* As muffled sounds occur not only in hearts which, post mortem, are found to be healthy, but are also observed in anatomical changes of the valves and of the muscles of the valves, nothing is gained in diagnosis by their recognition. Only for those who, under all circumstances, want to be on the safe side, their identification is a convenient means of easing the diagnostic conscience, inasmuch as in every case, no matter whether the autopsy demonstrates a pathological condition of the valvular apparatus or not, the findings at the bedside are at least not discredited.

In the presence of the signs which may be demonstrated by physical diagnosis, and which have partly been mentioned already, or which will be referred to later on, other symptoms occur in cardiac disease, such as palpitation, pain; they are, however, of little or only of minor significance, particularly as they occur also in other patients, especially neurasthenics, without any pathological anatomical changes in the heart.

DISEASES OF THE ENDOCARDIUM

ACUTE ENDOCARDITIS

The diagnosis of *acute endocarditis,* in the majority of cases, presents great difficulties, in fact sometimes there are no objective signs; their presence can, in such instances, only be surmised with a certain degree of probability, but they must *not be diagnosticated.*

Objective Findings.—The diagnosis of endocarditis *is principally based upon the objective phenomena found in the heart.* So long as the endocarditis does not have its seat in the valves and does not influence the function of the latter, no changes in auscultation and percussion can be expected. Endocarditis cannot be diagnosticated under such conditions, even if a diffused visible impulse of the heart, or changes in frequency of the apex-beat with arrhythmia of the pulse, occur, symptoms which are to be referred to an affection of the myocardium connected with endocarditis: a serous infiltration of the myocardium or a myocarditis which frequently complicates endocarditis, as pathological examinations have proved recently. Only when the valves, which, moreover, is usually the case, are more directly implicated by the inflammatory process, can a diagnosis of endocarditis be made. Most frequently, at least in 50 per cent of the cases, the *mitral valve* is the seat of the endocardial changes, much less frequently the aorta, and only very rarely the valvular apparatus of the right heart. Then the characteristic signs of endocarditis occur, principally *murmurs,* which are *constant* in character, and the diagnostic significance of which as a means of identifying an acute endocarditis will be discussed separately in describing the special valvular defects.

Acute Mitral Insufficiency.—In the greatest majority of cases of mitral insufficiency physical signs are present. In the first place, as previously remarked, because the mitral valve is the point of election for endocardial ulceration and excrescences; furthermore, because the latter almost always lead to insufficiency of the mitral valve, immaterial whether they are located in the papillary muscles, in the chordæ tendineæ, or in the velum of the valves. As has been recently insisted upon by Romberg, conducive to their occurrence in acute endocarditis are also myocardial changes (especially at the auriculo-ventricular border of the ventricle) which develop synchronously with these endocardial changes. Exceptional cases in which, besides insufficiency, or, in fact, without such, *stenosis* of the mitral orifice occurs with its characteristic præsystolic murmur (this may be expected only provided numerous excrescences occur at the base of the velum of the valve, or if firm agglutination of the free borders is brought about, or, possibly, if a mass of fibrinous plugs enters into the auriculo-ventricular orifice), are very rare compared to the usual cases of genuine mitral insufficiency the origin of which can be easily explained by their anatomical relations. Therefore, as a symptom of acute endocarditis there is generally heard only a *systolic blowing murmur,* loudest at the apex, more rarely in the region of the pulmonary artery, furthermore, an *accentuated second*

pulmonic sound. The impulse can be felt more diffused than normal, but it is not very strong or even heaving, because the left ventricle, on account of the insufficient closure of the mitral valve, loses in volume during the so-called closing period (see p. 20), during which it has to form the impulse. *Percussion* generally shows only an *enlargement of the cardiac area to the left,* in consequence of the increased diastolic filling of the left ventricle, while, for the time being, there is no reason for an increase of the heart towards the right side. It is true, the right ventricle, owing to engorgement in the lesser circulation, works under greater resistance, but by means of its reserve force it accommodates itself to the greater demands upon its activity. Only upon long-continued increase of tension, or if, at the same time, myocardial changes are present at the onset, will the ventricle suffer in loss of elasticity, and, as a consequence, dilatation of the right ventricle, and with it the extension of cardiac dulness to the right, will not fail to present itself.

Variations from the Usual Type.—If, exceptionally, instead of the mitral, the aortic valve is the seat of the endocardial changes, the above-described signs upon auscultation will be altered in the manner characteristic of the aortic valves—i. e., in this instance there will be heard at the second intercostal space, to the right of the sternum, a diastolic murmur as a sign of acute insufficiency, and cardiac dulness will be noted extending to the left. The first sound is then clear, or another murmur is heard taking its place; the cause of the latter may be that the conditions (as explained on page 4) which give rise to an accidental murmur are developing in the course of the disease, or it may be that the endocardial changes which cause the insufficiency of the aortic valves may prevent the full development of the latter. Very rarely there is found, besides endocarditis of the left heart, an endocarditis of the right side, and with it an insufficiency of the tricuspid valve with its characteristic phenomena: dilatation of the right ventricle, a systolic murmur at the right sternal border, and the præsystolic systolic venous pulse (see later).

The diagnosis of endocarditis is *based* upon these physical signs. But it is only confirmed by a simultaneous observation of the *ætiology* of the case.

Diagnosis by Means of the Ætiology.—*Endocarditis is an essentially infectious disease,* a view which has gained more and more adherents in consequence of the latest clinical and experimental investigations. From infectious endocarditis, in which bacteria may be demonstrated in the inflammatory tissue of the endocardium, that variety of endocarditis must be excepted in which in the aged an inflammation of the intima of the aorta develops without the aid of micro-organisms, and gradually, from above downward, involves the aortic and mitral leaflets. In the other varieties of endocarditis, however, the inflammatory products, which are present in the endocardium, are due to the presence of micro-organisms. Most of the known infectious diseases may give rise to endocarditis. As the various infections constantly show the presence of well-known bacteria that are capable of producing inflammation (staphylococcus pyogenes, streptococcus, pneumococcus), the similarity in the effect of the infective products must not be looked upon as a misplacement when the micro-organism, which is specific of an infectious disease, finds entrance to the endocardium. It is certain, however, that in *exceptional instances* specific micro-organisms (gonococci, tubercle bacilli, and others, as has frequently been ob-

served recently) are capable of causing a (specific) endocarditis, provided they reach the heart by way of embolism; but this is by *no means* the rule. If, therefore, the explanation of the conspicuous ætiological part played by the infectious diseases in the causation of endocarditis is sought for, it is necessary to recur to still another of their common effects, and this, in my opinion, can only be looked for in the toxic *chemical* action of their infectious material. Through them the soil is prepared upon which other bacteria, which enter the circulation from without and then reach the heart, are enabled to exert their inflammatory action. To assume such a local predisposition to inflammation, caused by the toxic chemical effect of the infectious products, is fully justified, in view of the more recent experimental investigations regarding the origin of endocarditis (Wyssokowitsch, Ribbert, Orth, and others). According to the results obtained by them, we may draw the conclusion that the bacteria are specially enabled to display their inflammatory and destructive effect in the endocardium when they happen to find a previously weakened tissue (in our case changed by the toxic chemical effect of the infectious material). Not all infective products are equally capable of producing endocarditis. The endocardium is especially endangered by acute articular rheumatism and by septicopyæmia; endocarditis is found less frequently as a result of diphtheria, scarlatina, peliosis rheumatica, chorea; comparatively rarely in enteric fever, erysipelas, pneumonia, and gonorrhœa. In keeping with the above-mentioned fact, that a weakened condition of the tissue prepares the way for the development of endocarditis and facilitates the action of micro-organisms upon the endocardium, it may be explained that endocarditis not infrequently develops in Bright's disease and during pregnancy, and that acute relapses of endocarditis ("recurring" endocarditis) quite often occur in pre-existing valvular defects.

Differential Diagnosis.—The ætiological factors just mentioned are to be considered in the diagnosis of acute endocarditis, as they materially complement the physical signs of endocarditis. Should the ætiological basis for the assumption of a probable endocarditis be absent in a case in which the physical signs, especially cardiac murmurs are present, it is necessary that great care should be exercised before a diagnosis is made.

Differential Diagnosis between Accidental Cardiac Murmurs and Endocarditis.—In the first place, it will be necessary in such cases always to exclude *murmurs in the anatomically intact endocardium.* We have already (on page 4) discussed the points which are essential to the diagnosis of accidental cardiac murmurs. The systolic character of the murmur, which is, as a rule, soft, its diffusibility and, even more so, the absence of dilatation of the heart and the accentuation of the second pulmonary sound, and besides, the presence of anæmia, etc., are almost always in favour of the diagnosis of an *accidental* (functional) cardiac murmur. In rare instances, however, all the diagnostic signs mentioned are not sufficient for a positive opinion, so that, for a time at least, there may be a doubt as to the correct diagnosis.

Differentiation between the Murmurs caused by Endocarditis and Myocarditis.—Still greater differential difficulties than accidental cardiac murmurs are caused by those which occur in the course of *myocarditis.* It is obvious, if the myocardial changes affect the muscular fibres of the auriculo-ventricular valves or the papillary muscles, that the entrance or firmness of the valvular opening becomes insufficient and with it a systolic murmur must occur. Naturally, the consequences of this conspicuous relative mitral insufficiency will be certain to make their appearance—viz., the extension of cardiac dulness dependent on dilatation of the heart, a systolic

murmur, and accentuation of the second pulmonary sound. The differential diagnosis is always difficult in such cases. In favour of *endocarditis*, uncomplicated by myocarditis, are only a moderate dilatation of the heart, a loud murmur, constant in its intensity, a relatively strong, uniform pulse, the marked accentuation of the second pulmonic sound, and the fact that new, especially diastolic, murmurs are beginning to appear with the original systolic mitral murmur, in consequence of further endocardial changes in the valves in the course of the affection. In favour of *myocarditis*, without an accompanying endocarditis, are the weakness of the impulse and of the radial pulse which, as a rule, is small, arrhythmic and frequent, and little or not at all influenced by cardiac stimulants, the feebleness of the systolic murmur (in consequence of the reduced velocity of the blood current caused by insufficient contraction of the heart muscle), and, furthermore, the considerable extent of cardiac dulness to the left and right of the præcordial area and the only moderate accentuation of the second pulmonic sound, the intensity of which is, at least partly, dependent upon the sustained energy of the right ventricle. However, the signs mentioned are not always sufficient for a positive differential diagnosis, so that frequently we can only figure on probabilities.

Differential Diagnosis between Endocarditis and Pericarditis.—Much easier than the differentiation between exocardial and endocardial murmurs is the diagnosis between the latter and pericardial friction sounds. The pericardial sounds are, as a rule, well characterized by being superficial, by their character known as friction, and by the fact that they do not fully coincide with either systole or diastole of the heart, and that they are intensified by external pressure, so that difficulties as to the diagnosis occur but rarely. More exact information regarding the acoustic properties of the pericardial friction sounds will be given later on when discussing the diagnosis of pericarditis. It is possible in many cases that, with coexisting pericarditis and endocarditis, the murmurs caused by the latter are covered by the louder pericardial friction sounds, and do not appear plainly until the pericarditis disappears.

Differentiation between Acute Endocarditis and Chronic and Recurrent Endocarditis.—The question much more difficult to decide is whether the endocarditis that has been diagnosticated is acute or chronic, or whether an acute exacerbation has been added to a pre-existing chronic endocarditis. Of importance in the individual case is absence of fever in the chronic variety, and the presence of *marked* hypertrophy in some parts of the heart muscle. This is never absent in prolonged chronic valvular disease, whereas in acute endocarditis the change in the heart muscle is found to consist in dilatation, and, as an increased effort is required, due to the blood entering to a greater extent, hypertrophy in its first stages may also be present. It is further diagnostic of acute endocarditis that the character of the murmurs is *changeable*, they gradually increase in intensity, and new sounds occur in the course of the affection added to those already present, which are labile in character. On the other hand, in chronic endocarditis in which the valvular defect is pronounced, the murmurs are characterized by marked *constancy*. A præsystolic murmur at the apex and aortic mur-

murs are in favour of chronic endocarditis—that is, actual valvular disease. Intercurrent fever and the sudden appearance of secondary phenomena, which will be referred to later on, are conditions favouring acute endocarditis. Although the diagnosis of acute endocarditis may be based entirely upon the findings of physical diagnosis, it is materially supplemented by the simultaneous appearance of phenomena due to the endocardial process, appearing in other parts of the body.

Secondary (Metastatic) Symptoms of Endocarditis.—The fact that thrombi formed in the course of the endocardial disease are carried with the blood current, gives rise to the formation of embolic *hæmorrhagic infarcts* in a number of organs, for instance, in the brain, generally causing hemiplegia and aphasia; in the spleen, characterized by swelling and pain; in the kidneys, producing bloody urine and pain in the renal region. Hæmorrhagic pulmonary infarcts are followed by hæmoptysis, circumscribed dulness, principally in the bases of the lungs, and dyspnœa. The liver does not as frequently become the seat of emboli as do the organs mentioned; still rarer are these conditions found in the mesenteric artery. Occasionally emboli are ushered in by chills, also by sympathetic vomiting, and these symptoms, if they supervene upon an endocarditis, generally indicate the appearance of metastases. These metastatic emboli also occur in the course of chronic endocarditis. However, the observation is important that, if chronic endocarditis has persisted for some years, without emboli having made their appearance, and now suddenly embolism becomes marked, an acute recurrent endocarditis has occurred. It is obvious that with this process of embolism, in which pus-forming bacteria abound, *suppuration occurs in secondary foci*, which result in abscess formation, and purulent exudates appear in various organs of the body.

Special Forms of Endocarditis.—After the diagnosis of endocarditis has been made, it remains to be determined which *special variety of endocarditis* presents itself. As this is of some importance in regard to prognosis, it should at least be attempted in every case. We may distinguish a mild and a severe variety of endocarditis. As representing the former may be considered most forms of endocarditis occurring in the course of acute articular rheumatism; as belonging to the latter, endocarditis the result of sepsis. Endocarditis which owes its origin to other infections than those mentioned, resembles occasionally the mild rheumatic, at other times the severe septic, form. It is not strictly proper, in my opinion, thus to classify endocarditis in the one or the other category, much less so because sometimes even a rheumatic endocarditis may give rise to a most severe pathological type, simulating the septic form, and, *vice versa*, septic endocarditis may, in rare cases, take a very mild and favourable course. I believe we may establish, in general, the axiom that *it is less a question of the kind, but of the severity of the infection, whether the endocarditis takes a mild or severe course.*

Mild Form.—The *mild* form is characterized by very moderate fever, which may even be entirely absent, the general condition of health is but little influenced, and the emboli, just mentioned, occur but rarely; pulmonary infarcts are the ones most often seen.

Severe Form.—Formation of *multiple* infarcts indicates a *severer* character of the endocarditis; the most important areas of infarction have just been mentioned, and it may be that the retina, the skin, and the mucous membranes may show numerous emboli, even hæmorrhages. If the infection is of an *especially septic* character, an unusual symptom generally shows itself in the fever curve. Falling and

rapidly rising temperature accompanied by chills, fever being frequently 105° F. to 107° F. The constitutional symptoms are *unusually severe*, and delirium is present; occasionally pronounced symptoms of (purulent) meningitis and encephalitis appear. The spleen presents acute swelling, as in other infections; the urine is of the same character as in infectious nephritis; the skin shows the most various forms of exanthems: Roseola, erythema, urticaria, pock-like pustules, pemphigus bullæ, etc. At times inflammations of the joints may occur—generally characterized by one or only a few joints being affected—smallest pulmonary abscesses, accompanied by dyspnœa and bronchitis, and thus strikingly simulating the picture of acute miliary tuberculosis. Of particular significance in the diagnosis are the changes of the fundus oculi, especially retinal extravasations which, occurring with or without white centre, in dubious cases speak for the presence of sepsis (and septic endocarditis), which becomes more certain if metastatic pustules or abscesses occur in the skin. The special significance of septic "malignant" endocarditis in the picture of (cryptogenic) septicæmia will be fully discussed later on in the chapter on infectious diseases. Gonorrhœal endocarditis may also run the course of the severe malignant form.

[The symptomatology of malignant, ulcerative, or septic endocarditis is so varied that it has become usual to divide the affection into four clinical types, which are frequently distinctive. Cardiac signs are common to all varieties.

Septico-Pyæmic Type.—This form occurs during the course of septicæmia, pyæmia, the puerperal state, or in the course of any septic infection. There is marked fever (pump-handle temperature), chills, and sweating. Cutaneous manifestations are common. Nervous symptoms are frequent, as are emboli and abscesses in the skin and various organs.

Typhoid Type.—Here the course of enteric fever is closely simulated.

Cerebral Type.—Symptoms relating to the cerebro-spinal system are prominent. Frequently the cardiac phenomena are slight.

Cardiac or Malarial Type.—This is the most common variety. It usually occurs in persons already suffering from chronic endocarditis. The symptoms and physical signs in this condition call attention to the heart.]

The above remarks show sufficiently that, in the individual case, we should not be satisfied with the simple diagnosis of endocarditis, but that, under all circumstances, it should be complemented ætiologically; the decision of the question upon which basis the individual endocarditis has arisen then generally coincides with the opinion as to the mild or severe character of the affection.

CHRONIC ENDOCARDITIS—VALVULAR DISEASE

Chronic Endocarditis.—While the direct diagnosis of acute endocarditis is difficult, on the other hand, that of chronic endocarditis—that is to say, *valvular disease*—is, in by far the majority of cases, very easy. Chronic endocarditis, as a rule, is the result of a preceding acute endocarditis, more rarely it is an accompanying condition of an atheromatous process. The anatomical changes produced in the valvular apparatus due to disturbance of function are characterized as stenosis and insufficiency.

Relative Insufficiency.—These latter conditions may develop even when the valves are absolutely intact anatomically, as dilatation, which may develop from any cause, may assume such dimensions that the curtains of the mitral or tricuspid valves, even after they have gradually become tense, are not sufficient to close the opening completely. The condition is then known as *relative insufficiency*, and in rare instances, in which marked distention of the root of the aorta or pulmonary artery occurs, may also affect the semilunar valves. Another cause of relative insufficiency is the insufficient function of the muscular apparatus of the valves—i. e., of the

papillary muscles or the muscular fibres which enter into the auriculo-ventricular valves from the auricular wall. Such *insufficiencies of the valvular muscles* may be caused by myocardiac changes or by muscular weakness (in consequence of anæmia or intoxication, that is, infection).

The general sequelæ of these valvular defects have already been discussed (p. 1)—namely, inevitable lowering of the blood pressure in the arterial system, the increased pressure in the veins, the lessening of the velocity of the current in the capillaries, the cyanosis resulting therefrom, anasarca, the accumulation of fluid in the various serous cavities, the engorgement of the liver and kidneys, the venous pulse, etc.; we also referred to compensation in the heart which is able to *delay* for some time those grave consequences of the circulatory obstruction which are dependent upon valvular defects. An observation of these stasis phenomena at the bedside proves at first but a continued, severe circulatory disturbance, which, nevertheless, as a rule, is due to valvular defects. The presence of the latter and the demonstration of the special form, however, can only be determined by physical examination of the heart; it forms, therefore, the basis for the diagnosis of the individual valvular lesions.

MITRAL INSUFFICIENCY

Diffused Pulsation.—On inspection of the bared breast diffused pulsation is noted over the entire cardiac area, due to the closely underlying right ventricle which has materially increased in size, the right border of which is pressed downward, and, resting obliquely, is in its entire extent contiguous to the diaphragm and the upper surface of the liver, and in this manner it produces visible pulsation in the *epigastrium*; to a lesser extent it is also (dilatation and) hypertrophy of the left ventricle, which is responsible for the increased pulsation.

Changes in the Impulse.—The *impulse* is not seen in the fifth intercostal space inside the mamillary line, as is normal, but is observed by the eye, and even more so by the palpating finger, to be several centimetres to the left of the axillary line and outward; the apex beat may even, at least in great hypertrophy of the left ventricle, be felt downward in the sixth intercostal space. But the latter condition is not at all usual; generally the impulse is only displaced outward, but can still be felt in the fifth intercostal space. The apex beat does not seem to be essentially increased, unless it is formed by the right ventricle; often a systolic (purring) thrill may be observed on palpation in the region of the apex of the heart.

Conditions of Percussion.—*Percussion* demonstrates a distinct increase in cardiac dulness, essentially dependent upon enlargement of the right ventricle,[1] which is brought about in consequence of the mitral insufficiency.

[1] Probably there is always an excentric hypertrophy of the right ventricle which is the cause of increase in the cardiac dulness, as demonstrable by percussion, at least when it is considerable (in which case the excentrically hypertrophied left ventricle and the non-dilated hypertrophied right ventricle may cause it), if the dulness extends beyond the middle of the sternum. It is obvious that, in mitral in-

The right border-line of the cardiac dulness does not extend along the left border of the sternum, as in the normal, but crosses to the right, frequently as far as the right border of the sternum and even beyond. Neither does the upper arc of cardiac dulness commence at the cartilage of the fourth rib, as normally, but more superiorly, according to the degree of enlargement of the right heart. Cardiac dulness has also considerably extended to the left, especially when hypertrophy of the left ventricle has assumed greater dimensions.

Auscultation reveals a more or less *loud systolic murmur*, which is heard most distinctly at the apex of the heart, only rarely circumscribed at the outermost portion of the apex beat to the left; generally this murmur is heard transmitted as well to the right and upward in a wide area. Sometimes the murmur is heard loudest at the punctum maximum of the pulmonary artery, exceptionally in this area exclusively. This is easily explainable if the natural position of the ostium venosum sinistrum is thought of (from the sternal end of the third right, to the lateral end of the cartilage of the second left, rib; the middle of the orifice, therefore, in the second intercostal space to the left of the sternum), and it is also considered that the murmur must be heard best in the direction of the regurgitating blood-current.

As a rule, there is absence of tone formation accompanying the murmur. This is quite natural, as during the first stage of the systole in the so-called "closing stage," on account of the defect in the closure of the mitral valve, the parts of the latter and of the ventricle which are able to vibrate, do not, as normally, tend towards a new centre of gravity and, in consequence of their inertia, swing around the latter, but assume it only gradually, do not go beyond it and, accordingly, do not vibrate sufficiently for the formation of a musical tone (R. Geigel). Consequently, there is no period of closure in the case of mitral insufficiency, and the formation of tone does not take place. The *systolic murmur* is brought about by the fact that the blood at the time of contraction of the ventricle enters through the open auriculo-ventricular space into the relatively wide auricle and thereby causes vibrations of those parts (bordering on the blood column) which are concerned in it. As, furthermore, the intensity of the murmur thus created depends upon the velocity of the blood current, it is obvious that the murmur is often only heard after exercise or in certain attitudes of the patient, in general only after the heart has displayed greater activity.

Pulmonary Artery.—Besides the changes in the heart, those in the great vessels are to be determined. The initial portion of the *pulmonary artery* shows a visible *systolic pulsation* in the second left intercostal space; when it is distended in consequence of regurgitation of the blood, it pushes the

---

sufficiency, the right ventricle hypertrophies according to the increased resistance in the pulmonary circulation, and that primarily *without dilatation* (Riegel). The latter assumption is undoubtedly correct; but if it is considered that, even in physiological conditions with increasing resistance besides the increased labour, very soon a decrease in the systolic diminution of the volume of the heart also becomes evident (consequently an abnormally large quantity of blood remains in the ventricle after the systole), and, besides, as the latest observations of Krehl and others teach, that in valvular changes quite usually the myocardium is also affected by (mostly infectious) noxæ, it is not astonishing that, besides the hypertrophy, in most cases there will also occur very early a dilatation of the right ventricle.

border of the lung aside, and thus becomes contiguous to the thoracic wall. The *diastolic closure of the valve* at the pulmonary orifice also becomes *visible and palpable* under these circumstances. This is of importance in the differential diagnosis between accidental (functional) and organic murmurs, but, as a rule, it is rare; on the other hand, the *accentuation of the second pulmonary sound,* heard upon auscultation, is almost pathognomonic, a fact to which Skoda was the first to call attention. It is due to the fact that the increase in pressure in the pulmonary circulation—i. e., in the pulmonary veins, capillaries, and, in connection with it, the pulmonary artery, reaches a high grade, which again causes increased activity of the right ventricle. At the beginning of the diastole in the (right) ventricle the pressure in the latter falls rapidly. The great difference between this and the high pressure in the pulmonary artery will manifest itself by the fact that the semilunar valves during this time are driven with great force against the ventricle and vibrate more powerfully than normally around the new centre of gravity towards which they tend very rapidly. The force of the second pulmonary sound, therefore, depends, in the first place, upon the intensity of the engorgement in the lesser circulation—i. e., upon the degree of the mitral insufficiency; in the second place, also upon the energy of the right ventricle, which was consecutively stimulated to greater activity. The latter hypertrophies, as we have seen, primarily without dilatation; later this occurs with hypertrophy, as soon as there is an increase in the amount of labour to be performed by the heart. If, in the further course of the affection, compensation fails which had been created by the hypertrophy of the heart, a passive distention of the right ventricle occurs, and with it, as higher grades of dilatation occur, a relative insufficiency of the tricuspid valve takes place. The consequence is, that there is a lessening of force of the second pulmonary sound, in contradistinction to its loud accentuation before the occurrence of that complication.

Pulse Curve.—It is easily understood that the *radial pulse, as a rule, does not show material deviations from the normal* (see Fig. 1*a*, on p. 19). As long as the compensation remains perfect, and particularly the left ventricle hypertrophied (excentrically)—inasmuch as the blood current flows under abnormally high pressure from the dilated and hypertrophied left auricle through the ostium venosum sinistrum into the left ventricle—the aortic system will still be filled in an approximately normal manner during the systole, in spite of the regurgitation of the blood into the left auricle. The pulse will therefore show an almost normal curve—that is, the pulse wave will suffer but little in size, in tension the pulse will be slightly diminished, provided the hypertrophy of the left ventricle does not compensate completely, which will be shown in a more decided recoil elevation and a diminution of the elasticity rise of the sphygmogram. In the majority of cases in which treatment is necessary, however, compensation is disturbed to a marked degree, and the pulse is irregular and small. This latter quality is principally found in the pulse of those cases of mitral insufficiency which are complicated by mitral stenosis (comp. pulse curve, Fig. 1*b*, p. 19).

Of the indirect *sequelæ* of mitral insufficiency mention, above all, must be made of the relatively frequent formation of *hæmorrhagic infarcts in the lungs.*

Differential Diagnosis.—The diagnosis of mitral insufficiency, this most frequent of all valvular defects, is generally easy, provided the physical signs that have been discussed are observed.

In the *differential diagnosis* the *functional systolic murmurs* are to be primarily taken into consideration. Their differentiation has already been enlarged upon; it need only be repeated here that the functional murmurs show great changes in intensity, do not occur with coincident enlargement of cardiac dulness and accentuation of the second pulmonary sound, and are generally heard best at the pulmonary orifice. The differential diagnosis of *relative mitral insufficiency* is more difficult (see p. 5). Practically, there is principally to be considered the cardiac hypertrophy of Bright's disease, fatty heart, myocarditis, idiopathic enlargement of the heart, and relative insufficiency in anæmic conditions and toxæmia. It is presupposed in all these affections that the relative incompetency is due to relaxation and stretching of the left ventricle (degeneration), which gives rise to a systolic murmur. These cases of relative mitral insufficiency may generally be distinguished from the organic variety. The co-existing disease must be taken into consideration, with the resulting changes in the heart, in each individual case. A fact most worthy of note is that the murmur of relative mitral insufficiency, as well as the functional murmurs, show decided *changes of intensity*, and in contrast to the organic murmur rather become weaker than more pronounced by energetic cardiac action, and may even disappear entirely if the heart is "whipped up," especially by digitalis, always provided that no degeneration of the heart muscle be present which may not be overcome. At the same time the heart beat is weak, the accentuation of the second pulmonary sound but moderate, the pulse small, and, eventually, irregular. Some cases of relative mitral insufficiency cannot be differentiated from true (that is, not combined with mitral stenosis) endocarditic mitral insufficiency, especially when chronic endocarditic mitral insufficiency has led to loss of compensation. However, cases of chronic "genuine" endocarditic mitral insufficiency are rare, upon the whole, as generally there is a stenosis of the mitral orifice combined with the mitral insufficiency, and with it appears also, besides the systolic, a præsystolic murmur—in short, the symptoms of the last-named valvular defect will make themselves prominent besides—to the analysis of which the following chapter is devoted.

STENOSIS OF THE MITRAL VALVE

Stenosis of the mitral orifice is in most cases combined with insufficiency. The, previously described, anatomical and clinical consequences of insufficiency are therefore almost regularly present also in stenosis of the mitral valve, but are essentially modified by the stenosis, so that the diagnosis of the latter, in spite of the synchronous insufficiency of the valve, is generally easily and definitely ascertained. Pure stenosis without insufficiency may occur when the valves without shrinking are funnel-shaped and coalescent from the base. But the proof that in these cases (exceedingly rare in comparison to the enormous majority of cases in which the stenosis is unquestionably combined with insufficiency) a complete closure of the valves is still possible, can but rarely be furnished with

certainty, even post mortem. Therefore, the diagnosis is to be relegated to the question in the great majority of cases, *What clinical phenomena entitle us to assume, besides insufficiency of the mitral valve, a synchronous stenosis of the orifice, and which of both these valvular defects predominates in the individual case?*

A simple consideration will show that the usually very material obstacle which impedes the inflow of blood from the left auricle into the left ventricle by the stenosis, is bound, under all circumstances, to cause a disturbance of the circulation, inasmuch as a powerful engorgement of the blood in the left auricle and further back in the lesser circulation and in the right heart occurs as the necessary consequence of every stenosis of the left auriculo-ventricular orifice. We find, accordingly, the right ventricle much hypertrophied, and, very soon, also dilated (see p. 12, foot-note), while in contrast to this the left ventricle and the aorta appear to be filled less, in proportion as the stenosis is more developed.

Inspection and Palpation.—Upon *inspection* there is observed diffused pulsation, the apex-beat, it is true, displaced far outward even to the left axillary line, but less downward than in pure insufficiency, because the hypertrophy of the left ventricle, which forms in the latter disease, is prevented from developing by the accompanying stenosis, according to the preponderance of the stenosis in part or entirely. In pure mitral stenosis a concentric atrophy of the left ventricle occurs, unless the left auricle interferes by compensating fully, which will be the case only at the onset of the stenosis, and then in not too marked grades of this valvular defect. Inasmuch as, in such cases, during the ventricular diastole never more than a full "beat volume"—i. e., the quantity of blood which is to be normally expelled by the ventricular systole—reaches the ventricle, the left ventricle adapts itself in activity and size to the lesser filling, so that it is attached to an enormously enlarged right heart as a relatively small appendix, as is often seen in a most striking manner at autopsies. Besides, marked epigastric pulsation occurs, a sign which at once indicates dilatation of the right ventricle. *Palpation* generally shows a weaker apex beat, a stronger heart-beat only if the latter is formed by the hypertrophied right ventricle; we generally feel, at the same time, a strong "*frémissement cataire*" (purring thrill), which varies from the tremor in pure insufficiency, inasmuch as it commences before the systole and is very jerky, conditions which are also present during auscultation, and which will be further commented on.

Percussion shows considerable enlargement of the right heart, the diffusion of cardiac dulness is found at its maximum to the right.

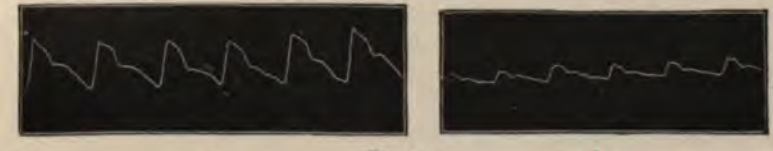
Auscultation.—Upon *auscultation* there is heard at the apex of the heart a murmur which varies in different cases. The murmur is, comparatively rarely, short, simply *diastolic*—i. e., a murmur which sets in directly at the beginning of the diastole of the ventricle, when the pressure of engorgement in the lesser circulation is so considerable that the blood is driven, at the beginning of the diastole, from the left auricle, which acts under high tension, and with great force into the relaxed ventricle. As a rule, this murmur does not make its appearance until the second part of

of insufficient function of the left ventricle combined with the insufficient filling of the coronary arteries. (Compare Fig. 1, *a* and *b*.)

In cases of genuine mitral stenosis in which compensation has taken place (not complicated by mitral insufficiency), von Noorden has observed even an abnormally *high* tension of the pulse with very pronounced elasticity vibrations.

Heart-Failure Cells.—In the sputum of patients who suffer from mitral stenosis, there are found, comparatively regularly, so-called *heart-failure cells*—i. e., large, mostly oval cells with more or less pronounced nucleus, in which is found either diffused or granular yellow or yellowish-brown pigment (a derivative of the blood pigment). The heart-failure cells are not pathognomonic of mitral stenosis, although they occur most frequently in this affection of the heart. They are solely the result of great engorgement in the lesser circulation (in brown induration of the lungs), especially in mitral disease, myocarditis, etc., and, besides in patients with valvular disease, they are occasionally found in the sputum of patients suffering from emphysema, and also in persons suffering from croupous pneumonia.

If the symptoms consequent upon stenosis of the mitral orifice, which have been enumerated, are developed, the presence of a stenosis may be considered, even if no diastolic, but a distinctly systolic murmur can be heard in the heart. It must not be lost sight of that mitral stenosis *under*



a. FIG. 1. *b.*

CASE OF UNCOMPLICATED COMPENSATED MITRAL INSUFFICIENCY. CASE OF MITRAL INSUFFICIENCY COMPLICATED BY MITRAL STENOSIS.

certain circumstances does not produce murmurs, and, on the other hand, is a very frequent complication of mitral insufficiency. I would therefore advise every one to be very careful when making a *diagnosis of a simple mitral insufficiency*, and to adopt the rule *only to make it when the simultaneous presence of a stenosis can be positively excluded according to what has just been said.*

INSUFFICIENCY OF THE AORTIC VALVES

This valvular defect, the next in frequency to mitral insufficiency, is recognised by such characteristic symptoms that the diagnosis can be made with almost absolute certainty.

Inspection and Palpation of the Cardiac Region.—Inspection shows very remarkable changes: The marked arching of the præcordial area, especially extending towards the left, which indicates hypertrophy of the left ventricle, also diffused pulsation of the thoracic wall, which extends to the left axillary line. The apex beat is markedly "heaving" in character, and is noticeable in the sixth or seventh intercostal space or still farther down, displaced towards the axillary line and outward. At times it is distinctly *diastolic*; the cause is to be looked for in various directions. It must be surmised theoretically that there is an absence of closing

ventricle, a slight mitral stenosis, the mitral valves slightly shortened besides. Slight hypertrophy of the right ventricle. *Pulmonary tissue very pale.*

Obviously the mitral stenosis was so insignificant in this case that the blood passed through the relatively wide orifice without producing a murmur, and, furthermore, no engorgement worth mentioning occurred in the lungs (pulmonary tissue quite anæmic); the second pulmonary sound, therefore, could be but slightly accentuated. But even the existing slight accentuation of the second pulmonary sound could not be diagnosticated on account of the marked accentuation of the second aortic sound. The latter, again, was caused by hypertrophy of the left ventricle, which was brought about as a result of the nephritis, and upon this latter condition depended also the strong, hard radial pulse, the character of which was, consequently, diametrically opposed to the character of the pulse which is usual in mitral stenosis.

Resulting Phenomena in the Circulatory Apparatus.—In my experience, the question whether mitral insufficiency and mitral stenosis can be distinguished when they occur conjointly can be decided not so much by the character of the murmur, but better even by phenomena resulting from mitral stenosis. Primarily, it is important to observe the character of the second pulmonary sound.

Pulmonary Artery.—As a result of the continued stasis of the lesser circulation, which occurs as a consequence of this valvular defect and of the marked hypertrophy of the right ventricle, the second pulmonary sound is loud, "booming" in character, and all the more noticeable as the second aortic sound is feeble in comparison, due to lack of blood in the aorta. In keeping with the powerful force of the blood in the pulmonary artery striking against the closed semilunar valves, the thoracic wall vibrates, and a visible and perceptible diastolic shock is noted in the second left intercostal space.

On the other hand, a decrease in the intensity of the second pulmonary sound occurs, provided the right ventricle relaxes, or if, finally, in consequence of the enormous engorgement in the right ventricle, a consecutive relative insufficiency of the tricuspid valve is formed, which opens an outlet to the blood of the right ventricle into the right auricle. Over the carotid and subclavian arteries there are more often found, as observed by Matterstock, murmurs which are systolic in time. They occur more frequently than in mitral insufficiency. Their origin has as yet not been satisfactorily explained. The *division of the second pulmonary sound*, which not rarely occurs, is of a certain diagnostic significance. Geigel, Sr., has offered as an explanation of this division that, owing to the non-synchronous closure of the semilunar valves in mitral stenosis, the overdilated, less elastic pulmonary artery retracts a moment later than does the poorly filled aorta.

Condition of the Pulse.—The condition of the radial pulse is of no less importance in the diagnosis of mitral stenosis than the disproportionately marked accentuation of the second pulmonary sound. The pulse wave, in contradistinction to that in uncomplicated mitral insufficiency, is remarkably small; the lessened tension in the arterial system shows itself, furthermore, in the greater prominence of the back-stroke elevation and the poor development of the elasticity rise. Besides, the pulse is frequent and, more than in other valvular defects, irregular on account

murs arising at the aortic valves, is probably found in the downward direction of the blood-current whirls which are caused by insufficiency of the semilunar valves. Moreover, the murmur, at the apex of the heart proper, is generally only noticeable as a faint diastolic whisper. Sometimes there may be heard, besides the diastolic murmur, a diastolic *sound,* which probably corresponds to the transmitted second sound of the pulmonary artery. During the *systole* there is heard, according as a closing period occurs or not, at the apex of the heart *the first sound,* which is pure, or, in place of it, sometimes a *systolic murmur.* The latter may be due to various causes, which will be further discussed when entering upon the differential diagnosis.

[In some cases a præsystolic murmur at the apex of the heart (the Flint murmur) is also noted. There is still great controversy regarding its cause, but there is no doubt that it is occasionally noted.]

In no other valvular defects of the heart is it less advisable to restrict one's self, from a diagnostic point of view, solely to the examination of the heart. There are in aortic insufficiency other conditions not affecting the heart proper which present some of the best points supporting the diagnosis. This is especially the case if it occurs in youthful individuals.

Changes in the Vessels. Pulse.—The arteries are lengthened; they appear as if their course were on the surface, like that of the brachial and radial arteries, they show a tortuous formation which can be explained as the visible expression of a decrease of their elasticity. Even small arterial branches—the arteries of the fingers, the tibialis postica, posterior to the inner malleolus, the dorsalis pedis, and others—pulsate visibly on account of the blood being thrown into the arteries with great force. On feeling the pulse it is easily recognised as *the pulsus altus et celer* (Fig. 2).

During the diastole of the left ventricle the blood not only flows from the auricle, but also, in consequence of the insufficiency of the aortic valve, a part of the blood regurgitates from the aorta into the ventricle. It is obvious that thus the volume of the left ventricle is increased and the myocardium has to perform a permanently increased amount of work. The pulse, therefore, must rise rapidly, to fall again as quickly in the diastole, on account of the regurgitation, not only in the capillaries, but also due to the insufficient aortic valves not giving a base of support to the blood current; the pulse, therefore, is bounding, *leaping—celer* in character. [This pulse is also known as the *Corrigan,* water-hammer, and collapsing pulse.]

The changes of the pulse are best expressed in the sphygmographic picture. The steep rise of the ascension line is the consequence of the increase in the pulsation volume—i. e., of the amount of blood thrown into the arteries during the systole. As then, furthermore, the outflow of the blood, not only to the peripheral circulation, but also to the centre, takes place very rapidly, the descension line shows a steep declivity in its initial part, the top of the curve, therefore, is decidedly *pointed.* At the catacrotic leg of the pulse picture there is shown at first at the acme, in some of the cases, a prong, which may be explained as the first elasticity elevation of the much-strained muscular wall; then follows the back-stroke elevation, and, finally, at the bottom, several faintly expressed elasticity

period,[1] and with it the arching of the ventricular wall with systole, causing the apex beat. Furthermore, the blood, which enters the left ventricle under great pressure during the diastole, will force the heart downward, and this locomotion is enhanced by the contraction of the auricle, which occurs in the second half of the diastole. *In the vast majority of cases, however, the heart beat is systolic,* inasmuch as the ventricle, during the closing period, in spite of the aortic orifice being open, is not able to overcome the high pressure in the aorta, and therefore contracts (the same as in the normal with complete closure of the semilunar valves) at the onset of the systole, thus producing a systolic heart beat. Over the base of the heart a diastolic tremor is sometimes felt.

Percussion.—Cardiac dulness, coinciding with hypertrophy and dilatation of the left auricle, is increased particularly towards the left. It begins high up and extends towards the axillary line, occasionally overlapping the visible apex beat towards the left side, and reaches towards the right, extending over the left sternal line; but the diffusion of the cardiac dulness on this side is always comparatively limited in proportion to the extension of the heart to the left. If the initial portion of the aorta and its arch is much dilated in its subsequent course, this is shown by dulness over and to the right of the manubrium sterni.

Auscultation.—Characteristic of this valvular defect is a *diastolic murmur* heard in the auscultatory area of the aorta or in the immediate vicinity of the same. The nature of this murmur is generally that of a long-continued rustling. Variations occur, since the murmurs sometimes appear to be more harsh, at other times, relatively more frequent, so delicate and faint that they are noticed only upon the most painstaking care.[2] These variations in the character of the murmur are of very inferior significance from a diagnostic standpoint; more important is the locality at which the murmur is heard most distinctly. The murmur is generally noticed *loudest over the upper part of the corpus sterni near the left sternal border*; the reason why it does not appear strongest in the second intercostal space to the right [the punctum maximum], the same as other mur-

[1] According to Martius, by "closing period" is meant that part of the systole in which both the auriculo-ventricular and the semilunar valves are closed. It coincides, regarding time, with the ascension of the cardiogram, commences with the closure of the venous valves, and ceases with the opening of the arterial valves (the highest point of the cardiogram) and which is then followed by the second part of the systole, the "*time of expulsion*" (corresponding to the first part of the descending line of the cardiogram).

[2] In rare cases the murmur may be *entirely* absent, thus at the onset of aortic insufficiency, while other symptoms of the latter, for example, pulsation of the spleen (Gerhardt), are already plainly present. In some instances a considerable decrease in the blood pressure and in the velocity of the blood current is the cause of the absence of the murmur, the intensity of which in general depends upon the velocity of the blood current; this is principally the case in the aged, in whom the elasticity of the arterial wall has disappeared, and, with it, the diastolic contraction of the arteries is gradually lost, so that only very little blood regurgitates into the ventricle through the, in some cases generally very wide, orifice of the aorta. It may also occur that the murmur is absent during rest, but appears plainly as soon as the patient exerts himself to vigorous movement with his body, and the blood pressure is thus increased.

murs arising at the aortic valves, is probably found in the downward direction of the blood-current whirls which are caused by insufficiency of the semilunar valves. Moreover, the murmur, at the apex of the heart proper, is generally only noticeable as a faint diastolic whisper. Sometimes there may be heard, besides the diastolic murmur, a diastolic *sound,* which probably corresponds to the transmitted second sound of the pulmonary artery. During the *systole* there is heard, according as a closing period occurs or not, at the apex of the heart *the first sound,* which is pure, or, in place of it, sometimes a *systolic murmur.* The latter may be due to various causes, which will be further discussed when entering upon the differential diagnosis.

[In some cases a præsystolic murmur at the apex of the heart (the Flint murmur) is also noted. There is still great controversy regarding its cause, but there is no doubt that it is occasionally noted.]

In no other valvular defects of the heart is it less advisable to restrict one's self, from a diagnostic point of view, solely to the examination of the heart. There are in aortic insufficiency other conditions not affecting the heart proper which present some of the best points supporting the diagnosis. This is especially the case if it occurs in youthful individuals.

Changes in the Vessels. Pulse.—The arteries are lengthened; they appear as if their course were on the surface, like that of the brachial and radial arteries, they show a tortuous formation which can be explained as the visible expression of a decrease of their elasticity. Even small arterial branches—the arteries of the fingers, the tibialis postica, posterior to the inner malleolus, the dorsalis pedis, and others—pulsate visibly on account of the blood being thrown into the arteries with great force. On feeling the pulse it is easily recognised as *the pulsus altus et celer* (Fig. 2).

During the diastole of the left ventricle the blood not only flows from the auricle, but also, in consequence of the insufficiency of the aortic valve, a part of the blood regurgitates from the aorta into the ventricle. It is obvious that thus the volume of the left ventricle is increased and the myocardium has to perform a permanently increased amount of work. The pulse, therefore, must rise rapidly, to fall again as quickly in the diastole, on account of the regurgitation, not only in the capillaries, but also due to the insufficient aortic valves not giving a base of support to the blood current; the pulse, therefore, is bounding, *leaping—celer* in character. [This pulse is also known as the *Corrigan,* water-hammer, and collapsing pulse.]

The changes of the pulse are best expressed in the sphygmographic picture. The steep rise of the ascension line is the consequence of the increase in the pulsation volume—i. e., of the amount of blood thrown into the arteries during the systole. As then, furthermore, the outflow of the blood, not only to the peripheral circulation, but also to the centre, takes place very rapidly, the descension line shows a steep declivity in its initial part, the top of the curve, therefore, is decidedly *pointed.* At the catacrotic leg of the pulse picture there is shown at first at the acme, in some of the cases, a prong, which may be explained as the first elasticity elevation of the much-strained muscular wall; then follows the back-stroke elevation, and, finally, at the bottom, several faintly expressed elasticity

vibrations. It is of especial importance that, in spite of the height of the curve, the back-stroke elevation is but very poorly developed (see Fig. 2) naturally, because it is normally brought about, according to the almost universal opinion, by the closing of the aortic valves, and, consequently, must be missing in insufficiency of the semilunar sacs.

Preservation of the Back-Stroke Elevation.—If the back-stroke elevation is, nevertheless, indicated, this is caused by the fact that the rebound of the blood

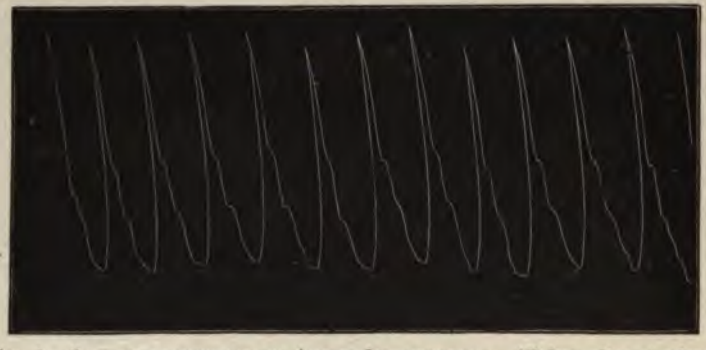


FIG. 2.—CASE OF UNCOMPLICATED AORTIC INSUFFICIENCY. (*Pulsus altus et celer.*)

from the ventricular wall and from the valves, although they are not able to close, or from the valvular remnants, will still allow the formation of a small back-stroke wave. The back-stroke elevation is relatively strongly expressed in coexisting mitral insufficiency, inasmuch as the larger quantity of blood which, under these circumstances, flows from the left ventricle during the diastole, offers sufficient resistance to the blood stream returning from the aorta, which causes greater reflection and with it the back-stroke elevation.

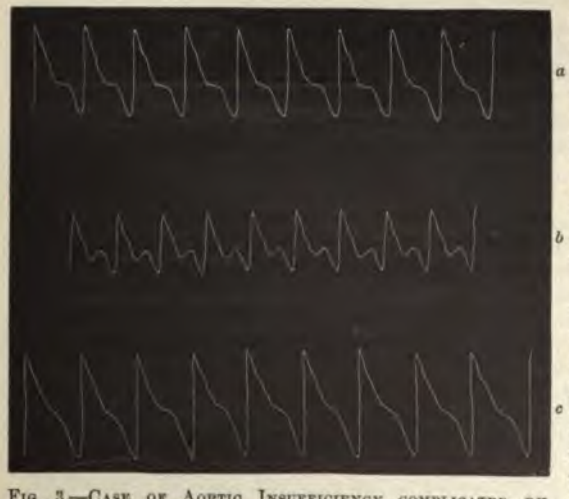


FIG. 3.—CASE OF AORTIC INSUFFICIENCY COMPLICATED BY MITRAL INSUFFICIENCY: *a*, pulse picture of aortic insufficiency before, *b*, during, *c*, after the complication by mitral insufficiency.

In Combination with Endocarditis of the Mitral Valve.—The following case, which was observed at my clinic and analyzed by R. Geigel, may serve as an example:

A girl, aged twenty years, suffered from aortic insufficiency with the usual accompanying symptoms, among which was also the receding back-stroke elevation in the sphygmographic picture. Recrudescence of the endocarditis with formation of an acute mitral insufficiency, during the course of which the tension of the blood column became lower and a back-stroke elevation appeared. After the mitral insufficiency had become quiescent, so that at last aortic insufficiency became prominent once more, the back-stroke elevation receded again. The above curves (Fig. 3, *a*, *b*, *c*) illustrate this in the most pro-

nounced manner. I also saw, in a combination of aortic insufficiency with *relative* mitral insufficiency, the back-stroke elevation most distinctly; it became indistinct when the systolic murmur at the apex disappeared after the administration of digitalis.

The peculiar conduct of the *descension line* may, after what has been said, be regarded in general as a characteristic sign of the existence of an aortic insufficiency; but it should not be lost sight of that the pulse picture is characteristic only if the aortic insufficiency is uncomplicated, if the myocardium functionates vigorously, and if simultaneously there is no considerable amount of atheroma, as the latter prevents rapid contraction of the artery and makes the top of the curve appear less pointed in this instance.

The pulse of other arteries is no less remarkable: thus, on the crural artery a jerking beat is felt with every systole; at the carotid arteries there is seen, from a distance, a marked pulsation and leaping, and even in the finest arterial branches the pulsus celer is visible and palpable; in the uvula, under the finger nails, upon the (artificially flushed) skin or on the laterally compressed lips, the *capillary* pulse appears very plainly, and also upon ophthalmoscopic examination of the retinal vessels; *liver* and *spleen* may eventually also pulsate [Quincke's pulse]. None of these symptoms, however, is pathognomonic of aortic insufficiency, as they all occur under different conditions, although less pronounced.

Auscultation of the Arteries.—Very characteristic changes can also be noted upon *auscultation of the peripheral arteries*. While, under normal circumstances, audible vascular sounds can, with lightly applied stethoscope, be observed only at the aorta, the carotid and subclavian arteries, in distinct aortic insufficiency the brachial, the radial, the crural, and the popliteal—in fact, still smaller arteries, show this phenomenon. However, even this systolic cardiac sound formation is not absolutely demonstrative, because it may, at least at the crural arteries, be observed also in other, especially febrile and anæmic, conditions. Yet, under all circumstances, the sound formation in these affections is weaker than in aortic insufficiency in which the conditions are the most favourable for tone production of the arteries. The elastic arterial wall may vibrate and sound upon sudden expansion as well as sudden contraction. It is true, under normal conditions the narrowing of the artery occurs too slowly to produce a diastolic heart sound; entirely different, however, is the case in aortic insufficiency. Not only is the wall even of smaller arteries, remote from the heart, made to produce sound by the strong blood current which is rushed with great force by the hypertrophied left ventricle, but even during the diastole (because the blood can flow to the periphery *and* to the heart) the vessel experiences so rapid a relaxation and contraction that the vascular wall now, too, i. e., during the diastole—suddenly assumes another centre of gravity around which it oscillates. Thus a systolic and diastolic sound, a so-called "double tone," is produced, which is most distinctly noticed in the crural, but sometimes also in the other arteries.

Crural Double Sound and Crural Double Murmur.—This has been heard partly in such a manner that the first sound coincided with the systole, the other with the diastole of the heart, and partly so that both sounds followed each other rapidly, succeeded by a longer interval (occurring during the diastole of the heart). As has been remarked, the most probable cause of the diastolic bruit in the former case is to be looked for in the sudden contraction of the artery following the distention (Traube). Several explanations are possible in the latter case, and I shall mention at least one of them. On account of the permanent opening of the aorta it is possible for the contraction of the left ventricle, especially if the pressure in the

aorta is small in the individual case, to push a blood wave into the aorta, which is followed by the larger wave produced by the contraction of the left ventricle. This will be represented as an anacrotism in the sphygmogram, and upon auscultation of the crural artery as a double sound which ought to be designated as (cardio-) præ-systolic-systolic.

If the double sound is to be heard upon auscultation of the crural artery, it is necessary to apply the stethoscope to the vessel without pressure. For, if the instrument is pressed even moderately upon the crural artery, one will hear, instead of sounds, murmurs—stenosis murmurs. At first one murmur, having the time of the systole, caused by the fact that the blood current, thrown with the systole into the artery, is thereby forced to pass through the artificially created narrow part of the arterial vessel; if the stethoscope is left pressed down, the systolic murmur will, for the same reason, be followed by a diastolic murmur, if the centripetally moving blood wave is developed sufficiently strong, as may be presumed as certain, in the crural artery in aortic valvular insufficiency. The presence of a double murmur in the crural artery, therefore, is of significance in the diagnosis of aortic valvular insufficiency (Duroziez). However, this murmur is sometimes, although less pronounced, observed in other conditions also.

At the carotid artery also there is heard, upon a light application of the stethoscope, a *diastolic heart murmur*. But the origin of this latter murmur has an entirely different causation: it is simply the murmur conducted from the insufficient aortic valves, which may be entirely absent if the conduction is bad.

Relative Insufficiency of the Aortic Valves.—Similarly as in other valves, a *relative* insufficiency may occur in the aortic valves, although this is rarer, inasmuch as the anatomically unchanged valves upon excessive distention of the aortic orifice are no longer able to close the same.[1] The occurrence of such relative aortic insufficiencies has been positively demonstrated by autopsies; it has been principally observed in the course of interstitial nephritis as the result of dilatation of the left ventricle and the root of the aorta.

Differentio-Diagnostical Points of View.—Again, to sum up the symptoms most essential to a diagnosis of insufficiency of the aortic valves, the *loud diastolic murmur* over the sternum is the most important of the diagnostic signs, without the presence of which the diagnosis must not, and, *vice versa*, upon its presence the diagnosis must, be made, even if other symptoms of the valvular defect are absent.[2] But the diagnosis is rendered more complete by the subsequent symptoms, which are particularly pronounced in this valvular defect: The hypertrophy and dilatation of the left ventricle, the pulsus altus et celer, the marked pulsation, and the murmur in the arteries, the double sound or double murmur at the crural artery, etc. If, besides the diastolic, a *systolic* murmur is also

[1] Still another cause of relative insufficiency of the aortic valves has been observed in my clinic, and was described by Fütterer: A tear of the aortic wall with formation of an ectasis, a kind of a "spurious pouch" which placed itself between the intact aortic valves and put one of them out of function.

[2] The presence of a *diastolic* murmur is, in rare cases, not the result of aortic insufficiency, but of an *accidental* nature. It has, in this instance, its origin in the *veins* adjacent to the heart and occurs especially in anæmics. It is to be heard either conducted from the inferior vena cava, at the inferior part of the sternum, or originating in the interior jugular vein more superiorly; in the latter case its intensity increases towards the clavicle. It can be readily observed that it is nothing but a venous (nun's) murmur, because upon pressure on the jugular veins it is modified in intensity or disappears entirely.

found in the heart, the diagnosis may become extremely difficult, because the latter may, as stated before, be due to a number of very different causes. In the first place, a coexisting stenosis of the aortic orifice is to be considered which, in fact, appears often enough associated with insufficiency. It is to be diagnosticated, besides the latter, if the systolic murmur is heard over the entire heart, yet most distinctly over the aortic orifice, if a systolic tremor is felt at the base of the heart, if the pulse is remarkably low in consideration of an insufficiency of the aortic valves, and if murmurs in the arteries are not present. Even when the loud systolic murmur is heard strongest at the base of the heart, caution in the diagnosis is necessary, as cases have been observed, nevertheless, in which no changes in the valves were found post mortem which could have been held responsible for the assumption of stenosis. In fact, it is probable that the normally wide orifice, in view of the large quantity of blood thrown with great force into the aorta, plays the part of a "physiological stenosis"; moreover, there will be another cause for the formation of a murmur if the orifice of the aorta dilates gradually, for which fact there is plenty of opportunity in aortic insufficiency by the material increase in the systolic volume of the beat. At any rate, in these cases, in contradistinction to the association of aortic insufficiency with stenosis, the condition of the pulse, characteristic of the former affection, as well as the murmur of the arteries continue to exist. But the possibilities of the origin of a systolic heart murmur are not exhausted with the above description. Aside from a simultaneous *mitral insufficiency, caused by endocarditis* (not a rare combination), it is possible that the papillary muscles of the dilated ventricle become flattened by the pressure to which they are exposed, so that their function is impaired in the course of time; besides, the same as in dilatation and hypertrophy of the left ventricle from other causes, here also the mitral valve may become *relatively insufficient* in the long run. The systolic murmur, under these circumstances, becomes soft, in contradistinction to that caused by a complicating stenosis of the aortic orifice. It is especially audible at the apex of the heart, the second pulmonary sound is intensified, and secondary hypertrophy and dilatation of the right ventricle will not fail to appear. The diagnosis of the origin of the systolic murmur, in such cases, is not difficult with a little attention, less so, if gradually a developing stasis and relative insufficiency of the tricuspid valve come into prominence.

Albuminuria may also occur as a consequence of the engorgement. The urine becomes dark, scanty, saline, will show much sediment, and it contains moderate amounts of albumin and hyaline casts. This albuminuria, however, coinciding with the changes named in the condition of the urine (stasis urine), is to be differentiated from an albuminuria which, in my experience, is not rare in insufficiency of the aortic valves, which is caused by nephritis complicating this heart lesion even during the time of complete compensation. In such instances the urine presents the characteristics of the urine of nephritis, particularly that of interstitial nephritis—i. e., it is copious, more or less clear, contains granular, besides hyaline, casts; neither does the amount of albumin vary as much as in stasis urine, in which the quantity of albumin depends upon the existing energy and activity of the heart.

Subsequent Symptoms of Hypertrophy of the Left Ventricle.—The cardiac lesion

in insufficiency of the aortic valves, in contrast to other valvular defects, makes itself felt in a manner very annoying to the patient: by the dilatation and hypertrophy of the left ventricle, even in the stage of full compensation; by heat and pressure in the head, pulsations, headache, and vertigo, in the worst cases by rupture of the cerebral arteries, which are then mostly atheromatous, with its sequelæ. It is necessary in the diagnosis of aortic insufficiency incidentally to take into consideration the last-named symptoms.

STENOSIS OF THE AORTIC ORIFICE

Much plainer than the conditions in insufficiency of the aortic valves are the signs to be considered in the diagnosis *of stenosis of the aortic orifice.*

Hypertrophy of the Heart—Apex Beat.—The necessary consequence of this cardiac lesion is *hypertrophy of the left ventricle*; but the size of the heart is less marked in these instances, because in the genuine cases at first nothing but a concentric hypertrophy develops, and only gradually a dilatation of the left ventricle, which, however, is not as extensive as the hypertrophy. So soon as stenosis is accompanied with insufficiency (as is often the case), greater dilatation combines with the hypertrophy, and then the size of the left heart becomes enormous.

The apex beat is palpable slightly downward and outward, less diffused, and generally not as strong as could be expected in consideration of the presence of a hypertrophy of the heart, in fact, in a number of cases it is entirely absent, as was emphasized by Traube. This is caused by the fact, as long as the heart acts vigorously up to a certain degree, that in stenosis of the aorta during the closing period of the valves, during which normally the heart beat occurs, the pressure in the aorta is relatively low, consequently the semilunar valves are able to open at the first onset of the systole, and the ventricle therefore empties itself from the beginning. Accordingly, there is no closing period and with it no apex beat; later, it is also possible that the insufficient filling of the coronary arteries, in connection with stenosis of the aortic orifice, and the thus decreased contraction of the myocardium, or an affection of the latter accompanying the valvular defect, may prevent the formation of a *palpable* apex beat.

Murmurs in the Heart.—A further and, diagnostically, the most essential consequence of a stenosis of the aortic orifice is the occurrence of a *systolic murmur,* produced by the forcing of the blood through the narrow orifice into the aorta; this murmur is, in keeping with its origin, very long and drawn out, hissing, singing, or whistling in character, audible even at a great distance from the chest. The greatest intensity is at the sternal border in the second intercostal space to the right of the sternum, and it loses in strength downward; besides, it can generally be heard very plainly all over the body of the heart, even at the apex. The first sound is absent almost without exception, because, as just explained, a closure period does not, as a rule, exist. If the first sound is audible, and this is very exceptional, it occurs, as I can confirm, in regard to time a little previously to the murmur (von Noorden), because the vibrations of the ventricular wall and of the closed mitral valves which cause the first sound (during the closure period which is present in such cases) precede the forcing of the

systolic blood current through the narrowed aortic orifice—i. e., therefore, preceding the formation of the murmur by a brief moment of time.

To the same extent, most intense at the base of the heart towards the right sternal border, there is a *systolic thrill* perceptible to the palpating hand. The *second aortic sound* is weak, due to lessened pressure in the aortic system which only reaches a higher degree upon full compensation. The second aortic sound cannot be heard at all in other cases beside the long-drawn systolic murmur, or it is replaced by a diastolic murmur (owing to the synchronous insufficiency of the valves). The faint second aortic sound is not transmitted to the carotid artery, so that no diastolic sound is audible here, but only a loud systolic heart murmur in place of the first carotid sound. On the other hand, nothing, particularly not a double murmur, can ever be heard in the crural artery.

As with the radial pulse in insufficiency of the aortic valves, owing to the valvular defect, the pulse adopts highly characteristic properties,

FIG. 4.—CASE OF STENOSIS OF THE AORTIC ORIFICE.

becoming a *pulsus altus et celer*, in the same manner there occur in stenosis of the aortic orifice no less remarkable changes of the pulse, which are of moment in the diagnosis. The pulse wave becomes *small* and *sluggish* (see Fig. 4).

The curve is *low*, because, in spite of hypertrophy of the left ventricle and of the powerful pressure which is present in the latter, in the course of time only a comparatively small amount of blood is forced into the arterial vessel through the narrow aortic orifice and flows towards the peripheral circulation. A *pulsus tardus* is characteristic of stenosis of the aortic orifice, as the dilatation of the arterial vessel to its utmost occurs more slowly, on account of the impeded influx of the blood into the aorta, and because there is generally a rigidity of the artery associated with the stenosis, and with it, too, the contraction of the vessel occurs only very gradually, the latter, therefore, remains longer than normal in its condition of dilatation; the acme of the pulse curve must, consequently, become more depressed. The pulse is, at the same time, mostly *hard*; the cause of this property of the pulse should naturally not be looked for in stenosis of the aortic valve, but in arteriosclerosis associated with this cardiac defect. If the apex beat is vigorous, which, it is true, is rare, the relative smallness and slowness of the pulse contrasts strikingly with the same. As a rule, the frequency decreases also, because the heart is stimulated to but comparatively rare systoles, on account of the insufficient filling of the coronary arteries, owing to stenosis of the aortic orifice.

According to the above, the diagnosis of stenosis of the aortic orifice is simple so long as it is a question of a *pure* stenosis; but these cases are rather rare. So soon, as is very usual, as there is a simultaneous aortic insufficiency, the signs mentioned above become greatly modified. In fact, according to my experience, it is possible for the characteristic condition of the pulse to be entirely absent in stenosis of the aortic orifice, even the

most evident *pulsus altus et celer* may be present in the sphygmographic picture, which occurs when the insufficiency materially exceeds the stenosis. It is possible that in such cases the diagnosis of a simultaneous stenosis may become impossible for other reasons, because the systolic murmur may be referred to secondary dilatation of the ascending aorta (see p. 25). On the other hand, sometimes there is observed an evident gradual transition from insufficiency to stenosis, inasmuch as the endocarditic vegetations or atheromatous changes which were originally limited to the free edges of the valves, increase in intensity, extend towards the insertion ring, and thus produce stenosis of the orifice. If the compensatory power of the left ventricle continues to relax in the course of the cardiac defect, the signs of cardiac insufficiency—viz., the diffusion of the right ventricle, the dyspnœa, the deterioration of the pulse, etc., do not fail to appear in this instance either.

Congenital Stenosis of the Aortic Orifice.—The subsequent signs of *congenital stenosis of the aortic orifice* are materially different from those of stenosis later acquired. In the former the foramen ovale and the ductus Botalli remain open. In complete atresia the entire blood of the left half of the heart flows from the left auricle through the open foramen ovale into the right auricle and through the pulmonary artery to the ductus Botalli, through which a part discharges into the artery. This causes stasis phenomena in the *right heart*, right-sided dilatation of the heart, and *cyanosis*. These facts would enable us to discern a congenital stenosis of the aorta; however, the latter is scarcely an object of clinical diagnosis, because children afflicted with it almost all perish during the first days of life, rarely only after several weeks.

Of **valvular lesions of the right heart** only insufficiency of the tricuspid valve is of any considerable clinical importance. The other valvular defects of the right heart are rare pathological pictures, the foundation of which was almost always laid to faulty development and fœtal endocarditis. This is also the rule regarding the genesis of tricuspid insufficiency; but, incidentally, it is also found, more frequently than is generally supposed, as the result of endocarditic processes of post-fœtal life. Comparatively common even is that tricuspid insufficiency which is brought about without anatomical change of the valve. The dilatation of the valvular insertion ring subsequent to a dilatation of the right ventricle may assume such dimensions that the closure of the tips of the valves becomes impossible, in spite of the absence of anatomical changes in the same, and thus the symptoms of a so-called *relative* tricuspid insufficiency develop. As this latter form of tricuspid insufficiency, in my experience, comparatively often complicates cases of cardiac affection in the later stages, it can be determined if, at the autopsy, the width of the tricuspid orifice is measured exactly and compared with the length of the individual tips of the valves. Their diagnosis requires separate discussion, while we may be brief regarding the diagnosis of the other valvular defects of the right heart, more so, because their symptoms are obvious from what we have said in reference to the consequence of valvular defects of the left heart, if we consider the modifications of the pathological picture depending upon the location of the respective valvular defect in the right heart.

A short sketch of the causative factors producing the individual defects of the valves of the right heart is, therefore, sufficient.

INSUFFICIENCY OF THE VALVES OF THE PULMONARY ARTERY

Symptoms of Insufficiency of the Pulmonary Artery.—The obvious consequence of this valvular defect is an enlargement (dilatation and hypertrophy), distinctly pronounced upon percussion, of the right ventricle, with slight displacement of the apex beat outward; the border of the enlarged right ventricle, which is situated lower, can be felt between the ensiform process and the right costal arch. In the second intercostal space to the left a very loud diastolic murmur can be determined, which becomes weaker towards the apex of the heart, and, in contradistinction to the murmur in insufficiency of the aortic valves, is audible neither at the carotid arteries nor over the abdominal aorta. Over the pulmonary artery there can be felt, furthermore, a diastolic thrill, which is transmitted to some distance, inferiorly to the xiphoid process, superiorly to the clavicle. Corresponding to the secondarily dilated pulmonary artery, a systolic pulsation may be palpated in the second intercostal space to the left; upon auscultation the first pulmonary sound is pure or a systolic murmur takes its place, for the same reasons that have been explained in the transformation of the first aortic sound into a murmur in aortic insufficiency. Recently our knowledge regarding the diagnosis of this valvular defect has been materially increased by Gerhardt. He called particular attention to a *double sound* which (analogous to the double sounds in the peripheral arteries in insufficiency of the aortic valves), upon auscultation of the lungs, is observed over their surface in the *branches of the pulmonary arteries.* Besides, one hears, if auscultation is performed as far remote from the heart as possible, that the vesicular breathing is interrupted by several intervals upon inspiration—a symptom which, beyond any doubt, must be considered as *a change of the vesicular respiration due to the capillary pulse in the region of the pulmonary artery,* the presence of which may be presumed theoretically in insufficiency of the valves of the pulmonary arteries.

STENOSIS OF THE PULMONARY ORIFICE

Symptoms of Stenosis of the Pulmonary Artery.—The consequences of valvular disease of the right heart are identical with those of the left heart, hence the phenomena in stenosis of the pulmonary artery are similar to those in stenosis of the aorta: hypertrophy of the right heart manifesting itself upon percussion by a moderate diffusion of cardiac dulness to the right, and by diffuse vibration of the lower half of the sternum and of the epigastrium. The heart beat, formed by the right ventricle, is only occasionally weak, or cannot be determined at all for the same reason as explained when discussing the sequelæ of stenosis of the aortic orifice. In the second intercostal space to the left a systolic murmur is audible widely diffused, which can also be felt as systolic thrills. The stasis which takes place in the venous system is shown by *marked cyanosis,* cool extremities, and a tendency towards hæmorrhage; the filling of the region of the pulmonary artery, which soon becomes insufficient, is shown by the remarkable weakness of the *second pulmonary sound,* by asthmatic attacks, and *by the disposition of patients to cheesy degeneration, principally tuberculosis of the lungs, to which they generally early succumb.*

Differential Diagnosis of the Various Forms of Pulmonary Stenosis.—The endeavour which has been made to distinguish the comparatively frequent *congenital stenosis of the pulmonary orifice* from that acquired later in life, has been generally futile. Of course, congenital stenosis must be thought of if cyanosis is present at birth; congenital stenosis should be suspected if there exists an *accentuated second pulmonary sound* besides the symptoms of stenosis of the pulmonary orifice, because accentuation of the closure of the valves indicates a very common complication in this case, the patulous condition of the ductus Botalli. The differential signs are also insufficient, in a diagnostic respect, between *stenosis of the conus arteriosus*

dexter, which will be enlarged upon later on, and stenosis of the pulmonary orifice depending upon changes in the valves, because they are constructed artificially.

The symptoms, however, of a *narrowing of the lumen of the pulmonary artery peripherally from the valves*, which may be caused by cirrhosis of the lungs and intrathoracic tumours, are sometimes slightly at variance with stenosis of the pulmonary orifice, and the diagnosis of those stenoses of the pulmonary artery may be made with a comparative degree of certainty. The greatest intensity of the systolic murmur in such cases does not concentrate itself at the punctum maximum of the pulmonary orifice in the second intercostal space to the left of the sternum; it can be heard more readily towards the right sternal border, but, above all, it is loudest posteriorly to the left as well, between the scapula and the spinal column. The most essential differential sign, however, I consider to be the *accentuation of the second pulmonary sound*, which, in this instance, is a necessary consequence, in contradistinction to a stenosis of the pulmonary orifice, in which, for obvious reasons, the second pulmonary sound must become faint, and, in fact, was almost without exception found so. If, in a case of the above-mentioned affections of the lungs, the following two symptoms are found—displacement of the greatest intensity of the murmur to a place located more peripherally in the course of the pulmonary artery, and the accentuation of the second pulmonary sound—and if, besides, the degree of the increasing dyspnœa is not proportionate to the demonstrable changes in the lungs, we are justified in making a diagnosis of stenosis of the pulmonary artery peripherally from the valves.

INSUFFICIENCY OF THE TRICUSPID VALVES

Considerably more frequent than the valvular defects of the right heart described this far is insufficiency of the tricuspid valves. The symptoms of this valvular defect are so conspicuous that the diagnosis can almost always be made with great certainty. The characteristic phenomena are the following:

Symptoms of Tricuspid Insufficiency.—Great diffusibility of cardiac dulness to the right in consequence of considerable dilatation and hypertrophy of the right auricle and ventricle, therefore a very diffused apex beat.

Systolic murmur, most marked at the right sternal border towards its lower portion, corresponding about to the fourth or fifth intercostal space, where a systolic tremor is also felt. The murmur is transmitted to some distance towards the right.

Corresponding to regurgitation of the blood in the right auricle and in the vena cava, the venous system is under especially great tension; the latter decreases all the more in the pulmonary vessels and in the aorta, the less the right ventricle compensates fully, so that, in contradistinction to other conditions of engorgement, especially in mitral insufficiency, the second pulmonary sound does not appear accentuated. The increase in tension in the venous system and the slowing of the circulation in the capillaries associated with the stasis causes cyanosis and œdema; the most important consequence, however, of the regurgitation of the blood from the right ventricle into the large veins of the body is the plainly visible and palpable *venous pulse*. This venous pulse, as has been previously explained, which is most distinctly observed in the jugular veins, differs from the pulse of simple engorgement by its intensity and by its rhythm, inasmuch as it is very intense and not diastolic-presystolic, but *presystolic-*

systolic in time. The differentiation at the bedside, however, is not as easy as it appears from the theoretical standpoint, because in practice there are generally difficulties to be encountered in obtaining a sphygmogram of the venous pulse. We can, to a certain extent, help ourselves with a long flag which, placed upon the region of the apex of the heart, allows a simultaneous inspection of the venous pulse and of the heart beat at one glance, and thus enables the timely determination of the various phases of the venous pulse. If the latter, in an individual case, can be positively recognised as presystolic-systolic, the diagnosis of tricuspid insufficiency may be made unhesitatingly, because a distinctly positive venous pulse is not observed under other conditions, except in the rare instance of the foramen ovale remaining open in combination with mitral insufficiency. The venous pulse in the throat cannot appear in its full strength so long as the valves of the jugular vein offer an impediment to the reflux of the blood current; but, under such conditions, the vigorous closing of the valve may be observed on palpation as a short beat over the bulb of the jugular vein, and on auscultation as a tone, "jugular valve sound" (Bamberger).

Crural-Vein Sound.—In the crural vein also a valve sound can be heard, which may be double, corresponding to the presystolic and systolic filling of the vein. If the valves of the veins have become insufficient in the course of the cardiac defect, and if, nevertheless, a double sound is heard at the crural vein, its origin is to be ascribed to the vibrations of the wall of the vein, which is made tense by presystole and systole.

If a combination of tricuspid and aortic insufficiencies exists, generally originating in such a manner that in the stage of loss of compensation in the latter valvular defect a relative insufficiency of the tricuspid valve forms, a double tone of the crural artery may be simulated, inasmuch as a systolic heart sound of the crural vein and a systolic heart sound of the crural artery may be heard together, not quite isochronously, and this double sound creates the impression as if arising in the crural artery alone. If the vein and the artery are auscultated individually, which can be done less successfully with the stethoscope than with Senator's sphygmophone, we may satisfy ourselves that, during the systole of the heart, one sound arises in the vein and one in the artery, and that the former precedes the latter by a short space of time, because the venous wave has to cover about 20 cm. less distance from the right heart to Poupart's ligament than the arterial blood current. This veno-arterial double sound should therefore be differentiated from the purely arterial double sound and be designated as a "*mixed crural double sound.*"

Hepatic Venous Pulse.—Pulsation is also palpable and visible in other veins of the body, the same as in the jugular and crural veins, although less constant. As specially important is to be mentioned the *hepatic venous pulse,* which corresponds to a presystolic-systolic dilatation of the hepatic vascular net, and which is of particular diagnostic significance, inasmuch as in insufficiency of the tricuspid valve it appears occasionally earlier than the more striking pulse of the jugular vein. It may incidentally be remarked that the latter can be accentuated by pressure upon the inferior vena cava—that is, upon the liver (A. Geigel Senior).

Differential Diagnosis.—To avoid errors in diagnosis, it should not be forgotten that isolated tricuspid insufficiencies, which depend upon actual valvular changes, are exceedingly rare affections. Less so is tricus-

dexter, which will be enlarged upon later on, and stenosis of the pulmonary orifice depending upon changes in the valves, because they are constructed artificially.

The symptoms, however, of a *narrowing of the lumen of the pulmonary artery peripherally from the valves*, which may be caused by cirrhosis of the lungs and intrathoracic tumours, are sometimes slightly at variance with stenosis of the pulmonary orifice, and the diagnosis of those stenoses of the pulmonary artery may be made with a comparative degree of certainty. The greatest intensity of the systolic murmur in such cases does not concentrate itself at the punctum maximum of the pulmonary orifice in the second intercostal space to the left of the sternum; it can be heard more readily towards the right sternal border, but, above all, it is loudest posteriorly to the left as well, between the scapula and the spinal column. The most essential differential sign, however, I consider to be the *accentuation of the second pulmonary sound*, which, in this instance, is a necessary consequence, in contradistinction to a stenosis of the pulmonary orifice, in which, for obvious reasons, the second pulmonary sound must become faint, and, in fact, was almost without exception found so. If, in a case of the above-mentioned affections of the lungs, the following two symptoms are found—displacement of the greatest intensity of the murmur to a place located more peripherally in the course of the pulmonary artery, and the accentuation of the second pulmonary sound—and if, besides, the degree of the increasing dyspnœa is not proportionate to the demonstrable changes in the lungs, we are justified in making a diagnosis of stenosis of the pulmonary artery peripherally from the valves.

INSUFFICIENCY OF THE TRICUSPID VALVES

Considerably more frequent than the valvular defects of the right heart described this far is insufficiency of the tricuspid valves. The symptoms of this valvular defect are so conspicuous that the diagnosis can almost always be made with great certainty. The characteristic phenomena are the following:

Symptoms of Tricuspid Insufficiency.—Great diffusibility of cardiac dulness to the right in consequence of considerable dilatation and hypertrophy of the right auricle and ventricle, therefore a very diffused apex beat.

Systolic murmur, most marked at the right sternal border towards its lower portion, corresponding about to the fourth or fifth intercostal space, where a systolic tremor is also felt. The murmur is transmitted to some distance towards the right.

Corresponding to regurgitation of the blood in the right auricle and in the vena cava, the venous system is under especially great tension; the latter decreases all the more in the pulmonary vessels and in the aorta, the less the right ventricle compensates fully, so that, in contradistinction to other conditions of engorgement, especially in mitral insufficiency, the second pulmonary sound does not appear accentuated. The increase in tension in the venous system and the slowing of the circulation in the capillaries associated with the stasis causes cyanosis and œdema; the most important consequence, however, of the regurgitation of the blood from the right ventricle into the large veins of the body is the plainly visible and palpable *venous pulse*. This venous pulse, as has been previously explained, which is most distinctly observed in the jugular veins, differs from the pulse of simple engorgement by its intensity and by its rhythm, inasmuch as it is very intense and not diastolic-presystolic, but *presystolic-*

found in the heart, the diagnosis may become extremely difficult, because the latter may, as stated before, be due to a number of very different causes. In the first place, a coexisting stenosis of the aortic orifice is to be considered which, in fact, appears often enough associated with insufficiency. It is to be diagnosticated, besides the latter, if the systolic murmur is heard over the entire heart, yet most distinctly over the aortic orifice, if a systolic tremor is felt at the base of the heart, if the pulse is remarkably low in consideration of an insufficiency of the aortic valves, and if murmurs in the arteries are not present. Even when the loud systolic murmur is heard strongest at the base of the heart, caution in the diagnosis is necessary, as cases have been observed, nevertheless, in which no changes in the valves were found post mortem which could have been held responsible for the assumption of stenosis. In fact, it is probable that the normally wide orifice, in view of the large quantity of blood thrown with great force into the aorta, plays the part of a "physiological stenosis"; moreover, there will be another cause for the formation of a murmur if the orifice of the aorta dilates gradually, for which fact there is plenty of opportunity in aortic insufficiency by the material increase in the systolic volume of the beat. At any rate, in these cases, in contradistinction to the association of aortic insufficiency with stenosis, the condition of the pulse, characteristic of the former affection, as well as the murmur of the arteries continue to exist. But the possibilities of the origin of a systolic heart murmur are not exhausted with the above description. Aside from a simultaneous *mitral insufficiency, caused by endocarditis* (not a rare combination), it is possible that the papillary muscles of the dilated ventricle become flattened by the pressure to which they are exposed, so that their function is impaired in the course of time; besides, the same as in dilatation and hypertrophy of the left ventricle from other causes, here also the mitral valve may become *relatively insufficient* in the long run. The systolic murmur, under these circumstances, becomes soft, in contradistinction to that caused by a complicating stenosis of the aortic orifice. It is especially audible at the apex of the heart, the second pulmonary sound is intensified, and secondary hypertrophy and dilatation of the right ventricle will not fail to appear. The diagnosis of the origin of the systolic murmur, in such cases, is not difficult with a little attention, less so, if gradually a developing stasis and relative insufficiency of the tricuspid valve come into prominence.

Albuminuria may also occur as a consequence of the engorgement. The urine becomes dark, scanty, saline, will show much sediment, and it contains moderate amounts of albumin and hyaline casts. This albuminuria, however, coinciding with the changes named in the condition of the urine (stasis urine), is to be differentiated from an albuminuria which, in my experience, is not rare in insufficiency of the aortic valves, which is caused by nephritis complicating this heart lesion even during the time of complete compensation. In such instances the urine presents the characteristics of the urine of nephritis, particularly that of interstitial nephritis—i. e., it is copious, more or less clear, contains granular, besides hyaline, casts; neither does the amount of albumin vary as much as in stasis urine, in which the quantity of albumin depends upon the existing energy and activity of the heart.

Subsequent Symptoms of Hypertrophy of the Left Ventricle.—The cardiac lesion

pid insufficiency associated with other (endocarditic) valvular defects; comparatively frequent, however, as previously stated, are *relative* tricuspid insufficiencies as a consequence of excessive dilatation of the right ventricle. A question for diagnosis is, therefore, as a rule, whether, outside of a mitral insufficiency, which also leads to diffusion to the right of the cardiac dulness, to a systolic murmur and venous engorgement, there exists tricuspid insufficiency as a complication, and whether the latter is a relative insufficiency. The best mode of procedure is at first to ascertain the presence of tricuspid insufficiency by determining the *considerable* diffusion of the cardiac dulness to the right, the audibleness of the systolic murmur as far as the right mamillary line, and, above all, of the *presystolic-systolic jugular pulse* or of the hepatic venous pulse and of an unaccentuated second pulmonary sound. If the existence of tricuspid insufficiency is thus established beyond doubt, the second question to be answered is whether, besides, mitral insufficiency is present or not. The decision is easy if the case has been observed for some time, if the time at which the symptoms of tricuspid insufficiency are unquestionably demonstrable is preceded by a period in which a less pronounced and only diastolic-presystolic venous pulse, and, above all, a markedly accentuated second pulmonary sound were present besides the systolic murmur and the diffusion of the cardiac dulness. If there be present also an accentuation and displacement of the systolic murmur to the right, more or less far beyond the right border of the sternum, if the venous pulse becomes more distinct, if its rhythm changes into presystolic-systolic, and if, in spite of the increase of the stasis symptoms in the venous system, the accentuation of the second pulmonary sound decreases, the diagnosis of tricuspid and mitral insufficiencies can be made with certainty. If, however, the patient comes under observation during a later stage with pronounced symptoms of tricuspid insufficiency, the diagnosis of simultaneous mitral insufficiency can be determined only as probable. But here the correct diagnosis may be arrived at upon observation of the fact, which was particularly insisted upon by Duroziez, that the systolic murmur is not audible dorsally in tricuspid insufficiency, in contradistinction to the condition in mitral insufficiency. If there is a reason to presume the presence of relative tricuspid insufficiency, a therapeutico-diagnostic experiment may be made with digitalis. If it is a question of a simple relaxation of the overtaxed heart with passive dilatation of the right ventricle, the symptoms of tricuspid insufficiency may disappear rapidly, and the signs of the recompensated mitral insufficiency will distinctly reappear. In other cases the symptoms of tricuspid *and* mitral insufficiencies disappear, although the latter was relative, caused by affections of the heart without valvular disease, fatty heart, etc.

A spontaneous disappearance of the venous pulse is, however, not *always* a sign of disappearance of the relative tricuspid insufficiency, because a decrease in the propelling force of the right heart accompanying an increase of circulatory disturbances does not assist in producing the jugular pulse less distinctly or cause it to disappear. In such cases of excessive passive dilatation of the right ventricle the venous pulse may finally be made to reappear by digitalis, in direct opposition to the

A short sketch of the causative factors producing the individual defects of the valves of the right heart is, therefore, sufficient.

INSUFFICIENCY OF THE VALVES OF THE PULMONARY ARTERY

Symptoms of Insufficiency of the Pulmonary Artery.—The obvious consequence of this valvular defect is an enlargement (dilatation and hypertrophy), distinctly pronounced upon percussion, of the right ventricle, with slight displacement of the apex beat outward; the border of the enlarged right ventricle, which is situated lower, can be felt between the ensiform process and the right costal arch. In the second intercostal space to the left a very loud diastolic murmur can be determined, which becomes weaker towards the apex of the heart, and, in contradistinction to the murmur in insufficiency of the aortic valves, is audible neither at the carotid arteries nor over the abdominal aorta. Over the pulmonary artery there can be felt, furthermore, a diastolic thrill, which is transmitted to some distance, inferiorly to the xiphoid process, superiorly to the clavicle. Corresponding to the secondarily dilated pulmonary artery, a systolic pulsation may be palpated in the second intercostal space to the left; upon auscultation the first pulmonary sound is pure or a systolic murmur takes its place, for the same reasons that have been explained in the transformation of the first aortic sound into a murmur in aortic insufficiency. Recently our knowledge regarding the diagnosis of this valvular defect has been materially increased by Gerhardt. He called particular attention to a *double sound* which (analogous to the double sounds in the peripheral arteries in insufficiency of the aortic valves), upon auscultation of the lungs, is observed over their surface in the *branches of the pulmonary arteries*. Besides, one hears, if auscultation is performed as far remote from the heart as possible, that the vesicular breathing is interrupted by several intervals upon inspiration—a symptom which, beyond any doubt, must be considered as *a change of the vesicular respiration due to the capillary pulse in the region of the pulmonary artery*, the presence of which may be presumed theoretically in insufficiency of the valves of the pulmonary arteries.

STENOSIS OF THE PULMONARY ORIFICE

Symptoms of Stenosis of the Pulmonary Artery.—The consequences of valvular disease of the right heart are identical with those of the left heart, hence the phenomena in stenosis of the pulmonary artery are similar to those in stenosis of the aorta: hypertrophy of the right heart manifesting itself upon percussion by a moderate diffusion of cardiac dulness to the right, and by diffuse vibration of the lower half of the sternum and of the epigastrium. The heart beat, formed by the right ventricle, is only occasionally weak, or cannot be determined at all for the same reason as explained when discussing the sequelæ of stenosis of the aortic orifice. In the second intercostal space to the left a systolic murmur is audible widely diffused, which can also be felt as systolic thrills. The stasis which takes place in the venous system is shown by *marked cyanosis*, cool extremities, and a tendency towards hæmorrhage; the filling of the region of the pulmonary artery, which soon becomes insufficient, is shown by the remarkable weakness of the *second pulmonary sound*, by asthmatic attacks, and *by the disposition of patients to cheesy degeneration, principally tuberculosis of the lungs, to which they generally early succumb*.

Differential Diagnosis of the Various Forms of Pulmonary Stenosis.—The endeavour which has been made to distinguish the comparatively frequent *congenital stenosis of the pulmonary orifice* from that acquired later in life, has been generally futile. Of course, congenital stenosis must be thought of if cyanosis is present at birth; congenital stenosis should be suspected if there exists an *accentuated second pulmonary sound* besides the symptoms of stenosis of the pulmonary orifice, because accentuation of the closure of the valves indicates a very common complication in this case, the patulous condition of the ductus Botalli. The differential signs are also insufficient, in a diagnostic respect, between *stenosis of the conus arteriosus*

under these circumstances, is first to learn the exact condition and to determine the essential element of the cardiac affection. Not until the predominating valvular defect, and the subsequent symptoms depending upon it, have been determined and excluded, is it proper to ascribe those signs which cannot be made to answer to the picture of the affection of the heart, to other valvular changes. If we start from the existence of a *diastolic murmur*, a pure systolic sound being present, the first lesion to consider is aortic insufficiency. The diagnosis of the latter can almost always be made readily and with certainty. It is true, in individual cases the diastolic murmur may be very faint, but this is less essential than the fact that the murmur is diastolic in time, beyond doubt, and can be heard most distinctly over the upper part of the sternum; the subsequent manifestations, inasmuch as they refer to the peripheral vascular circulation, even if they are not well developed, are so conspicuous that no difficulty is encountered in distinguishing aortic insufficiency (even in complicated pathological heart conditions). The question, whether the diastolic murmur may not be caused concurrently by the simultaneous affection of another valve, is practically of no importance, since insufficiency of the pulmonary artery belongs to the curiosities of valvular affections, and stenoses of the auriculo-ventricular orifices, which also produce a diastolic murmur, generally do not possess the distinctly diastolic character, and, above all, are almost without exception complicated by insufficiencies of the respective valves, so that the diastolic and presystolic murmur appears in combination with a systolic murmur.

Systolic Murmurs.—If we assume the second case, that a *purely systolic murmur* is audible over the heart, in regard to diagnosis only mitral insufficiency and stenosis of the aortic orifice are to be practically considered, and of these two mitral insufficiency is so much more frequent an affection of the valves that it is the first to be considered after a systolic murmur has been demonstrated. The differentiation of both valvular defects is easy, besides, as, without reference to the point of maximum intensity of the murmur, the condition of the pulse, the secondary effect of the valvular defect upon the conduct of the various parts of the heart, etc., are widely different in both defects of the valves. There will be, consequently, no actual difficulties encountered correctly to diagnosticate the combination of both valvular defects named, if they should happen to be present in one and the same case. The same holds good for the frequent combination, mitral and tricuspid insufficiency, both of which give rise to systolic murmurs. It must not be forgotten in this respect that the occurrence of tricuspid insufficiency not in combination with other valvular defects is exceedingly rare, and that in case of doubt the presence of a mitral, besides a positively determined tricuspid, insufficiency is probable, if the latter must not be considered as *relative*, depending, for instance, upon emphysema. When discussing tricuspid insufficiency we have explicitly dwelt upon the differential points for the diagnosis of the combination, insufficiencies of the mitral and tricuspid valves; and we only wish here to lay particular stress upon the importance of observing the accentuation of the second pulmonary sound.

systolic in time. The differentiation at the bedside, however, is not as easy as it appears from the theoretical standpoint, because in practice there are generally difficulties to be encountered in obtaining a sphygmogram of the venous pulse. We can, to a certain extent, help ourselves with a long flag which, placed upon the region of the apex of the heart, allows a simultaneous inspection of the venous pulse and of the heart beat at one glance, and thus enables the timely determination of the various phases of the venous pulse. If the latter, in an individual case, can be positively recognised as presystolic-systolic, the diagnosis of tricuspid insufficiency may be made unhesitatingly, because a distinctly positive venous pulse is not observed under other conditions, except in the rare instance of the foramen ovale remaining open in combination with mitral insufficiency. The venous pulse in the throat cannot appear in its full strength so long as the valves of the jugular vein offer an impediment to the reflux of the blood current; but, under such conditions, the vigorous closing of the valve may be observed on palpation as a short beat over the bulb of the jugular vein, and on auscultation as a tone, "jugular valve sound" (Bamberger).

Crural-Vein Sound.—In the crural vein also a valve sound can be heard, which may be double, corresponding to the presystolic and systolic filling of the vein. If the valves of the veins have become insufficient in the course of the cardiac defect, and if, nevertheless, a double sound is heard at the crural vein, its origin is to be ascribed to the vibrations of the wall of the vein, which is made tense by presystole and systole.

If a combination of tricuspid and aortic insufficiencies exists, generally originating in such a manner that in the stage of loss of compensation in the latter valvular defect a relative insufficiency of the tricuspid valve forms, a double tone of the crural artery may be simulated, inasmuch as a systolic heart sound of the crural vein and a systolic heart sound of the crural artery may be heard together, not quite isochronously, and this double sound creates the impression as if arising in the crural artery alone. If the vein and the artery are auscultated individually, which can be done less successfully with the stethoscope than with Senator's sphygmophone, we may satisfy ourselves that, during the systole of the heart, one sound arises in the vein and one in the artery, and that the former precedes the latter by a short space of time, because the venous wave has to cover about 20 cm. less distance from the right heart to Poupart's ligament than the arterial blood current. This veno-arterial double sound should therefore be differentiated from the purely arterial double sound and be designated as a "*mixed crural double sound.*"

Hepatic Venous Pulse.—Pulsation is also palpable and visible in other veins of the body, the same as in the jugular and crural veins, although less constant. As specially important is to be mentioned the *hepatic venous pulse,* which corresponds to a presystolic-systolic dilatation of the hepatic vascular net, and which is of particular diagnostic significance, inasmuch as in insufficiency of the tricuspid valve it appears occasionally earlier than the more striking pulse of the jugular vein. It may incidentally be remarked that the latter can be accentuated by pressure upon the inferior vena cava—that is, upon the liver (A. Geigel Senior).

Differential Diagnosis.—To avoid errors in diagnosis, it should not be forgotten that isolated tricuspid insufficiencies, which depend upon actual valvular changes, are exceedingly rare affections. Less so is tricus-

pid insufficiency associated with other (endocarditic) valvular defects; comparatively frequent, however, as previously stated, are *relative* tricuspid insufficiencies as a consequence of excessive dilatation of the right ventricle. A question for diagnosis is, therefore, as a rule, whether, outside of a mitral insufficiency, which also leads to diffusion to the right of the cardiac dulness, to a systolic murmur and venous engorgement, there exists tricuspid insufficiency as a complication, and whether the latter is a relative insufficiency. The best mode of procedure is at first to ascertain the presence of tricuspid insufficiency by determining the *considerable* diffusion of the cardiac dulness to the right, the audibleness of the systolic murmur as far as the right mamillary line, and, above all, of the *presystolic-systolic jugular pulse* or of the hepatic venous pulse and of an unaccentuated second pulmonary sound. If the existence of tricuspid insufficiency is thus established beyond doubt, the second question to be answered is whether, besides, mitral insufficiency is present or not. The decision is easy if the case has been observed for some time, if the time at which the symptoms of tricuspid insufficiency are unquestionably demonstrable is preceded by a period in which a less pronounced and only diastolic-presystolic venous pulse, and, above all, a markedly accentuated second pulmonary sound were present besides the systolic murmur and the diffusion of the cardiac dulness. If there be present also an accentuation and displacement of the systolic murmur to the right, more or less far beyond the right border of the sternum, if the venous pulse becomes more distinct, if its rhythm changes into presystolic-systolic, and if, in spite of the increase of the stasis symptoms in the venous system, the accentuation of the second pulmonary sound decreases, the diagnosis of tricuspid and mitral insufficiencies can be made with certainty. If, however, the patient comes under observation during a later stage with pronounced symptoms of tricuspid insufficiency, the diagnosis of simultaneous mitral insufficiency can be determined only as probable. But here the correct diagnosis may be arrived at upon observation of the fact, which was particularly insisted upon by Duroziez, that the systolic murmur is not audible dorsally in tricuspid insufficiency, in contradistinction to the condition in mitral insufficiency. If there is a reason to presume the presence of relative tricuspid insufficiency, a therapeutico-diagnostic experiment may be made with digitalis. If it is a question of a simple relaxation of the overtaxed heart with passive dilatation of the right ventricle, the symptoms of tricuspid insufficiency may disappear rapidly, and the signs of the recompensated mitral insufficiency will distinctly reappear. In other cases the symptoms of tricuspid *and* mitral insufficiencies disappear, although the latter was relative, caused by affections of the heart without valvular disease, fatty heart, etc.

A spontaneous disappearance of the venous pulse is, however, not *always* a sign of disappearance of the relative tricuspid insufficiency, because a decrease in the propelling force of the right heart accompanying an increase of circulatory disturbances does not assist in producing the jugular pulse less distinctly or cause it to disappear. In such cases of excessive passive dilatation of the right ventricle the venous pulse may finally be made to reappear by digitalis, in direct opposition to the

above-mentioned effect of the drug. But, if the venous pulse disappears after tricuspid insufficiency was determined, this can only be considered as a symptom of decrease of the latter, therefore as a favourable sign, if, at the same time, the general engorgement in the veins abates without question and also the radial pulse grows again more vigorous.

STENOSIS OF THE TRICUSPID ORIFICE

This valvular defect has almost invariably been observed with simultaneous tricuspid insufficiency and so rarely, that generally it is not a question of positive diagnosis. What, theoretically, is required as a result of this valvular defect, is in part only borrowed from the picture of stenosis of the mitral orifice:

Dilatation and hypertrophy of the right auricle, increase of tension in the system of the venæ cavæ, high-graded cyanosis, decrease of pressure in the pulmonary vessels and in the aorta—i. e., therefore, a weak second pulmonary sound and a small radial pulse. Presystolic or diastolic murmur over the tricuspid valve, which, however, may eventually be absent entirely, analogous to what has been said in mitral stenosis, as a case observed in my clinic has taught me. At the jugular vein there appears a venous pulse, at the anacrotic leg of which the *presystolic* elevation must be *relatively* very marked. *The character of the venous pulse in tricuspid stenosis is negative*—i. e., diastolic-presystolic, as I can state from experience, even when an insufficiency of the tricuspid valve forms simultaneously with the stenosis. But the effect of the former may become conspicuous in so far as the vein collapses less during the ventricular systole. If in a combination of stenosis with tricuspid insufficiency the latter predominates, the venous pulse is positive, presystolic-systolic, but differs from the usual venous pulse which occurs in insufficiency in so far as the distention of the vein remains, at least to a certain extent, during the entire duration of the ventricular diastole, in contradistinction to the diastolic shrinking of the jugular veins in uncomplicated tricuspid insufficiency. Owing to the crippled function of the right ventricle, thrombi are apt to form, which cause hæmorrhagic infarcts of the lungs and speedy death.

Tricuspid stenosis may be pronounced a *congenital* heart defect if cyanosis exists from birth, and if a congenital pulmonary stenosis can be excluded.

COMBINED VALVULAR DEFECTS

It would be very erroneous to believe that valvular defects always or even frequently present themselves at the bedside with such simplicity and clearness of the clinical picture as we have depicted, and which was necessary for purposes of description. Very often it is a question, in the individual case, rather of a *combination of various valvular defects,* as can be readily understood when the ætiology of the latter condition is considered. This renders the diagnosis in most instances more difficult, because symptoms which are characteristic of one valvular defect appear modified and partly masked by those of the others. A diagnostic axiom,

systolic murmur over the pulmonary artery is transmitted, although attenuated, into the aorta and left carotid artery in persistence of the ductus Botalli, not so, however, in pulmonary stenosis; finally, in the latter, if uncomplicated, the second pulmonary sound is not accentuated; in patulous ductus Botalli, however, it is quite marked.

PERSISTENCY OF THE AORTIC ISTHMUS

In those cases in which fœtal stenosis persists of that portion of the aorta which is located between the point at which the left subclavian artery branches off, and the inosculation of the ductus Botalli, the "aortic isthmus," persists, and still further narrows with the occlusion of the ductus Botalli, there will result certain clinical phenomena which allow of a diagnosis of this defect of development. The latter is found by far more frequently in the male. The arteries which branch off from the aortic arch to the upper half of the body are remarkably wide, the pulse large, and fluxion to the brain is present. *The arteries of the lower half of the body,* in marked contradistinction to this, *are narrow, the pulse in the abdominal aorta and in the crural arteries is small, heavy, and palpable later than the pulse of the radial arteries.* At the same time the *left ventricle* is *hypertrophied,* owing to the impeded circulation, and a conspicuously visible and palpable *collateral circulation* has developed between the upper and lower halves of the body.

Development of Collateral Circulation.—The *diagnosis* can be made with certainty from these three symptoms. It is supplemented by closer observation of the development of the collateral circulation: The anastomosis of the subclavian—internal mammary—crural arteries (by means of the superior and inferior epigastric), on the one hand, and of the descending thoracic aorta (by means of the anterior intercostal—posterior arteries—descending thoracic aorta) on the other hand; furthermore, the anastomosis of the subclavian—transversa colli—dorsalis scapulæ, which, located at the interior border of the scapula, communicate with the posterior intercostal arteries from the descending thoracic aorta. As a visible expression of this, making use of the anastomosis on the part of the blood current which branches from the aortic arch to the inferior half of the body, we see those vessels of communication greatly distended and markedly pulsating, especially in the region of the interior border of the scapula and the anterior thoracic wall. Over these dilated vessels *systolic heart murmurs* can be heard (especially in the region of the internal mammary), which can be distinguished from heart murmurs inasmuch as they follow, regarding time, the systolic, pure heart sound. Palpation will show a *systolic thrill* of the dilated arteries, caused by the extremely dilated arterial walls which thereby vibrate irregularly with the tension of the systole of the heart. The vigorous force of the blood, which is increased by the hypertrophy of the left ventricle, gradually dilates the *arch of the aorta,* and the pulsation of the latter can be felt from the throat posteriorly to the manubrium sterni.

Combination of Systolic and Diastolic Murmurs.—The matter becomes more complicated when, as is frequently the case, *a systolic and a diastolic murmur* is heard in the heart upon auscultation. In this case it is most important to ascertain the location of the greatest intensity of the murmurs, and, further, to observe which portion of the heart is hypertrophied most. Of course, there can be no doubt as to the diagnosis if both murmurs are most distinct over the upper part of the sternum and if the hypertrophy is concentrated in the left ventricle; in this case the defect which causes both murmurs is to be referred to the aortic orifice. Insufficiency is present in any case; whether stenosis exists synchronously or whether the systolic murmur, as often occurs, is only an accompanying symptom and a consequence of the insufficiency of the aortic valves, this is to be decided by the condition of the pulse and by an eventual auscultation of the arteries—details of the diagnosis which have been considered in particular previously, when it was a question of differential diagnosis, which facts were to be considered in insufficiency of the aortic valves (see p. 25). Stenosis and insufficiency of the aortic valve frequently occur together, but still more frequent is the combination of mitral insufficiency and stenosis of the mitral orifice. It is to be diagnosticated if the consecutive hypertrophy particularly affects the right heart and if the symptoms of mitral insufficiency are so modified (high-graded accentuation and eventual division of the second pulmonary sound, small pulse, modified diastolic, especially presystolic murmur, etc.), that we are justified in considering a simultaneous stenosis of the mitral orifice.

The not infrequent combination of mitral and aortic valvular defects, too, with murmurs in unequal phases of the cardiac cycle, is at times not difficult of recognition, as will be shown by a special example. Of course, it is necessary to consider carefully all the fundamental laws referring to each individual valvular defect, particularly in a combination of aortic with mitral insufficiency, the eventual presence of distinctly pronounced back-stroke elevations (see p. 22). In general, besides, I would recommend caution in the diagnosis of three or, may be, four different valvular defects in one and the same heart, and would advise the diagnostician only to make so complicated a diagnosis in case the assumption of two valvular defects is absolutely insufficient for the explanation of the symptoms present. We should not be satisfied if at the autopsy at least one or even two of the valvular defects diagnosticated are present, and only the diagnosis of the last one was wrong. As though it could be different if one of the three is found! It would have been much more correct in such a case positively to diagnosticate the cardinal defect, and to leave the remainder in suspense, than to make a detailed diagnosis which does not correspond to the anatomical findings.

It does not answer the purpose, nor possibly further the case, to enter into details; each individual instance of a combination of different heart lesions is to be specially considered and studied. To illustrate our explanation we shall give a short description of a case of combination of several valvular defects and shall analyze the various subsequent symptoms in a special instance.

on percussion by a tympanitic or, possibly, relatively dull sound, which is more pronounced in the infraclavicular, and, particularly posteriorly, in the scapular region, where also the other signs of pulmonary compression, bronchial respiration, etc., are encountered.

Inspection and Palpation.—Inspection shows a dilatation of the thorax in the cardiac area, a widening of the intercostal spaces, and a retraction of the left half of the thorax during respiration, quite analogous to the conditions in pleural exudates—sequels of exudation which, diagnostically, come into consideration only supplementarily. *Of diagnostic importance, however, is the condition of the apex beat.* The latter is drawn downward with the downward pressure of the diaphragm, and also slightly to the left, as the heart, under these circumstances, takes a more horizontal position. *But the most important change is that the cardiac dulness* (particularly the left leg of its triangle) *extends beyond the apex beat,* because a part of the exudate will still be situated to the left of the heart. The more fluid then accumulates in the pericardium during the course of the inflammation, the weaker becomes the apex beat until it finally disappears entirely. *On making the patient bend forward it will generally be possible,* inasmuch as the heart sinks anteriorly in the exudate, to cause *the reappearance of the previously absent apex beat.*

The following case, which I have observed, will show how important, diagnostically, is the weakness and position of the apex beat within the area of dulness:

In a patient—with mitral insufficiency—there *suddenly* occurs, with dyspnœa and an aggravation of the condition of the pulse, *a diffusion of cardiac dulness to the right beyond the sternum.* At the same time a *friction sound is heard over the middle of the sternum.* On the other hand, the apex beat can be felt as vigorously as before at the farthest border of the cardiac dulness to the left. The diagnosis of exudative pericarditis (pericarditis with effusion) was not made, owing to the latter fact, as was so suggestive, but, instead, that of acute enlargement of the heart with pericarditis sicca. The autopsy of the patient, who died two days later, showed acute dilatation of the right heart, especially of the right auricle, with acute mediastinitis, the latter having caused the friction murmur in the cardiac region.

Auscultation, Friction Sound.—More important than all the signs so far mentioned for a positive diagnosis of pericarditis is the *friction murmur,* which can be observed upon *auscultation* (sometimes also palpable), and which is brought about by displacement of the pericardial leaves which have become roughened by the inflammation. The friction sound is diagnostically very significant, because it almost always has so decided an acoustic character that the diagnosis can at once be made upon the mere presence of this sign.

Specific Properties of the Pericardial Friction Sound.—The friction sound insures the diagnosis also in those cases in which a fluid exudate is absent; it is remarkable that a friction sound is mostly also heard when larger quantities of exudate force the pericardial leaves apart. In the latter case it is to be looked for towards the base of the heart where the large vessels issue from the pericardium, because the leaves of the latter remain longest together here in progressive exudation. On the other hand, the murmur may be entirely absent, as a recent observation taught me, in spite of the absence of the exudate even when there is an enormous development of the pericardial inflammation, provided the latter is very soft and the action

of the heart very weak. In this case the diagnosis of pericarditis could be made the less, because the cardiac dulness was actually slightly enlarged, owing to the massy pericardial layers, whereas an exudate was almost entirely absent, and the weak apex beat coincided accordingly with the most extreme left cardiac dulness.

The acoustic character of the cardiac friction sound is extremely variable, and yet the friction sound is comparatively easily recognised as such. Its character cannot be learned from descriptions; it must be heard frequently, so that the ear is able to distinguish it from other, particularly endocardial, murmurs. It is therefore, in my opinion, of no value whatever to set up different varieties of the friction sound (grating, scratching, etc.). The diagnosis was always rendered easy to me by the observation of the *intermittent* course of the friction sound; in favour of the pericardial character of a sound is furthermore the defective relation of the friction sound with the systole or the diastole of the heart (the "trailing"). This symptom is conspicuous only in a slow heart beat or if attention is paid to the time of the interval upon an accidentally intermitting pulse. It is more important, in my experience, that the friction sound impresses us as very superficial, originating near the ear, and generally grows louder if the stethoscope is applied with a little more pressure. The friction sounds are mostly heard more distinctly if the patient is made to take a deep breath, to hold it at the height of the inspiration, while on expiration only exceptionally an intensification of the murmur occurs. Still more certain diagnostically than the last-named characteristics I consider the fact that, with a changed attitude of the body, particularly when sitting or on bending the body forward—i. e., pressing the heart against the anterior wall of the thorax—pericardial friction sounds appear which were not audible in the recumbent position, or that friction sounds, which were present in this position, become accentuated, and, finally, that the pericardial friction sounds are sometimes most distinctly audible in places which do not correspond to the puncta maxima of the valves, and are strictly localized, in contradistinction to endocardial murmurs [which are, as a rule, transmitted], so that they cannot be heard any more in the most immediate neighbourhood of the præcordial area. We can readily convince ourselves of this condition of the friction sound in many cases.

Concomitant Signs.—From the signs mentioned thus far the diagnosis of a pericarditis can, in an individual case, generally be made easily and positively. Whatever else is observed in the picture of pericarditis are subordinate factors which supplement the diagnosis; for instance, the pain in the cardiac region, the fever which is very fluctuating in character and has no specific curve, the impeded accelerated respiration. More important are a number of symptoms which depend upon the mechanical pressure of the exudate upon the heart and the *impediment to the diastole of the heart* thereby, inasmuch as the veins accordingly empty their blood less readily into the heart, the arterial system is badly filled, and a lowering of the blood pressure is bound to make itself felt. In such a manner these well-known disturbances of circulation occur: enlargement of the liver (with it, later, occasionally ascites), decrease in the amount of urine, general dropsy and cyanosis, finally also fainting attacks when the patient raises himself, and thus the already insufficiently filled arteries become still more depleted.

Sequels of the Pressure Upon the Adjacent Regions.—Let me further mention some symptoms which, independent of disturbance of circulation, owe their origin to the local pressure of the exudate upon organs which are contiguous to the pericardium, and, inasmuch as they represent affections especially referring to diseases of the pericardium, are sometimes of specific diagnostic significance. Among them are, besides *dysphagia, paralysis of the vocal cords, vomiting*, and *singultus*. Possibly the slowing of the pulse occasionally observed may also be included and be traced to an irritation of the vagus by the pressure of the exudate.

Differential Diagnosis.—It is obvious that the *diffusion of cardiac dulness* may be due to the most varied causes and give rise to errors in diagnosis. If the above triangular figure with obtuse apex favours the accumulation of fluid in the pericardium, then, outside of pericarditis, hydropericardium may be present. As it is possible that pericarditis also may be accompanied with symptoms of engorgement and that the friction sound in it may disappear as soon as the effusion becomes copious, it is evident that a confusion of both affections is possible, more so, as the fever which is generally allied with pericarditis may be absent in some instances (especially if it is a question of the chronic form of pericarditis). If there is an opportunity to observe the affection for some time, there is difficulty in deciding upon the assumption of one or the other condition, especially if the ætiological data in the individual case are also taken into consideration. The occurrence of the cardiac affection in the course of an infectious disease, as acute articular rheumatism, sepsis, tuberculosis, diphtheria, scarlatina, pneumonia, scurvy, respectively, of the hæmorrhagic diathesis in general, finally in inflammations of organs adjacent to the pericardium, speaks for the presence of pericarditis and against hydropericardium in which general or local disturbances of circulation must, by all means, be demonstrable as the original cause.

In cases in which these diagnostic indications are not sufficient (and such cases occur because Bright's disease, tuberculosis, etc., give rise to both conditions), *digitalis* should be given for diagnostic-therapeutic purposes. The disappearance of the engorgement symptoms and, simultaneously, of the diffusion of the cardiac dulness, is in favour of hydropericardium; if, however, the fluid remains in the pericardium—i. e., if the dulness remains diffused—the diagnosis of an inflammatory character of the accumulated fluid in the pericardium becomes probable. It is true that under these circumstances, owing to increased diuresis, the resorption of the pericardial exudate may be ushered in; but then it may be expected that the friction sound, which so far could not be heard, makes its appearance also, thus greatly facilitating the differential diagnosis. If it is a question of a rapid and positive diagnosis, an exploratory puncture should be made—a very simple, not dangerous operation; it should, however, be restricted to those cases in which it is indicated not only in a differential-diagnostic, but also in a diagnostic-therapeutic respect. The examination of the fluid obtained by puncture will show a high specific gravity in pericarditis, eventually cloudiness or pus may be contained in the same. At the same time the diagnosis becomes a certainty in the assumption of a *hæmapericardium*, the presence of which is to be considered from the beginning in those rare cases in which the accumulation of fluid in the pericardium develops rapidly with the symptoms of acute anæmia.

Enlargement of the Heart and Faintness of the Action of the Heart.—Greater difficulties than the differentiation of pericarditis from hydropericardium sometimes present themselves upon deciding the question whether the diffusion of the cardiac dulness is caused by an exudative pericarditis or by an enlargement of the heart. So long as the apex beat is distinct, its location easily decides the question. As soon, however, as it is situated at the outermost border of the dulness to the left, an enlargement of the heart is to be assumed, even if everything else is in favour of pericarditis; if, however, the apex beat is situated within the left border of the cardiac dulness, the diagnosis must be pericarditis. If, on the other hand, the apex beat disappears in both affections, in pericarditis owing

to the withdrawal of the heart from the wall of the thorax in consequence of the exudate, in enlargement of the heart owing to increasing cardiac weakness, the differential diagnosis is very difficult. If the pulse is comparatively vigorous, with absence of the apex beat, this incongruity of apex beat and pulse fulness naturally leads to the diagnosis of pericarditis. But, as we have seen, precisely a massive exudate, which causes the heart to recede and the apex beat to disappear, is also the cause of a deficient diastole, and thus of engorgement and low tension in the arterial system, so that the significance of the differentio-diagnostic signs mentioned is a limited one. Here, too, diagnostic advantage may be expected of digitalis, which is at the same time the most important therapeutic agent in such conditions.

Instead of digitalis, Th. Schott has recently recommended *resistance gymnastics*, to be undertaken systematically with the patient, as a means diagnostically to distinguish a dilated heart from a pericardial effusion in all doubtful cases. The diffusion of the cardiac dulness caused by dilatation of the heart recedes here rapidly, for instance, in half an hour, while the (diffused) dulness in pericardial exudate is not influenced by the gymnastic exercise. Less differential-diagnostic certainty is offered, in my experience, by another physical means of examination, viz., the variation of diffusion of cardiac dulness in different postures of the body. Theoretically, it should be assumed that, upon sitting up, or even *bending forward of the patient, the cardiac dulness increases considerably in pericarditis and not in dilatation of the heart*. But we can easily convince ourselves (especially in cases in which dilatation of the heart with complete adhesion of the pericardial membranes is found at autopsy, so that a hydropericardium can be excluded which might have been absorbed during the last hours of life) that the dilated heart also, in the various postures of the patient, exerts considerable influence upon the extent of cardiac dulness. Upon bending forward, the heavy, large heart is brought anteriorly, pushes back the margins of the lung, and thus makes the limits of the cardiac dulness appear, for the moment, considerably wider. Compared with the increase under such circumstances of the diffusion of dulness by the pericardial exudate, the increase owing to the voluminous heart is probably less; but no diagnosis should be made in doubtful cases based upon such relative conditions of diffusion of dulness. Only when, upon bending forward of the patient, the apex beat, which was absent until then, reappears distinctly within the area of dulness, the cause of the increase of cardiac dulness may be looked for in an accumulation of fluid in the pericardium.

Diffusion of the Cardiac Dulness by Pulmonary Retraction, etc.—By retraction of the lungs, the heart, even if not dilated, may be exposed to such an extent that a pericardial exudate may be simulated, and the same might be the case, considered theoretically—practically it will scarcely ever occur—if the borders of the lungs contiguous to the heart become airless. In the former case, the simultaneous high position of the diaphragm and the diffused movement of the heart, the situation of the apex beat at the outermost border of the cardiac dulness, also the visible and palpable vigorous pulsation of the pulmonary artery, will guard against serious errors. In the latter case, the absence of vesicular breathing, the increase of the vocal fremitus and the irregularity of the limits of the dulness in the infiltrated borders of the lungs will be factors determining the diagnosis. A mediastinal tumour may also accidentally develop in such a manner that the dulness caused by it connects immediately with normal cardiac dulness and makes the latter appear increased. In this instance, the absent dulness upon change of position, the conduct of the apex beat, and, above all, the manner in which the dulness grows, mostly unilaterally and irregularly, will insure a correct diagnosis.

Masking of the Pericardial Exudate by Pulmonary Emphysema.—Conversely, a pericardial exudate may remain hidden or appear smaller than it actually is, if the anterior borders of the lung are coalescent and, for this reason, cannot recede on accumulation of exudates in the pericardium, or if an emphysematous lung very

Differential Diagnosis.—It is obvious that the *diffusion of cardiac dulness* may be due to the most varied causes and give rise to errors in diagnosis. If the above triangular figure with obtuse apex favours the accumulation of fluid in the pericardium, then, outside of pericarditis, hydropericardium may be present. As it is possible that pericarditis also may be accompanied with symptoms of engorgement and that the friction sound in it may disappear as soon as the effusion becomes copious, it is evident that a confusion of both affections is possible, more so, as the fever which is generally allied with pericarditis may be absent in some instances (especially if it is a question of the chronic form of pericarditis). If there is an opportunity to observe the affection for some time, there is difficulty in deciding upon the assumption of one or the other condition, especially if the ætiological data in the individual case are also taken into consideration. The occurrence of the cardiac affection in the course of an infectious disease, as acute articular rheumatism, sepsis, tuberculosis, diphtheria, scarlatina, pneumonia, scurvy, respectively, of the hæmorrhagic diathesis in general, finally in inflammations of organs adjacent to the pericardium, speaks for the presence of pericarditis and against hydropericardium in which general or local disturbances of circulation must, by all means, be demonstrable as the original cause.

In cases in which these diagnostic indications are not sufficient (and such cases occur because Bright's disease, tuberculosis, etc., give rise to both conditions), *digitalis* should be given for diagnostic-therapeutic purposes. The disappearance of the engorgement symptoms and, simultaneously, of the diffusion of the cardiac dulness, is in favour of hydropericardium; if, however, the fluid remains in the pericardium—i. e., if the dulness remains diffused—the diagnosis of an inflammatory character of the accumulated fluid in the pericardium becomes probable. It is true that under these circumstances, owing to increased diuresis, the resorption of the pericardial exudate may be ushered in; but then it may be expected that the friction sound, which so far could not be heard, makes its appearance also, thus greatly facilitating the differential diagnosis. If it is a question of a rapid and positive diagnosis, an exploratory puncture should be made—a very simple, not dangerous operation; it should, however, be restricted to those cases in which it is indicated not only in a differential-diagnostic, but also in a diagnostic-therapeutic respect. The examination of the fluid obtained by puncture will show a high specific gravity in pericarditis, eventually cloudiness or pus may be contained in the same. At the same time the diagnosis becomes a certainty in the assumption of a *hæmapericardium*, the presence of which is to be considered from the beginning in those rare cases in which the accumulation of fluid in the pericardium develops rapidly with the symptoms of acute anæmia.

Enlargement of the Heart and Faintness of the Action of the Heart.—Greater difficulties than the differentiation of pericarditis from hydropericardium sometimes present themselves upon deciding the question whether the diffusion of the cardiac dulness is caused by an exudative pericarditis or by an enlargement of the heart. So long as the apex beat is distinct, its location easily decides the question. As soon, however, as it is situated at the outermost border of the dulness to the left, an enlargement of the heart is to be assumed, even if everything else is in favour of pericarditis; if, however, the apex beat is situated within the left border of the cardiac dulness, the diagnosis must be pericarditis. If, on the other hand, the apex beat disappears in both affections, in pericarditis owing

to the withdrawal of the heart from the wall of the thorax in consequence of the exudate, in enlargement of the heart owing to increasing cardiac weakness, the differential diagnosis is very difficult. If the pulse is comparatively vigorous, with absence of the apex beat, this incongruity of apex beat and pulse fulness naturally leads to the diagnosis of pericarditis. But, as we have seen, precisely a massive exudate, which causes the heart to recede and the apex beat to disappear, is also the cause of a deficient diastole, and thus of engorgement and low tension in the arterial system, so that the significance of the differentio-diagnostic signs mentioned is a limited one. Here, too, diagnostic advantage may be expected of digitalis, which is at the same time the most important therapeutic agent in such conditions.

Instead of digitalis, Th. Schott has recently recommended *resistance gymnastics*, to be undertaken systematically with the patient, as a means diagnostically to distinguish a dilated heart from a pericardial effusion in all doubtful cases. The diffusion of the cardiac dulness caused by dilatation of the heart recedes here rapidly, for instance, in half an hour, while the (diffused) dulness in pericardial exudate is not influenced by the gymnastic exercise. Less differential-diagnostic certainty is offered, in my experience, by another physical means of examination, viz., the variation of diffusion of cardiac dulness in different postures of the body. Theoretically, it should be assumed that, upon sitting up, or even *bending forward of the patient, the cardiac dulness increases considerably in pericarditis and not in dilatation of the heart*. But we can easily convince ourselves (especially in cases in which dilatation of the heart with complete adhesion of the pericardial membranes is found at autopsy, so that a hydropericardium can be excluded which might have been absorbed during the last hours of life) that the dilated heart also, in the various postures of the patient, exerts considerable influence upon the extent of cardiac dulness. Upon bending forward, the heavy, large heart is brought anteriorly, pushes back the margins of the lung, and thus makes the limits of the cardiac dulness appear, for the moment, considerably wider. Compared with the increase under such circumstances of the diffusion of dulness by the pericardial exudate, the increase owing to the voluminous heart is probably less; but no diagnosis should be made in doubtful cases based upon such relative conditions of diffusion of dulness. Only when, upon bending forward of the patient, the apex beat, which was absent until then, reappears distinctly within the area of dulness, the cause of the increase of cardiac dulness may be looked for in an accumulation of fluid in the pericardium.

Diffusion of the Cardiac Dulness by Pulmonary Retraction, etc.—By retraction of the lungs, the heart, even if not dilated, may be exposed to such an extent that a pericardial exudate may be simulated, and the same might be the case, considered theoretically—practically it will scarcely ever occur—if the borders of the lungs contiguous to the heart become airless. In the former case, the simultaneous high position of the diaphragm and the diffused movement of the heart, the situation of the apex beat at the outermost border of the cardiac dulness, also the visible and palpable vigorous pulsation of the pulmonary artery, will guard against serious errors. In the latter case, the absence of vesicular breathing, the increase of the vocal fremitus and the irregularity of the limits of the dulness in the infiltrated borders of the lungs will be factors determining the diagnosis. A mediastinal tumour may also accidentally develop in such a manner that the dulness caused by it connects immediately with normal cardiac dulness and makes the latter appear increased. In this instance, the absent dulness upon change of position, the conduct of the apex beat, and, above all, the manner in which the dulness grows, mostly unilaterally and irregularly, will insure a correct diagnosis.

Masking of the Pericardial Exudate by Pulmonary Emphysema.—Conversely, a pericardial exudate may remain hidden or appear smaller than it actually is, if the anterior borders of the lung are coalescent and, for this reason, cannot recede on accumulation of exudates in the pericardium, or if an emphysematous lung very

extensively covers the heart. In these conditions, the pericardial effusion can be recognised, above all, by the great extent of the relative cardiac dulness and its increase upon change of position.

As a rule, all the above-named difficulties can be easily overcome by the demonstration of the friction sound which is present in almost all cases of pericarditis, at least at certain stages of the disease.

Pericardial and Endocardial Murmurs.—This sound is of such a distinct character that it cannot easily be confounded with other murmurs near the heart. We have already learned the characteristics which cause the pericardial friction sound to be differentiated from the endocardial murmurs. By way of supplement, it may be stated here that the diagnosis of the character of the friction sound is sometimes rendered difficult by the fact that, besides the friction sound, the first or second sound is not heard pure, but that, during systole and diastole, only murmurs are noticed, or that pericardial and endocardial murmurs appear simultaneously in one and the same case. Generally the latter can then no longer be diagnosticated because the former are stronger; but the converse is also the case.

Pleuropericardial ("Extrapericardial") Friction Sound.—In a pneumonia affecting the peripheral parts of the lungs, or in pleurisy, which is located in the neighbourhood of the heart, it occurs comparatively not infrequently that the pleura, which has become inflamed and coarse adjacent to the heart, is so affected by the heart's action that there is produced a friction sound synchronous with the motion of the heart, the *pleuropericardial* or *extrapericardial* friction sound. As a rule, this phenomenon, observed at the bedside, corresponds post mortem to an inflammatory affection of both pleural surfaces, facing each other, of the sinus mediastinocostalis. If the inflammation is thus located, friction sounds are produced by the friction of the inflammatory coarse pleural leaves either during respiration or during the systole and diastole of the heart. For this reason, it is possible that the friction sound synchronous with the action of the heart may be confounded with pericardial friction sounds. *If the patient, after deep inspiration, is made to hold his breath, the friction sound generally disappears entirely, in contradistinction to the usual pericardial sound,* because the lung, which enters the mediastinocostal sinus upon inspiration, renders the friction of the coarse pleural surfaces difficult through the action of the heart. But this differential-diagnostic characteristic does not, in my experience, give any positive support to the diagnosis; on the contrary, pleuropericardial friction is, sometimes, particularly distinct during the height of the inspiration. The signs most valuable for the diagnosis are, that the friction sound in pericardial pleurisy is not only connected with the phases of the action of the heart, but, above all, with the respiration; furthermore, that, besides the friction sound produced by the action of the heart, a distinct pleural friction can be heard, the exudate in the pericardium is absent, and that the friction sound does not become more distinct upon the bending forward of the patient, in contradistiction to internal pericarditis. The differentiation between a pleuritis sicca and a pleuritis pericardiaca becomes impossible when the former is complicated by a pleurisy localized in the neighbourhood of the heart. Only the diagnosis of pericarditis (sicca) is conceivable in those cases in which the friction sound occurs in the centre of the cardiac dulness, in a region, therefore, which the mediastinocostal sinus does not reach. With due observation of these diagnostic points the recognition of a pleuropericarditis is not so difficult as it seems at first glance; I have been able almost always to make a positive and, as shown by autopsies, a correct diagnosis.

As a sequel to pericardial pleurisy Riegel described the peculiar phenomenon that the heart beat, which normally becomes fainter on inspiration, is subject to an expiratory diminution. He explains this in such a manner that band-like adhesions are situated between lung and heart and are of such a nature that

to the withdrawal of the heart from the wall of the thorax in consequence of the exudate, in enlargement of the heart owing to increasing cardiac weakness, the differential diagnosis is very difficult. If the pulse is comparatively vigorous, with absence of the apex beat, this incongruity of apex beat and pulse fulness naturally leads to the diagnosis of pericarditis. But, as we have seen, precisely a massive exudate, which causes the heart to recede and the apex beat to disappear, is also the cause of a deficient diastole, and thus of engorgement and low tension in the arterial system, so that the significance of the differentio-diagnostic signs mentioned is a limited one. Here, too, diagnostic advantage may be expected of digitalis, which is at the same time the most important therapeutic agent in such conditions.

Instead of digitalis, Th. Schott has recently recommended *resistance gymnastics*, to be undertaken systematically with the patient, as a means diagnostically to distinguish a dilated heart from a pericardial effusion in all doubtful cases. The diffusion of the cardiac dulness caused by dilatation of the heart recedes here rapidly, for instance, in half an hour, while the (diffused) dulness in pericardial exudate is not influenced by the gymnastic exercise. Less differential-diagnostic certainty is offered, in my experience, by another physical means of examination, viz., the variation of diffusion of cardiac dulness in different postures of the body. Theoretically, it should be assumed that, upon sitting up, or even *bending forward of the patient, the cardiac dulness increases considerably in pericarditis and not in dilatation of the heart.* But we can easily convince ourselves (especially in cases in which dilatation of the heart with complete adhesion of the pericardial membranes is found at autopsy, so that a hydropericardium can be excluded which might have been absorbed during the last hours of life) that the dilated heart also, in the various postures of the patient, exerts considerable influence upon the extent of cardiac dulness. Upon bending forward, the heavy, large heart is brought anteriorly, pushes back the margins of the lung, and thus makes the limits of the cardiac dulness appear, for the moment, considerably wider. Compared with the increase under such circumstances of the diffusion of dulness by the pericardial exudate, the increase owing to the voluminous heart is probably less; but no diagnosis should be made in doubtful cases based upon such relative conditions of diffusion of dulness. Only when, upon bending forward of the patient, the apex beat, which was absent until then, reappears distinctly within the area of dulness, the cause of the increase of cardiac dulness may be looked for in an accumulation of fluid in the pericardium.

Diffusion of the Cardiac Dulness by Pulmonary Retraction, etc.—By retraction of the lungs, the heart, even if not dilated, may be exposed to such an extent that a pericardial exudate may be simulated, and the same might be the case, considered theoretically—practically it will scarcely ever occur—if the borders of the lungs contiguous to the heart become airless. In the former case, the simultaneous high position of the diaphragm and the diffused movement of the heart, the situation of the apex beat at the outermost border of the cardiac dulness, also the visible and palpable vigorous pulsation of the pulmonary artery, will guard against serious errors. In the latter case, the absence of vesicular breathing, the increase of the vocal fremitus and the irregularity of the limits of the dulness in the infiltrated borders of the lungs will be factors determining the diagnosis. A mediastinal tumour may also accidentally develop in such a manner that the dulness caused by it connects immediately with normal cardiac dulness and makes the latter appear increased. In this instance, the absent dulness upon change of position, the conduct of the apex beat, and, above all, the manner in which the dulness grows, mostly unilaterally and irregularly, will insure a correct diagnosis.

Masking of the Pericardial Exudate by Pulmonary Emphysema.—Conversely, a pericardial exudate may remain hidden or appear smaller than it actually is, if the anterior borders of the lung are coalescent and, for this reason, cannot recede on accumulation of exudates in the pericardium, or if an emphysematous lung very

feriorly owing to contraction of the auricles immediately before the onset of the systole of the ventricle. This movement of the cardiac apex is essentially the result of the perpendicular position of the axis of the ventricle to the base of the heart, the transverse section of which shows, in place of the elliptic form it ought to have during the diastole, a more circular one during the systole (Ludwig). Thus, and because the ventricular wall then becomes more elastic, it is conceivable that the apex of the heart is displaced anteriorly to such an extent that an arching of the fifth intercostal space, towards which slopes the inclined plane formed by the diaphragm, may occur through the contracting heart situated upon the latter. So much space as is created by the heart diminishing in size, the borders of the lungs occupy by advancing during the systole. As soon as the latter can no longer take place, for instance, in shriveling and coalescence of the borders of the lung, in hypertrophy of the heart, there occur, as there is a frequent opportunity to observe, upon pronounced apex beat, retractions of the intercostal spaces in the entire cardiac region as an expression of the systolic diminution of the heart, which cannot be followed by a corresponding filling of the thus created empty space by the lungs. These retractions should be well differentiated from the *systolic retraction* [dimpling] *of the apex of the heart* with which we have to do.

Systolic Retraction in the Region of the Apex Beat.—This latter occurs only when the locomotion of the heart apex just described is anteriorly impeded. Because in this process of locomotion the base also moves forward, it will be a question, in adherence of the pericardium, whether they affect the basal part of the heart and interfere with its downward motion. As a matter of fact, it has been demonstrated at autopsies that even insignificant adhesions were able to cause systolic retraction of the fifth intercostal space, inasmuch as they affected the base of the heart, while even extensive adhesions at other parts passed without symptoms *intra vitam*. If, on the other hand, the obliterated pericardium presents, in place of the normal loose approximation, an adherence to the anterior wall of the thorax and to the spinal column by means of very tense masses of connective tissue, not only a retraction of the intercostal space *but also a retraction of a part of the anterior wall of the thorax will be the consequence of the contraction of the heart, which, however, should possess a certain amount of energy, distinctly to show the retraction.* The thorax wall will assume its normal position again during the diastole which occurs with a certain shock, with a *diastolic* heart beat, and with the production of a dull sound which, upon auscultation, is audible directly after the second heart sound. As first taught by Friedreich, there occurs, simultaneously with the diastole, a *collapse of the veins of the neck*, because the diastolic springing forward of the thorax wall favours a rapid emptying of the jugular veins. While the simple retraction of the intercostal space at the location of the apex beat is also observed in other rare cases, those retractions of the anterior wall of the thorax synchronously with the diastolic collapse of the jugular veins are symptoms which allow the positive diagnosis of the rigid adherence of the pericardium especially to the surrounding tissues. Of importance is also, above all, the consideration of the fact that a pericarditis preceded the symptoms referred to. If, during the course of such an affection, the apex beat has been plainly visible at first, and has disappeared later upon the cure of the pericarditis and upon normal extension of the limits of the cardiac dulness, the mere absence of the apex beat (a symptom which is very frequently observed under other circumstances and is mostly of no significance) is sufficient under these conditions to diagnosticate pericardial adhesions. This diagnosis will become still more positive if there exists a recoil of the apex or a retraction of the anterior wall of the thorax. The absence of a dislocation of the apex in the left lateral position may also be made use of in the diagnosis of adhesion of the pericardium to adjacent organs. [Occasionally there is a systolic retraction of the tenth and eleventh intercostal spaces on the left side of the chest inferiorly to the scapula. This is said to be due to adhesions of the pericardium to the diaphragm, and was first described by Broadbent (Broadbent's sign). Rarely retraction may also be noted on the right side in the same corresponding area.]

Pulsus Paradoxus.—If the large vessels, the aorta and the superior vena cava, are stretched and narrowed by connective-tissue adhesions due to a pericarditis, or a mediastinitis, there occur peculiar phenomena in the arteries and veins during in-

spiration. While in the healthy, as long as the respiration takes place *quietly*, no difference in the pulse beat can be observed during in- and expiration, or at least only a lowering of the pulse wave is intimated upon inspiration and a raising of the same during expiration; the condition is, however, different in calcareous mediastinal pericarditis. Here there is observed in the sphygmographic picture, or even on palpation, a very remarkable reduction of the pulse during each inspiration; in fact, if the latter is very deep, one even feels the pulse disappear under the palpating finger. This conduct of the pulse (pulsus paradoxus) is due to the narrowing to which the lumen of the aorta is subjected by the stretching adhesions upon inspiration. It is true, the pulsus paradoxus is not pathognomonic of calcareous mediastinal pericarditis, because it has been observed in various other pathological conditions and is bound always to occur when the normal inspiratory lowering of the blood pressure comes into greater prominence. Nevertheless, the pulsus paradoxus is most pronounced in mediastinal pericarditis, and the latter may be diagnosticated if, besides an exquisite pulsus paradoxus, there is simultaneously present another sign of vascular distortion, an inspiratory *distention of the jugular veins.*

Inspiratory Swelling of the Jugular Veins.—This latter symptom can easily be explained by the fact that the large venous trunks become narrowed upon inspiration by tension of the adhesion bands, and with the momentary blood engorgement there occurs a *swelling* of the jugular veins instead of the normal inspiratory *shrinking.*

PNEUMOPERICARDIUM

The presence of air in the pericardium is an exceptionally rare affection, which, however, causes very remarkable objective symptoms. It must not be forgotten in explaining it that analogous to the conduct in pneumothorax (for the origin of which, as I may incidentally state, similar ætiological factors are accountable, as for pneumopericardium), besides gas, also fluid—generally pus—accumulates in the pericardium. Accordingly there is found arching in the cardiac region and absence of the apex beat, which, however, eventually becomes noticeable when the patient sits up or bends forward. As the air in the pericardium ascends while the heart and the exudate descend posteriorly, we find upon *percussion,* in place of cardiac dulness, a clear tympanitic sound with a metallic accessory note (particularly upon pleximetric percussion) which assumes a varying pitch with the systole and diastole of the heart. The sound of a cracked pot has also been heard in pneumopericardium, even with a closed cavity. It is obvious that the fluid and the heart quickly change their location upon change of position of the patient, thus altering the note upon percussion.

Auscultatory Symptoms.—The *auscultatory* symptoms are, if this is possible, still more striking than the percussory; the most important is the metallic character of the heart sounds, which is generally so intense that the loud, ringing sounds can be heard at some distance from the patient. The fluid is generally put into a splashing motion by the moving heart, thus causing a succussion sound, which is also accompanied with a metallic note ("water-wheel murmur"). Neither is the sound of the falling drop wanting, the same as an eventual friction sound, which may have a ringing timbre. If the patient is made to bend forward, the fluid and the heart descend anteriorly, the clear sound in the cardiac region becomes dull, the apex beat palpable, the friction murmur distinct.

In view of the above very peculiar physical findings it is scarcely possible to confound pneumopericardium with any other affection.

feriorly owing to contraction of the auricles immediately before the onset of the systole of the ventricle. This movement of the cardiac apex is essentially the result of the perpendicular position of the axis of the ventricle to the base of the heart, the transverse section of which shows, in place of the elliptic form it ought to have during the diastole, a more circular one during the systole (Ludwig). Thus, and because the ventricular wall then becomes more elastic, it is conceivable that the apex of the heart is displaced anteriorly to such an extent that an arching of the fifth intercostal space, towards which slopes the inclined plane formed by the diaphragm, may occur through the contracting heart situated upon the latter. So much space as is created by the heart diminishing in size, the borders of the lungs occupy by advancing during the systole. As soon as the latter can no longer take place, for instance, in shriveling and coalescence of the borders of the lung, in hypertrophy of the heart, there occur, as there is a frequent opportunity to observe, upon pronounced apex beat, retractions of the intercostal spaces in the entire cardiac region as an expression of the systolic diminution of the heart, which cannot be followed by a corresponding filling of the thus created empty space by the lungs. These retractions should be well differentiated from the *systolic retraction* [dimpling] *of the apex of the heart* with which we have to do.

Systolic Retraction in the Region of the Apex Beat.—This latter occurs only when the locomotion of the heart apex just described is anteriorly impeded. Because in this process of locomotion the base also moves forward, it will be a question, in adherence of the pericardium, whether they affect the basal part of the heart and interfere with its downward motion. As a matter of fact, it has been demonstrated at autopsies that even insignificant adhesions were able to cause systolic retraction of the fifth intercostal space, inasmuch as they affected the base of the heart, while even extensive adhesions at other parts passed without symptoms *intra vitam*. If, on the other hand, the obliterated pericardium presents, in place of the normal loose approximation, an adherence to the anterior wall of the thorax and to the spinal column by means of very tense masses of connective tissue, not only a retraction of the intercostal space *but also a retraction of a part of the anterior wall of the thorax will be the consequence of the contraction of the heart, which, however, should possess a certain amount of energy, distinctly to show the retraction.* The thorax wall will assume its normal position again during the diastole which occurs with a certain shock, with a *diastolic* heart beat, and with the production of a dull sound which, upon auscultation, is audible directly after the second heart sound. As first taught by Friedreich, there occurs, simultaneously with the diastole, a *collapse of the veins of the neck,* because the diastolic springing forward of the thorax wall favours a rapid emptying of the jugular veins. While the simple retraction of the intercostal space at the location of the apex beat is also observed in other rare cases, those retractions of the anterior wall of the thorax synchronously with the diastolic collapse of the jugular veins are symptoms which allow the positive diagnosis of the rigid adherence of the pericardium especially to the surrounding tissues. Of importance is also, above all, the consideration of the fact that a pericarditis preceded the symptoms referred to. If, during the course of such an affection, the apex beat has been plainly visible at first, and has disappeared later upon the cure of the pericarditis and upon normal extension of the limits of the cardiac dulness, the mere absence of the apex beat (a symptom which is very frequently observed under other circumstances and is mostly of no significance) is sufficient under these conditions to diagnosticate pericardial adhesions. This diagnosis will become still more positive if there exists a recoil of the apex or a retraction of the anterior wall of the thorax. The absence of a dislocation of the apex in the left lateral position may also be made use of in the diagnosis of adhesion of the pericardium to adjacent organs. [Occasionally there is a systolic retraction of the tenth and eleventh intercostal spaces on the left side of the chest inferiorly to the scapula. This is said to be due to adhesions of the pericardium to the diaphragm, and was first described by Broadbent (Broadbent's sign). Rarely retraction may also be noted on the right side in the same corresponding area.]

Pulsus Paradoxus.—If the large vessels, the aorta and the superior vena cava, are stretched and narrowed by connective-tissue adhesions due to a pericarditis, or a mediastinitis, there occur peculiar phenomena in the arteries and veins during in-

spiration. While in the healthy, as long as the respiration takes place *quietly*, no difference in the pulse beat can be observed during in- and expiration, or at least only a lowering of the pulse wave is intimated upon inspiration and a raising of the same during expiration; the condition is, however, different in calcareous mediastinal pericarditis. Here there is observed in the sphygmographic picture, or even on palpation, a very remarkable reduction of the pulse during each inspiration; in fact, if the latter is very deep, one even feels the pulse disappear under the palpating finger. This conduct of the pulse (pulsus paradoxus) is due to the narrowing to which the lumen of the aorta is subjected by the stretching adhesions upon inspiration. It is true, the pulsus paradoxus is not pathognomonic of calcareous mediastinal pericarditis, because it has been observed in various other pathological conditions and is bound always to occur when the normal inspiratory lowering of the blood pressure comes into greater prominence. Nevertheless, the pulsus paradoxus is most pronounced in mediastinal pericarditis, and the latter may be diagnosticated if, besides an exquisite pulsus paradoxus, there is simultaneously present another sign of vascular distortion, an inspiratory *distention of the jugular veins.*

Inspiratory Swelling of the Jugular Veins.—This latter symptom can easily be explained by the fact that the large venous trunks become narrowed upon inspiration by tension of the adhesion bands, and with the momentary blood engorgement there occurs a *swelling* of the jugular veins instead of the normal inspiratory *shrinking.*

PNEUMOPERICARDIUM

The presence of air in the pericardium is an exceptionally rare affection, which, however, causes very remarkable objective symptoms. It must not be forgotten in explaining it that analogous to the conduct in pneumothorax (for the origin of which, as I may incidentally state, similar ætiological factors are accountable, as for pneumopericardium), besides gas, also fluid—generally pus—accumulates in the pericardium. Accordingly there is found arching in the cardiac region and absence of the apex beat, which, however, eventually becomes noticeable when the patient sits up or bends forward. As the air in the pericardium ascends while the heart and the exudate descend posteriorly, we find upon *percussion,* in place of cardiac dulness, a clear tympanitic sound with a metallic accessory note (particularly upon pleximetric percussion) which assumes a varying pitch with the systole and diastole of the heart. The sound of a cracked pot has also been heard in pneumopericardium, even with a closed cavity. It is obvious that the fluid and the heart quickly change their location upon change of position of the patient, thus altering the note upon percussion.

Auscultatory Symptoms.—The *auscultatory* symptoms are, if this is possible, still more striking than the percussory; the most important is the metallic character of the heart sounds, which is generally so intense that the loud, ringing sounds can be heard at some distance from the patient. The fluid is generally put into a splashing motion by the moving heart, thus causing a succussion sound, which is also accompanied with a metallic note ("water-wheel murmur"). Neither is the sound of the falling drop wanting, the same as an eventual friction sound, which may have a ringing timbre. If the patient is made to bend forward, the fluid and the heart descend anteriorly, the clear sound in the cardiac region becomes dull, the apex beat palpable, the friction murmur distinct.

In view of the above very peculiar physical findings it is scarcely possible to confound pneumopericardium with any other affection.

Differential Diagnosis.—Only upon very superficial examination is it possible that the resonance of the sounds in the stomach creates the impression of pneumopericardium. I have to admit, though, that at times the ringing of the sounds in this case is very remarkable. But the normal condition of the cardiac dulness and of the apex beat at once undeceive you; the last doubt is dispelled if the stomach is filled with water, thus at once silencing the "chiming of the bells" of the cardiac sounds. A confusion is more liable to occur if the cardiac sounds resound with a metallic quality *in a pulmonary cavity adjacent to the heart* or in a cavity contiguous to the heart, of a *sacculated pneumothorax*. These, however, are very rare occurrences in my experience. Here, too, the significance of the preservation of the cardiac dulness and of the apex beat guard against errors.

If the diagnosis of a pneumopericardium has been made certain, it is necessary to support the diagnosis by inquiring after the cause of the affection—i. e., to find out whether air has entered the pericardial cavity owing to trauma or perforation of an air-containing adjacent organ. In very rare cases it has not been possible at any place to demonstrate at autopsy the loss of continuity of the pericardial wall. There are several positively observed cases in the literature of accumulation of gas in the pericardium by disintegration of a pericardial exudate, and I myself am forced *nolens volens* thus to count a case in my own practice—it was the question of an œsophageal carcinoma which became gangrenous adjacent to the pericardial cavity—in which a pneumopericardium existed without my being able, in spite of the greatest painstaking, to demonstrate at autopsy any communication whatever between œsophagus and pericardial cavity. [In rare cases the gas-producing bacillus (bacillus ærogenes capsulatus) will be found.]

AFFECTIONS OF THE MYOCARDIUM

Affection of the Heart Muscle.—More difficulties, because less distinctly characterized by physical signs, are presented in the diagnosis of *affections of the myocardium*. Of the independent pathological conditions to be considered here there are practically only three of any clinical significance: (1) Myocarditis; (2) ("idiopathic") hypertrophy of the heart, which arises independently of other cardiac affections, and (3) fatty heart. These require special discussion, while all the remaining changes of the myocardium will be referred to only incidentally.

MYOCARDITIS

Myocarditis.—As to myocarditis, it may be stated in advance that it occurs either as a separate affection, or, as has been previously mentioned, frequently combined with endocarditis and pericarditis; therefore the symptoms of the latter quite generally combine themselves with those of myocarditis. The severe *disturbance of function of the affected myocardium* which, according to the character of the individual case, occurs either rapidly or gradually, is to be considered the most prominent symptom of myocarditis.

Changes of the Pulse and Engorgement Symptoms.—This disturbance presents itself *in the first place in the change of the pulse, which becomes small, frequent, and arrhythmic, and,* in contradistinction to conditions of simple relaxation of the overtaxed muscle subsequent to endocarditis, etc., not complicated by myocarditis, *which in the long run is but little*

or not at all affected by digitalis and similar cardiac stimulants. The diagnosis is supported by the other signs of insufficient action of the heart muscle—viz., feeble apex beat, palpitation of the heart, feeling of oppression, asthmatic phenomena, dyspnœa, diminished amounts of urine, disturbances of digestion, cyanosis, and the symptoms of engorgement in general.

Cardiac Murmurs.—The *dilatation* which gradually occurs in the myasthenic heart becomes more and more demonstrable on percussion; the cardiac sounds upon auscultation remain *clear*, or, instead, *murmurs* may be heard, and, besides, a moderate accentuation of the second pulmonary sound can be demonstrated owing to disturbances in the lesser circulation. Krehl has recently called attention to the fact that the closing of the valves essentially depends upon the intact condition of the various parts of the musculature of the heart. Thus it cannot be surprising that if the muscular fibres of the orifices or the papillary muscles are affected, that there may occur relative mitral insufficiency with its sequelæ (systolic murmur at the apex of the heart or at the pulmonary artery, enlargement of the heart with accentuation of the second pulmonary sound), and eventually a relative tricuspid insufficiency with venous pulse, etc. The results of auscultation alone are not sufficient positively to differentiate between endocarditis and myocarditis; *at most it is sometimes possible, from the faintness and the varying audibility of the murmur,* which in some cases of myocarditis may be absent for hours and days, again to appear, in this manner to conclude that a myocarditis is more likely to be present than an endocarditis. However, this differential-diagnostic expedient generally fails entirely if the muscular enfeeblement causes a *constant* insufficiency of the cardiac activity, owing to which permanent murmurs occur. The latter is particularly the case upon the torsion of a valve apex or upon perforation of the ventricular sæptum, etc. If there suddenly occurs in the course of a simple myocarditis, which so far was present with faint but clear cardiac sounds, a very loud cardiac murmur, the above-named complication of myocarditis becomes at least probable.

Emboli in the brain, spleen, etc., which complicate the condition, are of no significance diagnostically, while the fact that a cardiac insufficiency which arises acutely, takes a course with *fever*, may be considered in the diagnosis of an acute myocarditis. The diagnosis is difficult under any circumstances, and can be made with a degree of certainty only if the case has been observed for some time and if its course can be watched minutely.

Ætiological Factors.—The diagnosis becomes more certain by a consideration of the *anamnesis*, especially the simultaneous existence of pathological conditions which are apt to cause myocarditis, for instance, diphtheria, typhoid fever, and, above all, acute articular rheumatism or sepsis. It is true, we know that precisely these infectious diseases also give rise to endo- and pericarditis, but it appears that rheumatic fever affects the endocardium, the other infectious diseases the myocardium, more frequently and more severely.

Differential Diagnosis.—*If the diagnosis is to be made of uncomplicated myocarditis,* it is necessary that the cardiac sounds are to be found pure or that endocarditis can be excluded from the symptoms. If, however, the pronounced signs of endocarditis or pericarditis are encountered,

the question remains whether, besides, a myocarditis is also present. Decisive in this respect, is principally the consideration of the disparity between the symptoms of cardiac insufficiency and the intensity of the unquestionably demonstrable endocarditis and pericarditis. If the former predominate, and if the effect of digitalis and other cardiac stimulants is minimal or negative, the diagnosis of synchronous myocarditis may be made.

Chronic Myocarditis.—If it is a question, not of an acute, but of a chronic cardiac insufficiency, the prospects become still more difficult to recognise in the pathological picture a chronic myocarditis as the principal element of the cardiac affection. If the sounds are more or less clear in an existing cardiac insufficiency taking a chronic course and in dilatation of the organ, there may exist a chronic myocarditis, a fatty heart in the strictest sense, or a secondary fatty degeneration of the heart which has become idiopathically hypertrophic. Very frequently it is impossible to make a positive differential diagnosis in this instance; but the course of the affection and the history are conditions which sometimes are in favour of the one, at other times of the other, affection. In favour of *chronic myocarditis* especially are the absence in the pathological picture of a period during which symptoms of cardiac hypertrophy made themselves unmistakably conspicuous, also the absence of general adiposity. In the diagnosis of chronic myocarditis the following points may be made use of: The demonstration of indubitable delayed symptoms of syphilis, of a former infectious disease, or of an arteriosclerosis, which is pronounced beyond doubt in the peripheral arteries. In the latter case the probable conclusion, at least, is permissible that a coronary arteriosclerosis exists with necrosis of the heart muscle, which is so generally allied with it, and interstitial myocarditis.

Explanation of Cardiac Murmurs in Chronic Myocarditis.—If, upon auscultation, besides a chronic cardiac insufficiency, murmurs are observed in the heart, it is frequently impossible to decide whether the latter are caused by an endocarditis, which became associated with the myocarditis simultaneously or secondarily, or are due to a chronic myocarditis alone. It is true, generally the weak character and the variable nature of the murmur are in favour of its being a myocardial one, as it does not present the shrill note and the very distinct measure characteristic of most valvular murmurs. But in the majority of cases it will be necessary to hold the diagnosis in abeyance.

If in the individual case there have existed for some length of time signs which indicate an uncomplicated myocarditis—small, irregular pulse, etc.—and if these become associated with symptoms of *mitral insufficiency*, the fact is to be taken into consideration that the myocarditic process has been extended to the valvular musculature or that an endocarditis has supervened. As stated above, a *positive* decision as to these two possibilities is almost always out of the question; the simultaneous development of the signs of mitral stenosis is positively in favour of an endocarditic mitral insufficiency. If signs of tricuspid insufficiency occur with diffusion of the cardiac dulness, they are probably, in most instances, nothing but the consequence of a relative tricuspid insufficiency which developed owing to dilatation of the heart caused by the myocarditis. In cases, finally, in which the physical signs of *stenosis*

of the orifices, in particular of the aortic or pulmonary valves, complicate the pre-existing picture of myocarditis, the possibility should be considered of the development of a so-called "stenosis of the heart" owing to calcareous deposits in the heart, narrowing the lumen of the ventricles. But even under these circumstances there cannot be a question of even an approximately correct diagnosis, as the symptoms of a stenosis may also develop in consequence of a simple endocarditis of the respective valves, when up till then a myocarditis had existed. In such cases, therefore, we must postpone a decision; it is sufficient to make the diagnosis of chronic myocarditis with stenosis of the valves.

If the case has not been observed from the onset and in the various forms of its development, it will be best to withhold a positive opinion; this follows from what has been said above, and it becomes entirely obvious from the discussion of the diagnosis of idiopathic hypertrophy of the heart in its later stages, and of fatty heart, the clinical picture of which, as we shall see, coincides in the most essential points with that of chronic myocarditis.

THE FATTY HEART, ADIPOSE DEGENERATION OF THE MUSCULATURE OF THE HEART

The Fatty Heart.—The milder grades of fatty degeneration of the heart[1] cannot be diagnosticated; this also holds good of cases in which a complete fatty metamorphosis of several parts of the heart has actually occurred, but in which the greater part of the muscular structure of the heart still retains its normal condition. In fact, it may occur (as proved by a case which was observed recently in my clinic) that the entire apex of the heart is transformed into one mass of fat without the manifestation of a *single* clinical symptom. In those cases, however, in which there exists a diffuse fatty degeneration of the heart, the diagnosis may be made, with due consideration of the history and constitution of the individual, from certain symptoms and signs which are connected with the altered activity of the heart.

Diagnostic Signs of Fatty Heart.—Here, too, the symptoms of chronic cardiac insufficiency predominate in the picture: The cardiac dulness is increased owing to passive dilatation, the apex beat, at the same time, is weak, diffuse, or entirely absent; the heart sounds are faint, but clear, rarely accompanied by a systolic accidental murmur, the second pulmonary sound but slightly accentuated, unless the fatty degeneration affects only the left ventricle, and the right becomes consecutively hypertrophic. Lesser or greater degrees of engorgement (swelling of the liver, albuminuria, etc.) occur; the radial pulse, owing to the insufficient function of the myocardium, is weak, and in by far the majority of cases accelerated, arrhythmic (probably in consequence of the insufficient blood supply to the brain and the deficient curbing of the activity of the heart by the fibres of vagus caused thereby). Only very rarely there occurs, in my experience, a de-

[1] I consider it more correct, in general, from a clinical standpoint, to drop the customary diagnostic distinction of "fatty heart" and "fatty degeneration of the heart" and only speak of fatty degeneration of the heart, mentioning the particular cause of the fatty degeneration.

the question remains whether, besides, a myocarditis is also present. Decisive in this respect, is principally the consideration of the disparity between the symptoms of cardiac insufficiency and the intensity of the unquestionably demonstrable endocarditis and pericarditis. If the former predominate, and if the effect of digitalis and other cardiac stimulants is minimal or negative, the diagnosis of synchronous myocarditis may be made.

Chronic Myocarditis.—If it is a question, not of an acute, but of a chronic cardiac insufficiency, the prospects become still more difficult to recognise in the pathological picture a chronic myocarditis as the principal element of the cardiac affection. If the sounds are more or less clear in an existing cardiac insufficiency taking a chronic course and in dilatation of the organ, there may exist a chronic myocarditis, a fatty heart in the strictest sense, or a secondary fatty degeneration of the heart which has become idiopathically hypertrophic. Very frequently it is impossible to make a positive differential diagnosis in this instance; but the course of the affection and the history are conditions which sometimes are in favour of the one, at other times of the other, affection. In favour of *chronic myocarditis* especially are the absence in the pathological picture of a period during which symptoms of cardiac hypertrophy made themselves unmistakably conspicuous, also the absence of general adiposity. In the diagnosis of chronic myocarditis the following points may be made use of: The demonstration of indubitable delayed symptoms of syphilis, of a former infectious disease, or of an arteriosclerosis, which is pronounced beyond doubt in the peripheral arteries. In the latter case the probable conclusion, at least, is permissible that a coronary arteriosclerosis exists with necrosis of the heart muscle, which is so generally allied with it, and interstitial myocarditis.

Explanation of Cardiac Murmurs in Chronic Myocarditis.—If, upon auscultation, besides a chronic cardiac insufficiency, murmurs are observed in the heart, it is frequently impossible to decide whether the latter are caused by an endocarditis, which became associated with the myocarditis simultaneously or secondarily, or are due to a chronic myocarditis alone. It is true, generally the weak character and the variable nature of the murmur are in favour of its being a myocardial one, as it does not present the shrill note and the very distinct measure characteristic of most valvular murmurs. But in the majority of cases it will be necessary to hold the diagnosis in abeyance.

If in the individual case there have existed for some length of time signs which indicate an uncomplicated myocarditis—small, irregular pulse, etc.—and if these become associated with symptoms of *mitral insufficiency*, the fact is to be taken into consideration that the myocarditic process has been extended to the valvular musculature or that an endocarditis has supervened. As stated above, a *positive* decision as to these two possibilities is almost always out of the question; the simultaneous development of the signs of mitral stenosis is positively in favour of an endocarditic mitral insufficiency. If signs of tricuspid insufficiency occur with diffusion of the cardiac dulness, they are probably, in most instances, nothing but the consequence of a relative tricuspid insufficiency which developed owing to dilatation of the heart caused by the myocarditis. In cases, finally, in which the physical signs of *stenosis*

of the orifices, in particular of the aortic or pulmonary valves, complicate the pre-existing picture of myocarditis, the possibility should be considered of the development of a so-called "stenosis of the heart" owing to calcareous deposits in the heart, narrowing the lumen of the ventricles. But even under these circumstances there cannot be a question of even an approximately correct diagnosis, as the symptoms of a stenosis may also develop in consequence of a simple endocarditis of the respective valves, when up till then a myocarditis had existed. In such cases, therefore, we must postpone a decision; it is sufficient to make the diagnosis of chronic myocarditis with stenosis of the valves.

If the case has not been observed from the onset and in the various forms of its development, it will be best to withhold a positive opinion; this follows from what has been said above, and it becomes entirely obvious from the discussion of the diagnosis of idiopathic hypertrophy of the heart in its later stages, and of fatty heart, the clinical picture of which, as we shall see, coincides in the most essential points with that of chronic myocarditis.

THE FATTY HEART, ADIPOSE DEGENERATION OF THE MUSCULATURE OF THE HEART

The Fatty Heart.—The milder grades of fatty degeneration of the heart[1] cannot be diagnosticated; this also holds good of cases in which a complete fatty metamorphosis of several parts of the heart has actually occurred, but in which the greater part of the muscular structure of the heart still retains its normal condition. In fact, it may occur (as proved by a case which was observed recently in my clinic) that the entire apex of the heart is transformed into one mass of fat without the manifestation of a *single* clinical symptom. In those cases, however, in which there exists a diffuse fatty degeneration of the heart, the diagnosis may be made, with due consideration of the history and constitution of the individual, from certain symptoms and signs which are connected with the altered activity of the heart.

Diagnostic Signs of Fatty Heart.—Here, too, the symptoms of chronic cardiac insufficiency predominate in the picture: The cardiac dulness is increased owing to passive dilatation, the apex beat, at the same time, is weak, diffuse, or entirely absent; the heart sounds are faint, but clear, rarely accompanied by a systolic accidental murmur, the second pulmonary sound but slightly accentuated, unless the fatty degeneration affects only the left ventricle, and the right becomes consecutively hypertrophic. Lesser or greater degrees of engorgement (swelling of the liver, albuminuria, etc.) occur; the radial pulse, owing to the insufficient function of the myocardium, is weak, and in by far the majority of cases accelerated, arrhythmic (probably in consequence of the insufficient blood supply to the brain and the deficient curbing of the activity of the heart by the fibres of vagus caused thereby). Only very rarely there occurs, in my experience, a de-

[1] I consider it more correct, in general, from a clinical standpoint, to drop the customary diagnostic distinction of "fatty heart" and "fatty degeneration of the heart" and only speak of fatty degeneration of the heart, mentioning the particular cause of the fatty degeneration.

cided, though at times very considerable, slowing of the pulse to 20 beats and less per minute, which is to be accounted for by the insufficient nutrition and imperfect reaction of the myocardium, and which has been incorrectly considered as pathognomonic of the existence of a fatty heart. Sometimes such slowing of the heart's action is the forerunner of cerebral attacks which terminate with phenomena resembling *epilepsy* or apoplexy. They are, the same as other symptoms of the fatty heart, the consequence of deficient blood supply to the brain, which is, above all, proved by the celebrated case of Stokes, in which the patient was able to abort the attack by assuming the knee-elbow position and lowering the head. Still better known than these pseudo-apoplexies is, as a sign of long-lasting cerebral anæmia due to a fatty heart, Cheyne-Stokes respiration with its periodically varying phases of increasing and decreasing respiration, on the one hand, and complete respiratory rest, on the other hand. But neither is the Cheyne-Stokes phenomenon pathognomonic of the fatty heart—according to the cause of its origin it may always occur when a temporary exhaustion of the respiratory centre takes place owing to deficient blood supply and imperfect nutrition of the medulla oblongata. Palpitation of the heart, angina pectoris and dyspnœa, respectively cardiac asthma—i. e., the sequels of cardiac insufficiency brought about by the action of the nervous system—become conspicuous in fatty heart. We shall have to refer to their diagnosis in a separate chapter.

Differential Diagnosis.—The *exact diagnosis* of fatty heart, therefore, presents quite considerable difficulties; *above all it is concentrated upon the determination of the amount of cardiac insufficiency independent of valvular defects.* After this has been ascertained, and after it has also been determined that the insufficiency is not transitory only, being easily removed by digitalis and other cardiac stimulants, but a permanent one, it becomes necessary to search for the cause of the chronic insufficiency of the heart, and the question to be decided is whether a chronic myocarditis is present, in accordance with the points enlarged upon when discussing the diagnosis of this affection. If a myocarditis is improbable, the diagnosis becomes of itself directed to fatty heart, more so, *if ætiological factors prevail which favour the occurrence of fatty heart,* the observation of which factors render the diagnosis of the latter possible above all. Such factors are: Previous poisoning (particularly phosphorus and arsenic) and pronounced constitutional and metabolic diseases: Anæmia, marasmus (in phthisis, cancer, etc.), and, in particular, universal lipomatosis, which, in my opinion, in many cases is not to be considered as the simple result of injudicious eating, but as a permanently incorrect action of metabolism, and as such is pronounced precisely in the changes occurring in the myocardium also; we shall further dwell upon this when discussing the diagnosis of obesity. These latter conditions are the ones the determination of which allows us to make a diagnosis of "fatty heart in the strictest sense" in the respective instances. A very frequent cause of the fatty heart should be mentioned particularly: The fatty degeneration of the muscular structure of the heart following a pre-existing hypertrophy brought about by whatever cause. So long as the hypertrophied muscle

functionates more, according to the greater demands made upon its activity, more fat is destroyed and an accumulation of fat does not occur at the point at which the greater muscular work is performed. If, however, the muscle is stimulated to greater activity, and thus a change is ushered in in the direction of decomposition, in the sense of an increased transformation of fat, and yet the action of the muscle becomes insufficient in view of the obstacles to be overcome, then fat will be deposited undecomposed, and a gradual fatty degeneration of the muscle will take place. This variety of fatty degeneration is observed in the train of cardiac hypertrophy which is formed in the course of valvular affections, but likewise in all other forms of cardiac hypertrophy, to which we shall refer again in detail. In a great number of the so-called idiopathic hypertrophies of the heart, which belong to this category, there are certainly found post mortem, as has been recently demonstrated by Krehl, the anatomical characteristics of a chronic myocarditis. In general, it may be assumed that that part of the heart will become fatty first and to the greatest extent which has to perform the greatest amount of work; usually this will be, as is to be expected, the left ventricle.

SPONTANEOUS RUPTURE OF THE HEART

A rupture of the heart does not occur in a myocardium the texture of which has not been altered. The rupture is always preceded by the fact that the muscle loses its resistance. This occurs most frequently in consequence of atheroma of the coronary arteries, for this causes circumscribed areas of softening or formation of calcareous deposits to arise in the cardiac wall; more rarely it is due to circumscribed fatty degeneration of the heart muscle, to myocarditis, particularly syphilitic myocarditis or formation of gumma, to neoplasms, to abscesses of the heart, and to ulcerations of the cardiac wall. Atheromatous degeneration of the arteries, the most usual cause of rupture of the heart, being an affection of senility, it is comparatively most frequently observed in the aged. The *diagnosis* of rupture of the heart can almost never be made with even a degree of certainty. In some instances the rupture occurs rather suddenly; but here, too, it must be assumed, according to clinical experiences, that the final tearing of the wall of the heart was preceded first by smaller interruptions of continuity or displacements of a coronary artery, and to these are to be referred the feeling of oppression, præcordial anxiety, dyspnœa which, after prevailing for a short while or for several days, are followed by the final catastrophe (sudden collapse, pain in the cardiac region, acute cyanosis, absence of pulse, convulsions, rapid death). Rupture of the heart is to be *presumed*, if the last-named symptoms were undubitably preceded by those of chronic myocarditis with or without endo- and pericarditis, of fatty heart or of visceral syphilis, or if in cases in which the course of the cardiac rupture becomes protracted for some length of time, an increasing accumulation of the blood in the pericardial cavity makes itself known by means of physical diagnosis in a diffusion of the cardiac dulness.

HYPERTROPHY OF THE HEART

Hypertrophy of the ventricles and auricles as a sequel to the different affections of the heart was variously considered in the discussion of the latter conditions. Its recognition forms an integral part of the diagnosis of the latter, and therefore shall not be referred to again. But, on the other hand, hypertrophy of the heart also develops from other causes: Abnormal resistance owing to congenital stenosis of the aorta, arteriorsclerosis, aneurysm, in which cases principally the left ventricle hypertrophies, while

the right ventricle becomes hypertrophied in affections of the lung with circulatory disturbances in the lesser circulation, for instance, in pulmonary emphysema. All these conditions have, as a common cause of hypertrophy of the heart, the mechanical factor of circulatory disturbance, increased resistances in the vascular system.

More difficult to explain is the origin of hypertrophy of the heart in consequence of heavy physical labour. Every one knows that the pulse beats stronger and the pulsating heart becomes palpable upon vigorous muscular exercise, that the *frequency of the pulse increases, and the arterial blood pressure rises.* This is the result of experience of physicians and physiologists. This might be the consequence of irritation of the vaso-motor system, a direct or indirect effect of products of metabolism formed by the action of the muscles, or else be caused by transmission upon the heart of irritation of centripetal nerves upon muscular contraction. The physiological experiments, which were recently made by J. Jacob, systematically excluding the various influences which play a rôle in it, have shown that the increase in the pulse frequency is not caused by products of metabolism, but is a reflected one, *issuing from the sensory muscle nerves,* their irritation reflexly exciting the accelerator nerves. Increase of the blood pressure particularly must be considered a consequence of a reflex irritation of the vaso-motor centre, the effect of which, on great muscular contraction, overbalances the opposing pressure-reducing influence of the muscular vaso-dilators, which in the active muscle is normally brought about by the vaso-dilators. As always, when the increase in cardiac activity persists for some time, here, too, an accommodation of the heart to the increased demands occurs in such a manner that its muscular structure hypertrophies. This so-called "occupation hypertrophy of the heart" is principally found in people who, voluntarily or professionally, are subject to constant corporeal labour, in sportsmen, blacksmiths, wine-growers, soldiers, etc.

In a similar manner—i. e., under the influence of chemical and mechanical causes—it appears that cardiac *hypertrophy* is brought about *in patients afflicted with nephritis.* As here, in at least one half of the cases, only the left ventricle is found to be hypertrophied, it can be assumed that mechanical conditions in the arterial system, especially a pathological increase of the blood pressure, are the cause of hypertrophy of the left ventricle.

The most obvious supposition, that the destruction or the compression of numerous blood vessels in the atrophied, respectively inflamed, *renal parenchyma* increases the blood pressure (Traube), has apparently been proved to be untenable, after it has been demonstrated experimentally that the ligature of both renal arteries is not followed by an increase in blood pressure in the aorta. On the other hand, the fact that the injection of urea into the blood produces a transitory arterial spasm, suggests another explanation of the origin of hypertrophy of the heart—namely, that the surcharging of the blood with excrementitious materials causes in nephritis a contraction of the small arteries, a consecutive increase of the blood pressure, and a gradually occurring hypertrophy of the heart. The correctness of this assumption, the primary contraction of the small arteries through *chemical* irritation, is decidedly favoured by the *clinical* facts; above all, by the observation of Riegel

be looked for solely in pathological activity of the heart, have been designated as cardiac asthma in contradistinction to bronchial asthma. The lungs in cardiac asthma are normal in their anatomical conditions, or they only show changes which may be referred to every abnormal activity of the heart.

S. von Basch has, in his time, thoroughly investigated the condition of blood pressure and circulation, and has brought them in closer connection with physiological experiences, so that we have a better understanding of the occurrences in cardiac asthma.

The elasticity of the alveolar walls decreases with a greater filling of the capillaries of the lungs, and greater demands are accordingly made upon the powers of the inspiratory muscles as soon as the pressure increases in the pulmonary capillaries. This, however, will take place either in increasing or in decreasing pressure of the aorta; for, according to the positive demonstration of von Basch, an increasing blood pressure in the arteries, which may be accomplished by irritation of the vascular nerve centre, is combined with an increase of pressure in the pulmonary artery, thus producing a better filling of the capillaries of the pulmonary alveoli, causing the wall of the latter to become more *rigid* and diminishing the respiratory capacity of the lungs. We see a predisposition to dyspnœa, which is brought about in such a manner, occur, for instance, in patients with contracted kidneys, or in certain cases of arteriosclerosis. In *decreasing* blood pressure in the aorta, on the other hand, as is generally the case in valvular defects and cardiac insufficiency, the distribution of the blood is so changed that the venous system and the lesser circulation contain more blood, the pressure in the pulmonary artery increases, and the second pulmonary sound becomes accentuated; therefore, here also a better filling of the pulmonary capillaries, a greater rigidity, and increase in volume of the lungs result, and with it a constant predisposition to *cardiac dyspnœa*.

This deficiency may, however, be *compensated* if, in the same proportion as the lungs become less distended, owing to the above-named conditions, the inspiratory muscles act more energetically, and the expiratory muscles interfering with the process of respiration aid in producing a diminution in the size of the lungs. But every factor which prevents this compensation will cause the dyspnœa to become more rapidly prominent; for instance, the weakness of the respiratory muscular structure in debilitated or obese individuals, the diminished distensibility of the thorax or of the lung in the recumbent position or in a high position of the diaphragm due to accumulation of gas in the intestine, abnormal distention of the stomach, etc.

Under conditions, then, in which the heart *rapidly* changes its "condition of equilibrium," and that in the sense of a weakening of its activity, there occurs a sudden increase in the blood pressure of the pulmonary capillaries in individuals who, owing to contracted kidneys, arteriosclerosis, cardiac defects, lipomatosis, etc., incline to dyspnœa, that a suddenly appearing asthma—cardiac *asthma*—is the natural consequence. Such an abrupt change in the equilibrium of the heart may occur in a paresis as well as in a spasm of the heart, especially of the left ventricle. The final effect regarding the filling of the pulmonary capillaries will be the same in either case—namely, a greater filling of the left auricle and of the capillaries of the lungs and a distention and rigidity of the alveolar walls, which means dyspnœa. The left ventricle is dilated in paretic cardiac asthma, which is the decidedly much more frequent form of cardiac asthma, while in the spasmodic form this is not the case. The first, the *paretic*, form will occur if the left ventricle is to overcome unusual resistance and is well filled. As soon now as a new demand upon the activity of the heart supervenes upon the existing resistance more or less suddenly, the consequence

They have been repeatedly discussed by us and will be only briefly recapitulated here:

Diagnostic Signs of Hypertrophy of the Left Ventricle.—The *hypertrophy of the left ventricle* manifests itself by the following signs and symptoms: Apex beat lower and displaced to the left—i. e., in the sixth to eighth intercostal space towards the left axillary line—palpable in greater diffusion, strong, heaving; the entire cardiac region often noted in diffuse pulsation. Cardiac dulness diffused, principally downward and to the left, caused almost exclusively by the synchronous dilatation; loud cardiac sounds, especially, also, accentuated second aortic sound, sometimes even palpable in the second intercostal space to the right. The first sound at the apex of the heart sometimes markedly ringing, owing to strong systolic vibrations of the thoracic wall (Laennec's cliquetis métallique), radial pulse very tense, the carotids visibly strongly pulsating, two loud sounds audible upon auscultation of the same. At the same time flow of blood to the head, vertigo, *muscæ volitantes*, tinnitus, tendency to hæmorrhages, in particular to cerebral hæmorrhages.

Diagnostic Signs of Hypertrophy of the Right Ventricle.—*Hypertrophy of the right ventricle*: Cardiac dulness diffused to the right as far as the right border of the sternum and even beyond (due to dilatation); the apex beat in the fifth intercostal space displaced outward. Pulsation of the cardiac region particularly marked in the lower portion and in the epigastric region; accentuated second pulmonary sound. Engorgement of the lesser circulation, inclination to dyspnœa, to hæmorrhages from the pulmonary vessels, and to bronchitis.

Frequently a combination of both conditions can be determined. As has been stated above, upon longer existence of the hypertrophy and continuation of the originating factors of the same there occurs insufficiency of ventricular activity with the well-known symptoms of disturbed circulation.

Errors in diagnosis of hypertrophy of the heart may occur in pericardial exudates (see above, Differential Diagnosis), in covering of the large heart by emphysematous borders of the lung, so that the student is often surprised at the unexpectedly large size of the heart which is found at the autopsy of emphysematous patients. *Vice versa,* a heart of normal size may impress one as hypertrophied, if abnormal dulness occurs in the region of the cardiac dulness (owing to sacculated pleuritic exudates, aneurysms, and mediastinal tumours, especially if they press the heart forward), or if dulness of the heart appears enlarged in consequence of a considerable baring of the heart in atrophic conditions of the lungs. The main rule in such cases should be to observe the position and force of the apex beat, which under all circumstances should have left its normal position and *should occupy the outermost border of the dulness*, if the diagnosis of hypertrophy of the heart is to be well substantiated.

Whatever else occurs of changes in the muscular structure of the heart—rare processes of degeneration, neoplasms (carcinoma, etc.), and parasites (echinococcus, etc.), generally cause very indistinct pathological manifestations, so that here there can be no question of an absolute *diagnosis.*

On the other hand, departures from the normal condition of the heart caused by disturbances in innervation play so important a *rôle* in the domain of cardiac diseases either as important pathological symptoms or as independent affections, that we must devote particular attention and an exhaustive discussion to the "*neuroses of the heart.*"

THE NEUROSES OF THE HEART

NERVOUS PALPITATION OF THE HEART

The diagnosis of "nervous palpitation of the heart" does not present difficulties, if only such cases are included, in which attacks occur of increased frequency in the contractions of the heart, which affect the patient in a disagreeable manner, while no organic change in the heart, especially no diffusion of the cardiac dulness, can be demonstrated. It is of minor importance whether systolic "accidental" murmurs can be heard in these cases or not. As anæmia is a main cause of palpitation of the heart and of accidental murmurs, it is self-evident that both manifestations may occur in the same case. On the other hand, the demonstration of a diastolic murmur excludes the diagnosis of simply nervous palpitation of the heart. If no murmur is audible in the furious activity of the heart, myocarditis or fatty heart might be considered in the differential diagnosis. The difficulty in differentiating these two affections of the heart from nervous palpitation is, in my experience, more imaginary than real. The absence of dilatation and of stasis symptoms, and the fact that attacks of palpitation alternate with longer or shorter intervals during which the patient is free from palpitation, generally cause the character of the cardiac affection at once to be recognised as of nervous origin. The case becomes more complicated if the frequent pulse is intermittent during the attacks, or becomes arrhythmical. But here, too, the above-mentioned characteristics cause the diagnosis to become positive, the more so if the ætiology of nervous palpitation of the heart is sufficiently considered. In this affection it is generally a question of anæmic or, beyond doubt, nervous individuals, in whom the most various causes—irritation of the uterine, gastric nerves, etc., intoxications (after the use of coffee, tea, and tobacco), disturbances of metabolism, and, above all, psychic emotions of various kinds produce the palpitations.

I believe that the cause of nervous palpitation of the heart, and the manifestations associated with it, has been rendered more easy of comprehension by the investigations of His and Romberg, on the innervation of the heart. These authors found that the ganglia of the heart are sympathetic throughout, and for this reason (on account of embryological facts) they conceive them as *sensory*. In connection herewith would be that, if the greater irritability of the nerves, which is generally a peculiarity of anæmia, would extend to the ganglia and sensory nerves of the heart, this would produce the sensation of palpitation of the heart and (originating from the same source reflexly by stimulation of the accelerans) the increased activity of the heart.

ANGINA PECTORIS—STENOCARDIA

The symptoms of *angina pectoris* which occurs in attacks are very marked, as a rule—viz., most violent substernal *pain in the cardiac region, great anxiety and feeling of oppression, radiation of the pain* to the left arm, especially along the course of the left ulnar, rarely of other nervous tracts. Accessory symptoms are: Perspiration, paleness of the face, fainting, vaso-motor disturbances and spasm, urina spastica, etc. *The conduct of the pulse and of the action of the heart* varies in the individual cases.

DIAGNOSIS OF THE DISEASES OF THE LARGE VESSELS

ATHEROMA OF THE ARTERIES, ARTERIOSCLEROSIS

The atheromatous condition of the arteries is easy of recognition in so far as it concerns the visible and palpable peripheral vessels: *The arteries appear tortuous, pulsate visibly, and are hard and uneven to the touch; the pulse is tense and slow.*

Sphygmographic Pulse Picture in Arteriosclerosis.—This pulsus tardus is characterized in the sphygmographical picture by a long-drawn ascending line, a consequence of the diminished elasticity of the vascular wall, and by a *broad top,* inasmuch as the elasticity which is wanting causes the expanded artery to remain longer in its condition of dilatation and only gradually to return to its position of rest. At the same time, in the descending line the elevations are entirely absent or are only slightly indicated. The characteristics named leave no doubt in the individual case regarding the presence of an arteriosclerosis, and most of the grave subsequent symptoms may, under such circumstances, naturally be referred to those changes in the vessel wall.

On the other hand, it is often difficult, if the palpable peripheral arteries do not show pronounced signs of atheroma, to decide whether certain pathological symptoms of a more serious character may be considered as depending upon an atheroma of the vessels in the thorax, brain, etc.

General Sequential Symptoms: Hypertrophy of the Left Ventricle, etc.—One of these subsequent symptoms of arteriosclerosis, which occurs, not exactly rarely, but by far not so often as is generally supposed, is *hypertrophy of the heart,* especially of the left ventricle. If the latter can be demonstrated beside atheroma, it is a question whether it is the consequence of the atheroma or whether the latter, the same as the hypertrophy of the heart, is a coeffect of the same ætiological factors, or, finally, whether the atheroma is the result of blood pressure permanently increased by the idiopathic hypertrophy of the heart. The decision of these questions is often impossible in the individual case, besides, clinically, rather unimportant. The hypertrophy of the heart which is associated with atheroma of the arteries manifests itself by increased apex beat and accentuated second aortic sound, also by increase of the limits of percussion of cardiac dulness. It is true, sometimes the latter is not demonstrable, as a simultaneous pulmonary emphysema—arteriosclerosis mostly affects the aged in whom pulmonary emphysema is, as is well known, of very usual occurrence—masks the increase of the cardiac dulness.

Cardiac Murmurs.—Sometimes *murmurs* are found instead of the pure sounds in the heart and over the aorta; then it is a question whether they are caused by changes in the valvular apparatus or solely by the atheroma of the aorta. There can be no doubt regarding the cause of an eventual

diastolic murmur. It may always be considered as the consequence of an aortic insufficiency, which is not infrequent in atheroma, if a simultaneously existing aneurysmal sac is not considered, in which, in rare cases, diastolic murmurs may occur with intact aortic valves. The diagnosis will be more difficult if the murmurs occur with the *systole of the heart*. It is true, they, too, may be caused by an encroachment of the atheromatous process upon the semilunar valves and a stenosis of the aortic orifice may be produced thereby. However, even with intact valves a systolic murmur may be audible, if a more or less pronounced distention of the lumen of the aorta occurs and with it are given the conditions previously discussed for the formation of a murmur.

Which of these possibilities exists in an individual case is decided by other symptoms and signs: The demonstration of dulness on percussion over the manubrium sterni points to aneurysm, the gradual growth and subsequent symptoms of which render the diagnosis more and more certain. Stenosis of the aortic orifice causes so loud a murmur that confusion with the dull, murmur-like sound, which is brought about by the atheroma of the wall of the aorta as such, is scarcely possible, especially because the relatively weak or missing apex beat of the hypertrophied heart in stenosis of the aortic orifice, and also the remaining symptoms of the same, point directly to those as the cause of the systolic murmur.

Cardiac Insufficiency owing to Atheroma.—As long as the hypertrophied heart completely compensates the abnormal resistances in the aortic system, there are no subjective symptoms on the part of the patient. But as compensation relaxes, the symptoms of insufficiency of the cardiac activity, although in quite a moderate degree at first, will occur—i. e., upon greater muscular efforts sensations of oppression in the chest and dyspnœa become manifest, stasis in the system of the pulmonary vessels, chronic bronchitis, vertigo, etc. A graver pathological picture occurs if the heart does not become weak very gradually, but transitorily relaxes *acutely and* an attack of cardiac asthma or of angina pectoris becomes imminent thereby. Later the frequently described symptoms of permanent cardiac weakness occur, engorgement-œdema, and diminished secretion of urine, etc.

Particularly injurious, on account of damaging the nutrition of the most vital organs of the body, is atheromatous degeneration of the coronary arteries of the heart and of the cerebral arteries.

Atheroma of the Coronary Arteries.—*Sclerosis of the coronary arteries* is of frequent occurrence; its consequence is usually necrosis of the muscular structure of the heart and interstitial myocarditis. The *diagnosis* of atheroma of the coronary arteries is not at all a positive one; but in many cases it may be made with a certain degree of probability, when palpitation of the heart occurs, the pulse becomes abnormally slow or irregular and loses in strength, while the peripheral arteries are hard and tortuous to the touch, in which case a lessening in the pulse frequency to 20 beats per minute and below can be observed. With bradycardia there may occur attacks resembling apoplexy or epilepsy, which frequently recur without giving rise to paralysis. During the interval, owing to greater demands upon the activity of the heart, attacks of cardiac asthma and angina pec-

DIAGNOSIS OF THE DISEASES OF THE LARGE VESSELS

ATHEROMA OF THE ARTERIES, ARTERIOSCLEROSIS

The atheromatous condition of the arteries is easy of recognition in so far as it concerns the visible and palpable peripheral vessels: *The arteries appear tortuous, pulsate visibly, and are hard and uneven to the touch; the pulse is tense and slow.*

Sphygmographic Pulse Picture in Arteriosclerosis.—This pulsus tardus is characterized in the sphygmographical picture by a long-drawn ascending line, a consequence of the diminished elasticity of the vascular wall, and by a *broad top,* inasmuch as the elasticity which is wanting causes the expanded artery to remain longer in its condition of dilatation and only gradually to return to its position of rest. At the same time, in the descending line the elevations are entirely absent or are only slightly indicated. The characteristics named leave no doubt in the individual case regarding the presence of an arteriosclerosis, and most of the grave subsequent symptoms may, under such circumstances, naturally be referred to those changes in the vessel wall.

On the other hand, it is often difficult, if the palpable peripheral arteries do not show pronounced signs of atheroma, to decide whether certain pathological symptoms of a more serious character may be considered as depending upon an atheroma of the vessels in the thorax, brain, etc.

General Sequential Symptoms: Hypertrophy of the Left Ventricle, etc.—One of these subsequent symptoms of arteriosclerosis, which occurs, not exactly rarely, but by far not so often as is generally supposed, is *hypertrophy of the heart,* especially of the left ventricle. If the latter can be demonstrated beside atheroma, it is a question whether it is the consequence of the atheroma or whether the latter, the same as the hypertrophy of the heart, is a coeffect of the same ætiological factors, or, finally, whether the atheroma is the result of blood pressure permanently increased by the idiopathic hypertrophy of the heart. The decision of these questions is often impossible in the individual case, besides, clinically, rather unimportant. The hypertrophy of the heart which is associated with atheroma of the arteries manifests itself by increased apex beat and accentuated second aortic sound, also by increase of the limits of percussion of cardiac dulness. It is true, sometimes the latter is not demonstrable, as a simultaneous pulmonary emphysema—arteriosclerosis mostly affects the aged in whom pulmonary emphysema is, as is well known, of very usual occurrence—masks the increase of the cardiac dulness.

Cardiac Murmurs.—Sometimes *murmurs* are found instead of the pure sounds in the heart and over the aorta; then it is a question whether they are caused by changes in the valvular apparatus or solely by the atheroma of the aorta. There can be no doubt regarding the cause of an eventual

diastolic murmur. It may always be considered as the consequence of an aortic insufficiency, which is not infrequent in atheroma, if a simultaneously existing aneurysmal sac is not considered, in which, in rare cases, diastolic murmurs may occur with intact aortic valves. The diagnosis will be more difficult if the murmurs occur with the *systole of the heart*. It is true, they, too, may be caused by an encroachment of the atheromatous process upon the semilunar valves and a stenosis of the aortic orifice may be produced thereby. However, even with intact valves a systolic murmur may be audible, if a more or less pronounced distention of the lumen of the aorta occurs and with it are given the conditions previously discussed for the formation of a murmur.

Which of these possibilities exists in an individual case is decided by other symptoms and signs: The demonstration of dulness on percussion over the manubrium sterni points to aneurysm, the gradual growth and subsequent symptoms of which render the diagnosis more and more certain. Stenosis of the aortic orifice causes so loud a murmur that confusion with the dull, murmur-like sound, which is brought about by the atheroma of the wall of the aorta as such, is scarcely possible, especially because the relatively weak or missing apex beat of the hypertrophied heart in stenosis of the aortic orifice, and also the remaining symptoms of the same, point directly to those as the cause of the systolic murmur.

Cardiac Insufficiency owing to Atheroma.—As long as the hypertrophied heart completely compensates the abnormal resistances in the aortic system, there are no subjective symptoms on the part of the patient. But as compensation relaxes, the symptoms of insufficiency of the cardiac activity, although in quite a moderate degree at first, will occur—i. e., upon greater muscular efforts sensations of oppression in the chest and dyspnœa become manifest, stasis in the system of the pulmonary vessels, chronic bronchitis, vertigo, etc. A graver pathological picture occurs if the heart does not become weak very gradually, but transitorily relaxes *acutely and* an attack of cardiac asthma or of angina pectoris becomes imminent thereby. Later the frequently described symptoms of permanent cardiac weakness occur, engorgement-œdema, and diminished secretion of urine, etc.

Particularly injurious, on account of damaging the nutrition of the most vital organs of the body, is atheromatous degeneration of the coronary arteries of the heart and of the cerebral arteries.

Atheroma of the Coronary Arteries.—*Sclerosis of the coronary arteries* is of frequent occurrence; its consequence is usually necrosis of the muscular structure of the heart and interstitial myocarditis. The *diagnosis* of atheroma of the coronary arteries is not at all a positive one; but in many cases it may be made with a certain degree of probability, when palpitation of the heart occurs, the pulse becomes abnormally slow or irregular and loses in strength, while the peripheral arteries are hard and tortuous to the touch, in which case a lessening in the pulse frequency to 20 beats per minute and below can be observed. With bradycardia there may occur attacks resembling apoplexy or epilepsy, which frequently recur without giving rise to paralysis. During the interval, owing to greater demands upon the activity of the heart, attacks of cardiac asthma and angina pec-

DIAGNOSIS OF THE DISEASES OF THE LARGE VESSELS

ATHEROMA OF THE ARTERIES, ARTERIOSCLEROSIS

The atheromatous condition of the arteries is easy of recognition in so far as it concerns the visible and palpable peripheral vessels: *The arteries appear tortuous, pulsate visibly, and are hard and uneven to the touch; the pulse is tense and slow.*

Sphygmographic Pulse Picture in Arteriosclerosis.—This pulsus tardus is characterized in the sphygmographical picture by a long-drawn ascending line, a consequence of the diminished elasticity of the vascular wall, and by a *broad top*, inasmuch as the elasticity which is wanting causes the expanded artery to remain longer in its condition of dilatation and only gradually to return to its position of rest. At the same time, in the descending line the elevations are entirely absent or are only slightly indicated. The characteristics named leave no doubt in the individual case regarding the presence of an arteriosclerosis, and most of the grave subsequent symptoms may, under such circumstances, naturally be referred to those changes in the vessel wall.

On the other hand, it is often difficult, if the palpable peripheral arteries do not show pronounced signs of atheroma, to decide whether certain pathological symptoms of a more serious character may be considered as depending upon an atheroma of the vessels in the thorax, brain, etc.

General Sequential Symptoms: Hypertrophy of the Left Ventricle, etc.—One of these subsequent symptoms of arteriosclerosis, which occurs, not exactly rarely, but by far not so often as is generally supposed, is *hypertrophy of the heart*, especially of the left ventricle. If the latter can be demonstrated beside atheroma, it is a question whether it is the consequence of the atheroma or whether the latter, the same as the hypertrophy of the heart, is a coeffect of the same ætiological factors, or, finally, whether the atheroma is the result of blood pressure permanently increased by the idiopathic hypertrophy of the heart. The decision of these questions is often impossible in the individual case, besides, clinically, rather unimportant. The hypertrophy of the heart which is associated with atheroma of the arteries manifests itself by increased apex beat and accentuated second aortic sound, also by increase of the limits of percussion of cardiac dulness. It is true, sometimes the latter is not demonstrable, as a simultaneous pulmonary emphysema—arteriosclerosis mostly affects the aged in whom pulmonary emphysema is, as is well known, of very usual occurrence—masks the increase of the cardiac dulness.

Cardiac Murmurs.—Sometimes *murmurs* are found instead of the pure sounds in the heart and over the aorta; then it is a question whether they are caused by changes in the valvular apparatus or solely by the atheroma of the aorta. There can be no doubt regarding the cause of an eventual

diastolic murmur. It may always be considered as the consequence of an aortic insufficiency, which is not infrequent in atheroma, if a simultaneously existing aneurysmal sac is not considered, in which, in rare cases, diastolic murmurs may occur with intact aortic valves. The diagnosis will be more difficult if the murmurs occur with the *systole of the heart*. It is true, they, too, may be caused by an encroachment of the atheromatous process upon the semilunar valves and a stenosis of the aortic orifice may be produced thereby. However, even with intact valves a systolic murmur may be audible, if a more or less pronounced distention of the lumen of the aorta occurs and with it are given the conditions previously discussed for the formation of a murmur.

Which of these possibilities exists in an individual case is decided by other symptoms and signs: The demonstration of dulness on percussion over the manubrium sterni points to aneurysm, the gradual growth and subsequent symptoms of which render the diagnosis more and more certain. Stenosis of the aortic orifice causes so loud a murmur that confusion with the dull, murmur-like sound, which is brought about by the atheroma of the wall of the aorta as such, is scarcely possible, especially because the relatively weak or missing apex beat of the hypertrophied heart in stenosis of the aortic orifice, and also the remaining symptoms of the same, point directly to those as the cause of the systolic murmur.

Cardiac Insufficiency owing to Atheroma.—As long as the hypertrophied heart completely compensates the abnormal resistances in the aortic system, there are no subjective symptoms on the part of the patient. But as compensation relaxes, the symptoms of insufficiency of the cardiac activity, although in quite a moderate degree at first, will occur—i. e., upon greater muscular efforts sensations of oppression in the chest and dyspnœa become manifest, stasis in the system of the pulmonary vessels, chronic bronchitis, vertigo, etc. A graver pathological picture occurs if the heart does not become weak very gradually, but transitorily relaxes *acutely and* an attack of cardiac asthma or of angina pectoris becomes imminent thereby. Later the frequently described symptoms of permanent cardiac weakness occur, engorgement-œdema, and diminished secretion of urine, etc.

Particularly injurious, on account of damaging the nutrition of the most vital organs of the body, is atheromatous degeneration of the coronary arteries of the heart and of the cerebral arteries.

Atheroma of the Coronary Arteries.—*Sclerosis of the coronary arteries* is of frequent occurrence; its consequence is usually necrosis of the muscular structure of the heart and interstitial myocarditis. The *diagnosis* of atheroma of the coronary arteries is not at all a positive one; but in many cases it may be made with a certain degree of probability, when palpitation of the heart occurs, the pulse becomes abnormally slow or irregular and loses in strength, while the peripheral arteries are hard and tortuous to the touch, in which case a lessening in the pulse frequency to 20 beats per minute and below can be observed. With bradycardia there may occur attacks resembling apoplexy or epilepsy, which frequently recur without giving rise to paralysis. During the interval, owing to greater demands upon the activity of the heart, attacks of cardiac asthma and angina pec-

toris will set in. If in this picture there occurs an unjustified *sudden* collapse and *acute* decrease of the pulse regarding its strength and frequency, and if this is quickly followed by death, a thrombosis of the coronary arteries should be thought of. Sometimes the symptoms preceding the catastrophe are of an extremely insignificant nature, and death occurs almost without warning; in other cases sclerosis of the coronary arteries runs quite a chronic course resembling the clinical picture of chronic myocarditis (see p. 50).

Other Sequential Symptoms of Arteriosclerosis.—*Atheroma of the cerebral arteries,* finally, is, as will be but briefly mentioned here, quite generally the cause of cerebral hæmorrhages and softening of the brain with their accompanying symptoms. Here, too, the atheromatous changes of the peripheral arteries with their sequels are almost always very pronounced. To the latter belong also, among other signs, a uniform decrease of the motor ability of the muscles of the extremities, which manifests itself in the lower extremities on walking and standing, as stiffness and weakness of the legs, especially also in the form of "intermittent claudication"; in other cases a spontaneous *gangrene* of the extremities may develop.

AORTIC ANEURYSM

Aortic aneurysm generally occurs as the result of atheroma. It is particularly the result of syphilitic affection of the arteries, and the just-described symptoms may, therefore, indirectly also be used in the diagnosis of aneurysm. The symptoms of developed aneurysm are, as a rule, extremely characteristic; they are, however, not always fully developed, so that usually we must be contented with a portion of the symptoms from which to make the diagnosis. In fact, at the beginning of the formation of an aneurysm, or when the aneurysm is only small, a diagnosis is not at all possible.

Pulsation.—The latter is easy when the distention of the intrathoracic aorta, which is to be first described, grows to the formation of a more or less large, visibly *pulsating tumour,* which, after eroding the ribs or the sternum, and after disappearance of pressure of the muscular structure, becomes situated immediately under the skin. The latter is smooth, glistening, tense over the surface of the tumour, thin, turns reddish gradually and gangrenous before perforation. *Pulsations of the tumour* are in all directions, as well from above downward as *lateral* [expansile], while pulsations which are communicated to tumours of a different character by the vessels situated below them, appear to originate only from one side. On palpation of the pulsating tumour a systolic shock is felt dilating the aneurysm, not infrequently also a double shock; the second weaker one is the usual back stroke caused by the closing aortic valves. The systolic shock may be so strong that its force surpasses even that of the apex beat of the heart. Sometimes there is felt, in place of the pronounced shock, a distinct oscillation, produced by the vibrations of the aortic wall which arise in the suddenly widened channel of the blood current.

Auscultation, Murmurs.—On auscultation there is heard also, for the

same reason, a systolic heart murmur; a diastolic murmur, too, is sometimes observed owing to the vibrations of the aneurysm wall, which is brought about by the blood current regurgitating during the diastole through the relatively narrow isthmus into the wide sac of the aneurysm of the aortic arch (respectively of the ascending aorta). Of course, the murmurs may also be transmitted from the aortic valves if they are degenerated and have given rise to stenosis of the orifice or to insufficiency of the valves, a combination which is the more apt to occur in aortic aneurysm, because the latter, the same as aortic defects, is usually a consequence of atheroma. Besides, in place of the murmurs there are not infrequently two clear sounds audible. This can be especially looked for when the aneurysmal sac attains its width quite gradually, or when thrombi, becoming deposited in layers on the inner wall, so narrow the lumen of the aneurysm that it fully or almost equals the original lumen of the aorta. But percussion will, in such cases, as in aneurysm generally, still show more or less considerable dulness over the aneurysm.

If the aneurysm does not form a pulsating tumour, the most important palpatory symptoms available for the diagnosis will be wanting. But the aneurysm of the aortic arch may still be made accessible to palpation also in cases in which it has not attained a large enough size to adjoin the thoracic wall, and that from the jugular fossa, inasmuch as the finger placed downward in the same reaches the level of the aneurysm. On the other hand, the percussory and auscultatory signs remain the same as in the pulsating tumour. Above all, there also exist *pressure symptoms* which are caused by compression of the organs adjoining the aneurysm.

Pressure Symptoms.—Apart from the pain, which is of very little significance for the diagnosis, primarily *dyspnœa* and the *asthmatic* symptoms are to be considered here. The course of the former may resemble the clinical picture of a bronchiostenosis if the trachea or a bronchus, especially the left bronchus nearest to the aortic arch, be compressed. In my experience, it is particularly suspicious *if asthmatic phenomena occur upon a change of position of the patient.* If an aneurysm of the aortic arch exercises a pressure upon the left bronchus, the latter, and with it the larynx, is pushed downward with every systolic pulsation of the aneurysm. This becomes perceivable at the trachea and at the larynx by a pulsatory *downward movement* of the same, and becomes particularly distinct if the patient is made to raise his chin, and if the cricoid cartilage is carefully lifted slightly upward (*Oliver's symptom*) [tracheal tugging].

Not rarely one finds, in the laryngoscopical picture, paralysis of the vocal cords, generally only of the left cord, as the *left recurrent laryngeal nerve* takes an upward direction posteriorly to the arch of the aorta and naturally is much more frequently subject to the pressure of aneurysm than the right recurrent laryngeal nerve, which is only indirectly affected by aortic aneurysms when the innominate artery takes part in the aneurysmal distention. As an early effect of the pressure of an aneurysm developing in the neighbourhood of the recurrent laryngeal nerve there may occur a spasm of the glottis in the form of periodically recurring attacks of suffocation.

In a similar manner as the recurrent laryngeal nerve, other nerves may be subject to pressure, for instance, the intercostal nerves; eventually also the bone marrow, after erosion of the vertebræ of the spinal cord, may be pressed upon (paralysis of the lower extremities, etc.). Compression of the *vagus* causes vomiting, lesion of sympathetic fibres, a change in the *size* of the pupils, narrowing of the œsophagus, dysphagia.

More important in a diagnostic respect than all the pressure symptoms named is the effect of an aneurysm upon the organs of circulation. In the heart a hypertrophy of the left ventricle is not at all constantly demonstrable; more frequently a dislocation of the heart in such a manner that it appears displaced to the left inferiorly, and the apex beat is noted in the sixth intercostal space outside of the mamillary line.

Changes of the Pulse.—Of particular significance in the diagnosis are certain *changes in the force and rhythm of the pulse* in the peripheral arteries: Delay or marked variance in the force of the pulse in the arteries of the upper or lower extremities, according to the seat of the aneurysm in the ascending or descending aorta; furthermore, unequal condition of the pulse of the radial artery of one side in opposition to that of the pulse on the other side.

The *pulsus differens* of the radial (fainter pulse on one side) is caused in such a manner that the orifice of the arterial trunks which branch off from the aortic arch is unevenly narrowed in some cases of aneurysm. This may be due to slit-like distortion of the lumen of the vessels, or to coagulation in the aneurysmal sac, or to more pronounced atheromatous changes in one of the two subclavian arteries (especially the left). At any rate, the latter cause is the most frequent, and it follows that sclerosis of the one subclavian artery may bring about an exquisite pulsus differens without the presence of an aneurysm, as has recently been clearly demonstrated by von Ziemssen's investigations. At the same time, the pulse of the less-filled radial artery may, owing to its simultaneous atheroma, show the pronounced characteristics of a pulsus tardus and create the impression of an after-beat, as the top of the curve is thus reached later on the respective side. To avoid errors it should, of course, not be forgotten that in comparatively many individuals one radial artery is normally slightly narrower than the other and, therefore, the pulse on one side is more difficult to feel and appears weaker than that of the other side.

From what has been said, it appears that the pulsus differens is of only very limited value in the diagnosis of aneurysm. But its presence is of material support, nevertheless, provided other symptoms depending upon aneurysm are combined with it, such as intercostal neuralgia, paralysis of the recurrent laryngeal nerve, and bronchiostenosis. The unilateral pulse of the retinal artery, which is demonstrated ophthalmoscopically, may also be employed diagnostically; habitual hæmoptyses are also to be considered as precursors of a fatal perforation of the aneurysm into the lungs.

Compression of Veins.—If one or the other vein of the thorax becomes compressed by the aneurysm, stasis occurs with œdema in the region of the veins entering the same, on compression of the left innominate, in the face, neck, and upper portion of the chest on the left side; on compression of the superior vena cava, on both sides. *If an aortic aneurysm perforates into the last-named vein,* venous engorgement symptoms occur, the same as in compression of the vena cava by the aneurysm, *and that especially*

in the upper half of the body (congestion of the frontal veins, distention of the bulbi, swelling of the neck and tongue, difficulty of speech and dysphagia, and œdema of the upper extremities). A systolic venous pulse above the location of the communication may also appear on perforation. The engorgement develops suddenly to an enormous extent (owing to the difficulty of emptying the vena cava into the right auricle) and is less equalized by the development of venous collateral channels than is the case in simple compression of the vena cava by the aneurysm.

From the above it is obvious that the symptoms of aneurysms cannot always be alike, but must vary very much according to the seat of the aneurysm; the diagnosis, therefore, must not restrict itself to the presence of an aneurysm in general, but *must determine in each individual case which part of the large arteries is the subject of aneurysm.* In this respect the following points are of importance:

Aneurysm of the Ascending Aorta.—In favour of an aneurysm of the *ascending aorta* are: Dulness extending to the right of the manubrium sterni, from the second intercostal space upward, pulsation in the first and second intercostal spaces, displacement of the heart with its apex beat to the left inferiorly, retardation of the pulse of the peripheral arteries in contrast to the heart beat, pressure symptoms in the region of the superior vena cava, and pressure upon the pulmonary artery with dilatation and hypertrophy of the right ventricle, dyspnœa and, eventually, pulmonary phthisis.

Aneurysm of the Arch of the Aorta.—In favour of an *aneurysm of the arch of the aorta* are: Pulsation in the jugular fossa, dulness over the manubrium sterni and to the left of the sternum in the first intercostal space, displacement and distortion of the left innominate, carotid, and subclavian arteries branching off the arch, and, accordingly, inequality in the size of the pulse of the arteries of the head and arm of one half of the body, pressure upon the left innominate vein, engorgement and œdema in the region of the left sides of the head and neck, paralysis of the left vocal cord, compression of the left bronchus with symptoms of a bronchiostenosis.

Aneurysm of the Descending Aorta.—An aneurysm of the *descending thoracic aorta* becomes likely when pulsation occurs to the left of the spinal column in the height of the scapular angle, if a remarkable feebleness of the crural pulse sets in, in contrast to the radial pulse; furthermore, if symptoms of compression of the azygous, respectively hemiazygous veins and of the spinal column with gradual erosion of the latter, and paraplegia, and, finally, stenosis of the œsophagus and of the left bronchus occur.

Aneurysm of the Abdominal Aorta.—An *aneurysm of the abdominal aorta*, finally, is to be considered when a pulsating tumour develops to the left over the umbilicus near the spinal column. Sometimes a tremor can be observed in this region, and, instead of the systolic sound, a systolic murmur, rarely a double sound, is heard; furthermore, the smallness of the crural pulse in comparison to the force of the apex beat and the radial pulse should be considered; the crural pulse may also be retarded, but these theoretically supposed pulse changes are by no means constantly observed. Besides, sacral pains, gastric symptoms (vomiting, cardialgia, etc.), intestinal symptoms (constipation, diarrhœa, etc.), and dysphagia may be present. Great care should be taken, however, not to confound an aneurysm of the descending aorta with the conduct of the non-dilated vessel if the latter is seen and felt pulsating considerably owing to great emaciation. In such cases the aorta is very apt to appear as if the seat of an aneurysm; but then the systolic murmur, the changes of the crural pulse, etc., are absent.

[Finally, the value of the X-rays as a means of diagnosis must be mentioned. This should be resorted to in all doubtful cases.]

DIAGNOSIS OF THE DISEASES OF THE RESPIRATORY ORGANS

DISEASES OF THE LARYNX

The diagnosis of diseases of the larynx has been radically improved since the introduction of laryngoscopy. Symptoms of an affection of the larynx, such as hoarseness, aphonia, dyspnœa, etc., are no longer tenable as a diagnosis; they are rather to be looked upon as symptoms which call for a laryngoscopic examination. Diagnosis can only and exclusively result from an examination with the laryngoscope. We shall therefore, in discussing the diagnosis of the various affections of the larynx, only consider the respective laryngoscopical findings.

ACUTE LARYNGITIS—ACUTE LARYNGEAL CATARRH

The laryngeal picture in the laryngoscopic examination shows *hyperæmia* and *swelling* in various parts of the larynx which may affect either the entire mucous membrane of the larynx or only certain parts of the same, the epiglottis, the vocal cords, etc. The intensity of the hyperæmia and swelling varies greatly in the individual case—from a slight injection to a dark-red bulge-like swelling; sometimes only the motility of the vocal cords is slightly interfered with, sometimes the glottis is so narrowed that stenotic changes may occur. The mucous membrane may then be deprived of its epithelial covering and become rough, show excoriations or greater losses of substance, especially on the vocal cords and the vocal process; hæmorrhages into the mucous membrane are rarely found with acute hyperæmia and swelling. The secretions, at the onset are sparse, viscous, and hyaline, gradually turning opaque, grayish-yellow, but never very copious. The symptoms depending upon these changes—cough, hoarseness, aphonia, tickling in the throat, etc.—complement the clinical picture.

SPECIAL FORMS OF ACUTE LARYNGITIS

Epiglottitis, etc.—Accordingly, as the epiglottis, the ventricular bands, etc., are primarily affected, or are alone implicated by the acute catarrhal process, an "epiglottitis," chorditis superior, etc., may be spoken of. By the way, an absolutely unnecessary multiplication of the clinical nomenclature! It is sufficient to add to the diagnosis "inflammation of the larynx," "with special affection of the epiglottis," etc. It may be mentioned that pareses of the vocal cords (probably the consequence of inflam-

matory infiltration of individual laryngeal muscles) occur as a very frequent complication in acute catarrh of the larynx.

Catarrh of the Larynx in Small Children.—If the *catarrh of the larynx* attacks *small children,* the picture becomes slightly modified owing to the narrowness of the glottis. Here a relatively slight swelling of the vocal cords suffices to render respiration difficult. This impediment to respiration is increased to dyspnœa if, during sleep, secretions are deposited upon the glottis, or, more so, if the inflammatory swelling affects the submucous tissue of the mucous membrane and causes it to project bulge-like. If, under such circumstances—the cause is not known in *all* cases—a transitory occlusion of the glottis occurs, there arise the well-known, notorious attacks of "*pseudo-croup,*" with barking cough and the whistling sound of stenosis, audible at some distance, which symptoms relax after a duration of one or two hours or less. Often the laryngoscopical examination is impossible, owing to insurmountable difficulties.

Laryngitis Acuta Sicca.—From the common form of laryngitis, generally *laryngitis acuta "sicca"* is differentiated as a separate form, characterized by the peculiarity of the catarrhal secretion, that it may dry up easily or adhere closely to the mucous membrane, and that it generally forms crusts which are blood-tinged, which, on laryngoscopical examination, are easily recognised.

Laryngitis Acuta Submucosa.—More important than the differentiation of laryngitis acuta sicca from the common form of laryngitis is that of *laryngitis acuta submucosa,* because this latter modification of acute laryngitis represents a grave affection of the larynx which may place the patient in serious danger. In this form of laryngitis the inflammation extends to the *deeper* submucous tissue of the laryngeal mucous membrane. Some places of the larynx are especially the favourite points of attack: The *epiglottis,* the *aryepiglottic ligament,* the *false vocal cords,* and the *subcordal areas.* The fact that the swelling of the tissues is a considerable one in these areas causes dangerous symptoms of stenosis, especially when the inflammatory swelling has its seat under the vocal cords. The patient then presents the picture of suffocation, shows during inspiration pronounced inspiratory and expiratory stenotic râles and cyanosis; upon laryngoscopical examination we find under the normal or flushed vocal cords two thick red bunches which appear like duplicates of the vocal cords and which cause intense dyspnœa because they do not separate upon inspiration. Abscess formation with swelling of a circumscribed area of the interior larynx and perforation of the pus internally occur but rarely.

Differential Diagnosis.—Mistaking this affection for œdema of the glottis can be best avoided by observation of the flushing and the coarse swelling, while in non-inflammatory œdema the bulge-like tumefaction is more springy, jelly-like, and of a paler colour. Submucous laryngitis can be differentiated with difficulty only or not at all from perichondritis, unless the latter has developed into an uncovering of the cartilages. The distinct picture of inferior vocal chorditis does not give rise to mistakes; submucous laryngitis, on the other hand, which affects a ventricular band or the plica interarytænoidea, may present a laryngoscopical picture quite similar to perichondritis, especially, as in both immobility of the vocal cords supervenes and perichondritis may also be a complication of the submucous laryngitis. If the pus is

evacuated, the differential diagnosis becomes certain, because in perichondritis the uncovered cartilage is then recognized as such by the sound, or it is expectorated.

CHRONIC LARYNGITIS

Chronic laryngitis frequently originates from the acute form or develops chronically from the beginning; it is, as a rule, accompanied with chronic pharyngitis, or it may be due to syphilis or tuberculosis. It is characterized in the laryngoscopical image by moderate to intense blue-red, in part varicose, injection of the interior larynx, especially of the epiglottis, of the ventricular bands, the vocal cords, the plica interarytænoidea, which forces itself between the vocal cords and prevents their movement. Besides congestion there principally exists a thickening of the inflamed areas in the vocal cords in the form of nodular eminences (*chorditis tuberosa*). Erosions are not infrequently found; especially worth mentioning are the slit-like erosions (rhagades) in the interarytænoid fold. The motility of the vocal cords is impeded by the thickening and by secondary muscle paresis. The sparse, rather thick secretion is deposited in the shape of threads or globules upon the inflamed mucous membrane, it may also dry and form into crusts (*laryngitis chronica sicca*) and represent the continuation of a pharyngitis sicca upon the larynx.

Atrophy and Hypertrophy of the Mucous Membrane.—*Atrophy of the mucosa* with pale gray discoloration of the vocal cords and crust formation develops in the larynx also as a consecutive condition of chronic laryngitis, the same as in other catarrhal processes of the mucous membrane. On the other hand, the outcome of the chronic inflammation is occasionally a *glandular hypertrophy*, which gives the mucous membrane of the larynx a granulated appearance; eventually there occur upon the upper surface of the vocal cord papillary proliferations which may develop into abscesses.

Chronic Submucous Laryngitis.—There also exists a *chronic submucous laryngitis*, originating either as a result of the acute form or due to the chronic superficial catarrh. The affection is located principally in the epiglottis, the posterior wall of the larynx, the ventricular bands and the vocal cords and, above all, in the subcordal portions of the organ. The affection is characterized laryngoscopically by pale red, coarse tumefaction of the respective parts; in chronic hypoglottic (hypertrophic) laryngitis there appear two thick, rigid, mostly smooth, bright-red masses which represent something like inferior duplicates of the vocal cords and which may cause symptoms of stenosis, especially if the vocal cords are infiltrated at the same time and if a viscid mucus enters the previously narrowed glottis.

DIPHTHERITIC LARYNGITIS; CROUP OF THE LARYNX

Croup is characterized anatomically by a fibrinous exudation upon the free surface of the mucous membrane and into the tissue. The cause of croup is the diphtheritic virus, in relatively rare cases the anatomically uniform process is due to scarlatinal infection, still more rarely to violent chemical and thermic effects upon the larynx. Only that form of croup

which arises as a result of diphtheritic infection is of clinical significance. The diagnosis of the affection does not present any material difficulties, although no exact laryngoscopical examination can be made in this disease. A diphtheritic inflammation of the pharynx ushers in the laryngeal affection in some of the cases, in others, which are more rare, however, the larynx is the organ which is primarily affected. The latter occurs in children, but sometimes also in adults, as I recently observed in a case which terminated fatally. The autopsy showed that in this instance the larynx was actually the exclusive seat of the affection.

The most important symptom is *stenosis of the larynx*, which is characterized especially by obstructed, snarling inspiration and expiration, and which calls into activity all the auxiliary respiratory muscles, by drawing in the epigastrium, the intercostal spaces, and the throat, and by inspiratory lowering of the larynx. The affection is accompanied by a barking, and finally soundless cough. The attacks of suffocation increase gradually; restlessness, cyanosis, and insomnia owing to carbonic-acid poisoning take place, the pulse becomes small, frequent, and intermittent, until death occurs owing to exhaustion and suffocation. If a laryngoscopical examination is possible, which, however, is not frequently the case, as stated above, we find the entire interior of the larynx, or some portions of the same, covered with grayish-white, sometimes delicate, at other times coarse membranes. Besides, a thick viscid mucus may be present in the interior of the larynx and contribute to the occlusion of the glottis. The motility of the vocal cords is impeded partly by this occlusion, partly by the membranes, and partly, finally, by the paresis of the musles which are saturated with serum. The *fever* is moderate, generally higher, if the process, spreading downward, causes a catarrhal pneumonic infiltration.

Differential Diagnosis.—As stated, the diagnosis of laryngeal croup does not present any difficulties, as a rule; the only possibility is, at least temporarily, to mistake it for *pseudo-croup*. The latter sets in suddenly, after the child has been quite well until then, while in croup generally cough, hoarseness, fever, or angina with diphtheritic deposits precede the characteristic croupy cough and the laryngeal stenosis. The dyspnœic attacks last only a short while, at most a few hours in pseudo-croup; in croup, on the other hand, the stenosis of the larynx with its dangerous symptoms is more constant, although not quite uniform, inasmuch as attacks of severe dyspnœa alternate with periods of freer breathing; however, the respiration is always impeded, even during the periods that are free from attacks. The diagnosis becomes certain by means of the laryngoscopical examination, which, however, is not always successful; furthermore, by the demonstration of diphtheritic membranes on the tonsils and by the fact that shreds of fibrin are expectorated in which diphtheria bacilli can be demonstrated. (For particulars, see Diphtheria, Infectious Diseases.) Confounding the affection with other diseases which lead to stenosis of the larynx, like œdema of the glottis, retropharyngeal abscess, etc., is impossible on careful examination.

LARYNGEAL ŒDEMA; ŒDEMA OF THE GLOTTIS

The œdema, according to its seat and extension, is either latent or is accompanied by the gravest symptoms of suffocation from the onset. The laryngoscopical examination shows intense swellings of the laryngeal mucous membrane—that is, of the submucous tissue—most frequently in the epiglottis, which represents a globe-like tumour; furthermore, in the aryepiglottic folds in the shape of two large pale-red, elastic, or soft masses which meet in the centre; the vocal cords are very rarely œdematous, more frequently the ventricular bands; all these changes are calculated to impede inspiration—in the severer grades also expiration—to the utmost. The diagnosis is easy, but the discovery of the cause often presents difficulties.

Ætiological Diagnosis.—The majority of laryngeal affections lead occasionally to œdema, most frequently *submucous laryngitis*, in which the swelling represents a particularly coarse, dark-red tumour; œdema is also found in perichondritis and in the various varieties of abscesses of the larynx. Œdema of the larynx, representing the peripheral waves of the inflammation, furthermore combines with phlegmon of the throat and with retropharyngeal abscess; more rarely with glossitis, parotitis, etc. In other cases the affection is the consequence of engorgements in cardiac diseases, mediastinal tumours, goître, etc., or it may be due to Bright's disease, amyloid degeneration, carcinoma, and other cachectic diseases. Œdema of the glottis may also develop in the course of the infectious diseases, enteric fever, scarlatina, etc.; above all, in the course of erysipelas as a metastatic inflammatory affection. Likewise a *primary laryngeal erysipelas*—i. e., a primary invasion of the streptococcus erysipelatis in the larynx—occurs. It must be admitted, finally, that in rare cases, if a cold or similar doubtful ætiological factors are not taken into consideration, no direct cause for the origin of the œdema of the glottis can be demonstrated (cryptogenetic œdema of the glottis).

At any rate, the diagnosis of a cryptogenetic œdema of the glottis should be accepted only when it is absolutely impossible, in spite of the most careful search, to discover a local or general cause of the œdema. If the latter develops slowly, or if an acute œdema becomes protracted for some length of time, we may speak of a *chronic œdema of the glottis.*

PERICHONDRITIS

Laryngeal perichondritis, in the rarest instances a primary affection, is usually due to trauma and compression of the larynx or to deep-seated inflammatory and ulcerating processes in the larynx or its neighbourhood. In its course it leads to accumulation of pus between cartilage and perichondrium, to abscess formation near the respective cartilage, and, because the latter obtains its nutrition from the perichondrium, to necrosis of a more or less large portion of the cartilage. If the abscess bursts, the necrotic cartilage is accordingly uncovered and cast off. The perforation of the abscess occurs internally into the larynx, the œsophagus, or to the surface of the skin with formation of a fistula (laryngeal fistula). The clinical picture of perichondritis develops according to these anatomical changes. There arise: Tumefactions in the interior of the larynx, stenosis of the larynx, inspiratory dyspnœa, localized laryngeal pains, disturbances

in phonation, furthermore, dysphagia, œdema, and formation of fistula in the skin of the throat.

The seat of the affection is most frequently in the arytænoid cartilage, most rarely in the epiglottic cartilage. Arytænoid perichondritis is generally due to tuberculous ulcerations at the posterior portion of the vocal cords, more rarely to typhoid ulcers, and is accompanied by immobility of the respective vocal cord; while perichondritis of the plate of the cricoid cartilage leads to paralytic conditions of the posterior crico-arytænoid muscles and its consequences (see p. 87).

Perichondritis manifests itself *laryngoscopically* in the first place by circumscribed swellings of certain portions of the interior of the larynx and by symptoms of abscess formation. Later, after perforation of the pus, fistulæ remain through which the necrotic cartilage may be felt by means of the sound, or eventually eliminated, perhaps expectorated. The diagnosis of perichondritis becomes positive only upon this demonstration of necrosis of the cartilage; the inflammatory and ulcerative symptoms which precede the denudation of the cartilage are also peculiar to other processes, especially to submucous laryngitis.

Laryngeal Herpes.—In connection with the inflammations of the larynx it may be mentioned that herpetic eruptions are also, though rarely, observed in this organ—white-yellowish vesicles, in appearance like herpetic vesicles, which burst and form superficial abscesses. Their herpetic character becomes marked in particular by their simultaneous presence on other mucous membranes or upon the external skin.

ULCERS OF THE LARYNX

The diagnosis of these *ulcers* is generally easy by means of the laryngoscopical examination as soon as they are of a certain size; small ulcers, however, give rise to mistakes in diagnosis. If, in particular, small inequalities are found upon the inflamed mucous membrane, especially at the free border of the vocal cords, it is possible to explain those areas in the mucous membrane which appear as depressions alongside of the prominences, as losses of tissue, especially when shreds of mucus are noticed here. Besides, in some cases, small ulcers are not visible if the adjacent tissue is very swollen, or if the borders arise tumour-like over the fundus of the ulcer, covering the latter more or less. This is observed principally in the cleft-like ulcers of the interarytænoid fold. If an ulcerating defect of the mucous membrane has been ascertained, the second, not less important object of diagnosis is to decide upon the character of the laryngeal ulcer found. We distinguish in this respect:

1. *Catarrhal* ulcers.
2. *Pressure* ulcers.
3. *Infectious* ulcers (tuberculous, enteric, septic, syphilitic).
4. Ulcers produced by the disintegration of *neoplasms* (see Neoplasms).

Catarrhal Erosive Ulcers.—1. *Catarrhal inflammatory* ulcers are rare, as a rule; they arise in the course of a non-specific superficial laryngitis as "*erosion ulcers*"—i. e., as a defect of the epithelium, especially of the plate epithelium of the vocal cords, at the anterior surface of the arytænoid

cartilage and the posterior surface of the epiglottis. They are distinguished by their superficiality, and present themselves as superficial, small, or, at the utmost, superficially more extended ulcers, generally at that place at which the mucous membrane is exposed to greater friction and pressure, consequently at the cartilage of the glottis. Another cause of inflammatory ulcers is, that suppuration and necrosis of the inflamed tissue occur at some places, owing to preceding, deep-seated inflammations (submucous laryngitis, perichondritis), and an ulcer forms by perforation of the overlapping mucous membrane. The diagnosis in these cases is based, above all, upon the observation of *deep*-seated inflammation which precedes and accompanies its formation; the edges of the ulcers in the latter case are bulged and undermined.

Pressure Ulcers.—2. *Pressure Ulcers.* As stated above, pressure plays a certain ætiological part, favouring their origin, in the production of superficial catarrhal ulcers. Those ulcerations which develop owing to the deposition of a foreign body in the larynx, arise beyond doubt in consequence of the effect of pressure. Those ulcers also which form in the posterior commissure in debilitated patients, especially those suffering from enteric fever, should probably be explained in part (see below) as the effect of pressure in the acceptation of decubital ulcerations at other parts of the body.

Infectious Ulcers.—3. By far the greatest number of laryngeal ulcers are of an infectious character. The lion's share in the formation of this variety of ulcers and of laryngeal ulcerations in general falls to *tuberculosis.*

Tuberculous Ulcers, Origin and Appearance.—This disease, it is true, causes other pathological conditions of the larynx also: Anæmia of the mucous membrane, simple catarrh of the larynx, and disturbances of innervation, especially pareses of the laryngeal muscles. This catarrh comes and goes in the course of pulmonary phthisis. However, all these pathological conditions are of subordinate significance compared with the pronounced *tuberculous ulcers* of the larynx which form the principal part of laryngeal consumption. They arise from subepithelial, cellular infiltrations which project hump-like from the surface of the mucous membrane; in some cases even, like tumours, become caseous, disintegrate and, in perforating the epithelial covering, form ulcers which mostly present a crater-like appearance and elevated margins. E. Fränkel's investigations have recently demonstrated that the tuberculous changes in the larynx are but rarely brought about by the introduction of the tubercle bacilli from the blood or lymph channels, but by their direct entrance from the surface—i. e., through the more or less intact epithelia; frequently other micro-organisms (staphylococci and streptococci) are found in the ulcerated region beside the tubercle bacilli. In the neighbourhood of the ulcers (in the submucosa, the perichondrium, more rarely in the muscles) genuine tubercles are found, which again may disintegrate into punctiform ulcers, thus contributing to an increase of the original ulceration.

The seat of the tuberculous ulcers changes; the infiltrations and consecutive formations of ulcer occur most frequently, in my experience and that of others, in the *interarytænoid fold*, often so early that it is not possible at that time to demonstrate changes in the lungs. The ulcers in this locality generally show very tumid margins, covered with papillomatous excrescences, which may entirely cover the fundus of the ulceration, espe-

cially when the latter are formed cleft-like. Infiltrations and superficial ulcers occur next in frequency in the *ventricular bands*, which may be so infiltrated and distended that nothing can be seen of the vocal cords. Particularly characteristic is, furthermore, the infiltration of the region of the arytænoid cartilages, which swell globe-like and the locomotion of which is impeded. The *vocal cords* proper are, in my experience, just as often affected by the ulceration. They appear at first uneven and thick, more infiltrated above and below, so that they seem arched lengthwise in the centre. Here, in contradistinction to infiltrations of the interarytænoid fold, the ulceration takes place very soon, and preferably occupies the region of the vocal process, evidently because here the most friction occurs; the vocal cord is gradually affected in its entire length and destroyed.

More rare is the infiltration and ulceration of the aryepiglottic ligaments and of the epiglottis (in contradistinction to syphilitic ulcers which are located preferably in the epiglottis). The ligaments become transformed into coarse bundles, the epiglottis into a formless mass, which is distended tumid-like towards the margin and slightly sunk in the centre; the infiltration ulcerates at various points, either superficially or in the form of deep-seated ulcers with undermined margins. So far, it has scarcely ever been possible to recognise miliary tubercles as such laryngoscopically—i. e., by controlling the finding *intra vitam* by the finding at the autopsy—although I do not wish to doubt the possibility of demonstrating gray-yellowish tubercular nodules by means of laryngoscopical examination.

Diagnosis of Tuberculous Ulcers.—Although the tuberculous character of the ulcers is, of course, probable in the individual case, according to the above-mentioned indications, yet an ulcer can never be recognised with certainty as tuberculous from its external appearance. The diagnosis does not become *probable* until an undoubtedly tuberculous affection has been demonstrated in another part of the body, especially in the lungs; but the diagnosis is not certain even then, because in some, although extremely rare cases, absolutely non-specific ulcers have been also found in consumptives. But, on the other hand, there exist also undoubted primary tuberculous affections of the larynx. In those exceptional cases in which it is positively impossible to demonstrate any changes in the lungs, it should only be assumed that it is nevertheless a question of a tuberculous ulcer *when tubercle bacilli can be demonstrated in the sputum emanating from the larynx.* The experimental injection of Koch's tuberculin to determine the tubercular character of a laryngeal ulcer is, at present at any rate, done only in the rarest instances, owing to the powerful specific reaction, which usually follows, with rapid, considerable increase in the size of the ulcer.

Syphilis becomes localized in the larynx relatively frequently and gives rise to the most various affections, which may be considered as syphilitic, either by their well-characterized laryngoscopical conduct or because they are, according to experience, frequent in the course of lues and promptly disappear upon antisyphilitic treatment, while they are very refractory to any other therapy.

Syphilitic Laryngitis and Condylomata.—This refers principally to *syphilitic laryngitis.* Laryngoscopically it does not at all differ from non-specific catarrh of the larynx and its various forms and consecutive conditions, erosions, etc. Much rarer

(as an expression of syphilis) are *broad condylomata* of the larynx which are located relatively most frequently in the vocal cords, the epiglottis, the interarytænoid fold, etc., and which occur in the usual form of mucous papules of the pharynx, eventually also as small elevations with whitish epithelial cover.

Gummatous Ulcerations.—Syphilis becomes localized in the larynx generally in its later course only, and then it forms deep-reaching numerous infiltrations and small nodes (*gummata*). These present a pronounced tendency to decay and ulceration. The ulcerations thus formed are characterized by distended margins, distinct demarcation, deep fundus, also by rapid dissemination, so that considerable destruction in the larynx occurs, on the one hand, and, on the other, in case of cure, extensive distortions of the laryngeal organs and formation of stenoses take place owing to cicatrization. As to the *seat* of the ulcers, generally the *epiglottis* is affected by the destruction, and from here ulceration and destruction generally extend along the aryepiglottic ligament to the ventricular and vocal cords. In their further course perichondritis and necrosis of the cartilages are very apt to occur. Although a very specific appearance of the ulcerations does not exist, in my opinion, yet, from the mode of progress of the ulceration from the epiglottis, the diagnosis of syphilis of the larynx can be made laryngoscopically with a certain degree of probability, especially if the ulceration is located upon the upper surface of the epiglottis. This assumption is supported by the presence of gummatous ulcers of the pharynx, of the skin, of syphilitic affections of the bones, etc., and of cicatrices of syphilitic affections which have healed in consequence of specific treatment. Syphilitic ulcerations of the larynx can in this manner, as a rule, easily be distinguished from tuberculous ulcers, although in the latter, too, especially when the epiglottis is destroyed, the external picture of the destruction is similar to that of laryngeal syphilis. The syphilitic ulcers differ from the carcinomatous ones principally in so far as in the former destruction predominates, in the latter, nodular proliferations.

Typhoid Affections of the Larynx.—Laryngites of a milder and severer type—i. e., simple catarrhs with submucous or croupous inflammations—occur in the course of the most various infectious diseases, such as measles, scarlatina, erysipelas, variola; this is also the case in enteric fever. The laryngoscopical finding in the latter, however, is of greater significance in so far as the laryngeal changes which arise during the course of the typhoid process assume an ulcerative character more often than in the other infectious diseases of the larynx, so that the frequency of laryngeal ulcers in individuals who have died of enteric fever has been estimated to be not less than 20 per cent; however, in the epidemics of enteric fever in central and southern Germany which I observed, the occurrence of laryngeal ulcers in enteric fever was always a rather rare occurrence. The ulcers arise from an infectious, very cellular infiltration, which is sometimes rather circumscribed, more rarely diffused, and which affects the lymphatic system, especially the tunica propria, with its inclosed leucocytes. The *seat* of the infiltrations and of the ulcerations originating from them is principally the epiglottis, the ventricular bands, the inner surface of the arytænoid region, the posterior and anterior commissures. The ulcers present, according to their formation, tumid, undermined margins, and a tendency to deep extension and lead to necrosis of the cartilage. Aside from typically enteric ulcers there are also found superficial erosions or, at times, secondarily diphtheritic ulcers which do not possess the tumid margins of ulcers caused by infiltration. The enteric ulcers, in contradistinction to those of a syphilitic character, mostly heal without leaving cicatrices;

upon *deep* destruction, however, the healing may lead to the formation of stenoses. The diagnosis of typhoid ulcers is based primarily, aside from the laryngoscopical finding, upon the presence of indubitable symptoms of enteric fever—enlargement of the spleen, fever, eruption, etc. Occasionally the presence of typhoid bacilli in the secretion of the ulcer has been actually demonstrated.

The diagnosis of laryngeal changes in the infectious diseases is mostly founded essentially upon the general pathological picture of the infectious disease in question. For, although these changes in the larynx are of a specific character, at least in some forms, and their appearance from the beginning points sometimes to the presence of some variety of infectious disease, the *positive* diagnosis is never possible, unless, besides the alterations in the larynx, the characteristic symptoms of the respective infectious disease can be demonstrated. This is the case in the previously named, infectious ulcers of the larynx and also with laryngeal ulcers in the course of leprosy with its profuse node formation, the ulcers of glanders, etc.

CICATRIZATION IN THE LARYNX—LARYNGEAL STENOSIS

Small superficial cicatrices may develop in the larynx without producing symptoms, especially when the glottis remains unaffected. Extended, deep-reaching cicatrizations, however, cause, according to their location, the gravest disturbances in phonation and respiration: hoarseness, aphonia, dyspnœa, in consequence of stenosis of the larynx. In rare cases cicatrizations are the cause of formation of actual membranes, of membranous coalescences of the vocal cords, etc. The consequence is again laryngeal stenosis to a greater or lesser extent.

Moderate stenosis of the larynx, caused either by cicatricial narrowing of the lumen, by neoplasms, croupous membranes, or œdema of the glottis, etc., manifests itself by a slight impediment in respiration which assumes a dangerous aspect only when greater demands are made upon the respiration (when speaking, walking, etc.). In the severer degrees of the stenosis, on the other hand, the air-hunger of the patient is pronounced even during rest. The well-known picture of dyspnœa sets in: retardation of respiration with greatest exertion of the auxiliary respiratory muscles, far-sounding, whistling, rasping sounds, which are produced when the respired air passes the stenosed point. The picture of the dyspnœa varies according to the obstruction to inspiration or expiration or to both simultaneously. If a polyp, a croupous membrane, etc., is located below the glottis, so that a valve is formed which leans towards the glottis upon expiration, the picture of *expiratory dyspnœa* presents itself—i. e., long-drawn, noisy, difficult expiration following easy and quick inspiration. To increase the energy of expiration the abdominal muscles are contracted during expiration, the spinal column is bent forward. Quite different is the condition in *inspiratory dyspnœa*, which prevails much more frequently in laryngeal stenosis: in œdema of the aryepiglottic folds, of the epiglottis, in croup, in paralysis of the posticus, etc. In these cases expiration takes place without difficulty—it is rapid and noiseless, inspiration, however, with greatest exertion, much prolonged. The sterno-cleidomastoidea, the

scaleni, the levatores alæ nasi, etc., become active, the epigastrium relaxes during inspiration, the larynx, in contradistinction to its action in tracheal stenosis, makes violent respiratory excursus. In mixed, i. e., *inspiratory and expiratory,* dyspnœa, both acts of respiration are impeded and forced.

The patients are able, owing to the retarded, but deeper respirations, fully to satisfy the demands for oxygen, so that these patients are in position to respire, although with difficulty, but successfully enough regarding the inhalation of oxygen, until this regulatory protraction and deepening of the respirations relaxes or becomes insufficient on account of the size of the obstacle, and suffocation develops slowly but surely. The central nervous system gradually loses its tonicity owing to the constant deficiency of oxygen, and the compensatory muscular exertions relax, and thus the picture of gradual suffocation becomes manifest. The mucous membranes discolour to a blue gray, the skin becomes cool, the respirations superficial and frequent, the sensorium becomes clouded, somnolence becomes more and more pronounced until death occurs in this condition.

The picture of acute suffocation is entirely different, as we have an opportunity to observe, although very rarely, in some stenoses of the larynx which develop very rapidly; thus in peracute œdema of the glottis, and especially in spasm of the glottis. Here, symptoms of irritation, above all, become prominent. Besides anxiously gasping respiration and high-graded cyanosis, dilatation of the pupils occurs and general convulsions.

Differential Diagnosis.—It is easy, from the above, to make a diagnosis of laryngeal stenosis, particularly when it is possible to perform laryngoscopical examination, and thus determine the cause of the stenosis. If the examination of the larynx meets with insurmountable difficulties, the differential diagnosis may sometimes waver between laryngeal and tracheal stenosis. Generally the manner alone of the stridor during respiration does not admit of any doubt as to the locality of the stenosis of the respiratory canal; a great downward locomotion of the larynx with every inspiration is still more in favour of a laryngeal stenosis. Aphonia and the straight or backward extension of the spinal column are also considered characteristic of laryngeal, in contradistinction to tracheal, stenosis. Both these facts are, of course, not evidence of the former, because aphonia is sometimes found in tracheal stenosis also (caused by paralysis of the recurrent laryngeal nerve, which again is usually the coeffect of the cause of compression of a tumour producing tracheal stenosis), and the dyspnœic backward extension of the spinal column may be associated with every intense form of dyspnœa.

NEW GROWTHS IN THE LARYNX

The neoplasms occurring in the larynx are, according to their character, either benign—papillomata, fibromata, lipomata, cysts, myxomata, lymphomata, enchondromata, and angeiomata—or they are malignant—carcinomata, sarcomata.

Of the *benign tumours* only the two first named are of importance in a clinico-diagnostic respect, while the others may occur, but are found

rarely and are more of a pathologico-anatomical curiosity than of clinical interest. At best, only cysts and enchondromata are of some practical significance, besides papillomata and fibromata. The symptoms caused by tumours of the larynx, the changed voice, the cough, the dyspnœa, etc., being too ambiguous, are of no importance in diagnosis; the latter should be made *only* from the laryngoscopical findings.

Papilloma.—"Papilloma" (pachydermia verrucosa) is the most frequent neoplasm in the larynx, and presents itself in the form of tumours of verrucose construction, varying in size from that of a millet-seed to that of a walnut; sometimes they form a group of small plugs, at others mulberry or cauliflower-like protuberances. They generally develop upon a broad base, rarely they are pedunculated; they grow quite rapidly, and are very apt to recur. Their colour changes from light gray to dark red; they are located almost without exception on the vocal cords, especially in their anterior two thirds, rarely at other places of the larynx.

Pachydermia diffusa is a more *diffuse* form of proliferation of the basement epithelium in the larynx, in which the change takes place preferably in the superior connective tissue, in the mucous membrane. It belongs in the same class with pachydermia verrucosa in every respect, and develops, the same as the latter, upon the base of a chronic inflammation; its region of localization is more the *posterior* portions of the vocal cord and the immediate neighbourhood of the vocal process of the arytænoid cartilage. In this locality, especially, *oval, puffy tumefactions* are found, with *an oblong, superficial depression in the centre.* The latter occurs, as Virchow convincingly taught, in that, at the place mentioned, the mucous membrane closely adheres to the subjacent cartilage and therefore rises less than the surrounding parts. The formation of this depression is evidently still more furthered by the mechanical pressure which is exercised in this locality by the bounding of both vocal cords, respectively vocal process, upon one another (B. Fränkel).

Fibroma.—The fibromata of the larynx form globular or pear-shaped, sometimes nodular, generally pedunculated tumours (*polypi*) with *smooth* surface, of a whitish-gray to dark-red colour; they are from a lentil to a hazel-nut in size, rarely larger; they grow more slowly and are not apt to recur, in contradistinction to papillomata. They, too, occur most frequently in the vocal cords, originate sometimes at the superior, at other times at the inferior surface of the same.

The *cysts* (mucous polypi) are much rarer, small tumours of the larynx, attaining sometimes the size of a cherry. They are scarcely ever pedunculated, are generally situated upon a broad base as globular protuberances, and usually arise from Morgagni's ventricles or the epiglottis. Their surface is smooth, elastic, sometimes fluctuating. If cut they discharge a viscid fluid.

Enchondromata, originating in the cricoid or thyreoid cartilage, grow from here as flat, nodular tumours, covered by the mucosa, into the interior of the larynx, and may have the appearance of a polyp, but are easily distinguished from the latter, as well as from other tumours of the larynx, by their hardness.

Carcinomata.—*The malignant neoplasms of the larynx* are much rarer than the benign papillomata and fibromata. Of the two malignant tumours occurring in the larynx, *carcinoma* and *sarcoma*, the former is by far the more frequent new growth. Of the various forms of carcinoma, medullary carcinoma and scirrhus are rare, the usual form being *epitheli-*

oma. While the latter usually represents verrucose, coarsely uneven, or cauliflower-like formations, the medullary carcinoma forms rapidly growing vascular, ulcerating nodes. The seat of a carcinoma is principally the vocal cord and the ventricle of Morgagni, particularly often also the ventricular ligament and the epiglottis.

Differential Diagnosis.—The diagnosis is not difficult if it is a question of a (actually extremely rare) secondary carcinomatous formation, or also, if the primary carcinoma of the larynx is fully developed; if diffuse intumescences and ulcerations of the larynx present themselves besides tumefactions of the lymph glands in the external region of the throat; if the tumour proliferates into the pharynx; if the affected individual has passed the fortieth year and becomes cachectic in a relatively short time "without reason." But then, too, confounding these growths with syphilitic affections may still occur; as a rule, the ulcerations of the syphilitic infiltrations develop more rapidly, and cicatrized places may eventually be present, besides the ulcerations, whereas they are wanting in carcinoma, which forms ulcerations with an uneven base and bulging margins, and which is distinguished by the development of irregular verrucose nodes. The tuberculous ulcers are much easier of differentiation, especially as bacilli can almost without exception be demonstrated in the sputum. Much more difficult is the diagnosis of carcinoma at the beginning of the development of the tumour, so long as no ulceration has taken place; then it may present a picture simulating benign tumours, above all papilloma. Carcinomata form a more uniform infiltration of the tissue; they are more vascular, and are very apt to ulcerate; a microscopical examination of an excised *piece of tissue, not too small, and taken from the lower portions of the tumour,* is advisable under all circumstances, even if positive results are not always obtained thereby. It is important, according to Virchow, that at the bottom of the epithelial cover, in the connective tissue, no trace is present of epithelial structures; the contrary proves that the neoplasm in question is of a carcinomatous nature. It is not very well possible to confound laryngeal carcinoma with leprosy, in the first place, because the latter never occurs primarily.

Sarcomata, extremely rare, malign neoplasms of the larynx, do not present anything characteristic in appearance; they may be flaccid, verrucose, lobulated, showing a whitish or red colour. The diagnosis, at any rate, is not possible by a simple laryngoscopical examination but only after a microscopical investigation of excised particles of the tumours.

FOREIGN BODIES IN THE LARYNX

According to the size and shape of the foreign body and according to the place which it occupies in the larynx, either suffocation occurs at once or a severe laryngospasm. The attacks of suffocation may be frequently repeated, especially if the foreign body changes its position, owing to respiration or to attacks of cough, or transitorily occludes the glottis. If the foreign body remains in the larynx for some length of time—i. e., if it is not expectorated or passed downward into the bronchus—stenosis of the larynx with its characteristic dyspnœic symptoms occurs owing to a secondarily formed swelling and inflammation of the soft parts; smaller foreign bodies are retained for a remarkably long time without causing any material trouble. Usually the laryngoscopical examination dispels any doubt as to the presence and

locality of a foreign body in the larynx; but this examination is often very difficult owing to the high-graded dyspnœa.

NEUROSES OF THE LARYNX

Among these are comprised *all those affections of the larynx in which there is nothing, from a pathological-anatomical standpoint, that prevents us from referring them exclusively to the nervous system.*

The nerves of the larynx are of a sensory and motor nature—the *sensory* nerve, as is well known, is the *superior laryngeal nerve.* The latter has two branches, the exterior and the interior rami; the former imparts motor fibres to the crico-thyreoid muscle and sensory fibres into the lower lateral portion of the laryngeal mucous membrane. The remaining portions of the mucous membrane are supplied with sensory fibres by the purely sensory internal ramus of the superior laryngeal nerve which passes through the hyothyreoid membrane. Irritation of the sensory fibres causes the most varied sensations in the larynx, and reflexly cough. The motor fibres of the superior laryngeal nerve probably all originate (the same as those of the inferior) in the spinal accessory nerve, which sinks a great many of its fibres into the vagus trunk.

The *motor* nerve for all laryngeal muscles (except the crico-thyreoid muscle) is the recurrent laryngeal nerve. Branching off from the vagus within the thorax, it twines to the left around the aortic arch, to the right around the right subclavian artery antero-posteriorly, and ascends between trachea and œsophagus to spread over the laryngeal muscles. Irritation of the recurrent laryngeal nerve causes laryngospasm, and section of the same, the picture of recurrent laryngeal paralysis, which will be described later on.

SENSORY NEUROSES

Hyperæsthesia.—Hyperæsthesia and anæsthesia of the laryngeal mucous membrane are generally not very frequent affections. *Hyperæsthesia* occurs as a pure neurosis principally in hysteria and neurasthenia, and manifests itself by a feeling of titillation, as of a foreign body sticking fast in the larynx, etc.; no changes from the normal condition of the larynx can be detected by laryngoscopical investigation. The hyperirritability of the sensory channels in other instances leads to spasmodic "nervous" cough or laryngospasm. Such attacks of cough and suffocation are most marked in the course of tabes, known under the name of laryngeal crises ("crises laryngées"). In other cases again, the increased irritability of the sensory fibres manifests itself in the picture of an actual laryngeal neuralgia with paroxysms of pain and pronounced areas of pain in the throat. It is as yet inexplicable why one or the other form of the sensory irritability occurs in a given case. At all events, the negative laryngoscopical finding is characteristic; touching the laryngeal mucous membrane with the sound may artificially produce the above-named expressions of increased irritability.

Anæsthesia.—*Hyperæsthesia* and *anæsthesia* of the laryngeal mucous membrane, on the other hand, manifest themselves by insensibility of the membrane to the touch with the sound or laryngeal electrode in otherwise normal laryngoscopical conditions. The question is still undecided whether the glottis detractors (thyreo- and aryepiglottic muscles) are innervated by the superior laryngeal nerve, a branch of the internal laryngeal nerve; the effect of the irritation of the superior laryngeal nerve does not indicate it, but clinical observation (von Ziemssen) makes it probable that in paralysis of the superior laryngeal nerve immotility of the glottis occurs besides anæsthesia; the glottis appears, in such cases, in the laryngoscopical picture upward posteriorly, entirely immotile and leaning against the fundus of the tongue. The entrance of food into the larynx might be facilitated by the coincidence of paralysis of the epiglottis muscle; the food remains in the interior of the larynx or advances farther down into the bronchi, because the sensibility of the laryngeal mucous membrane is absent and the reflex tract is interrupted. It is obvious that, in paralysis of the superior laryngeal nerve, also that of the crico-thyreoid muscle is to be ex-

pected; we shall refer to this condition later on in discussing the paralyses of the laryngeal muscles. Anæsthetic conditions of the larynx are also found in hysteria; furthermore, in connection with diphtheria, and, finally, in consequence of central disturbances of innervation in cerebral foci diseases (in this case unilateral anæsthesia) and in bulbar paralysis.

Paræsthesia.—The *most important diagnostic* rule in *paræsthesias of the larynx*, which not infrequently occur in hysteria and neurasthenia and manifest themselves by a feeling of heat, cold, pressure, etc., in the larynx, is that the laryngoscopical examination does not show any abnormal condition or, according to whether hyperæsthesia or anæsthesia of the laryngeal mucous membrane is connected with it, yields only those few positive facts which we have just described as the state occurring in those conditions.

DISTURBANCES OF MOTILITY OF THE LARYNX

They manifest themselves either as spasm or as debility or complete paralysis of the muscles of the larynx.

SPASM OF THE MUSCLES OF THE LARYNX

The commonest sequence of spasmodic contraction of the laryngeal muscles is the picture which is known under the names of *spasmus glottidis,* laryngismus stridulus, laryngospasm, etc.

LARYNGOSPASM

Diagnosis of Laryngospasm.—If laryngospasm is not a secondary symptom of affections of the larynx, but a pure neurosis, *no anatomical changes are found in the larynx during the time in which no attacks occur*, upon laryngoscopic examination. Such an examination is almost never feasible during an attack; if, exceptionally, we succeed in making it, the glottis is found firmly closed in its entire length, the vocal processes slightly projecting. The diagnosis, therefore, is based less upon the objective examination than upon the clinical picture of spasmus glottidis, which is very marked indeed. It is characterized by an inability of the glottis to open during respiration which occurs in attacks of short duration. This closing of the glottis, which is complete in children, less so in adults, is ushered in by several noisy, hissing inspirations with short, likewise noisy, expirations, followed by a more or less complete suspension of respiration. This condition is combined with cyanosis, expression of the greatest anxiety, stretching of the neck with the head bent backward, restlessness, protrusion of the eyes with dilatation of the pupils, incontinence of urine and fæces, convulsions—in short, *the picture of acute suffocation.* After a duration of from a few seconds to two minutes, the dyspnœa—the fatal termination of the attack is very rare—passes into the normal condition of the respiration, to return sooner or later. In adults the attacks are of a much milder character, the convulsions, in particular, which depend upon the dyspnœa, are almost always absent; in hysterical individuals, however, the same as in children, there occur general muscular spasms which are not dependent upon the dyspnœa, and which either precede or follow the suspension of respiration. The mildest form of laryngospasm in adults manifests itself in a feeling of constriction of the larynx occurring in attacks, also in spasmodic inspiration and expiration without the appearance of actual dyspnœa.

Differential Diagnosis.—The affection can scarcely be mistaken for any other one, and it is absolutely unnecessary to name the factors which distinguish it from croup, œdema of the glottis, pertussis, laryngeal polypi, etc. The observation of absolute well-being [health] between the attacks and of the laryngoscopical findings during this time, also the absence of cough, etc., make the diagnosis certain. At most, the affection may, at the first glance, be confounded with attacks of pseudocroup or paralysis of the posticus [crico-arytænoid posticus]. The attacks in the former condition usually last much longer, a barking cough is present, and the inter-

vals are less free from pathological symptoms. In paralysis of the posticus, in contradistinction to laryngospasm, it is a question of a *permanent* condition of impairment of respiration, which may increase to dyspnœa upon the slightest cause; however, the laryngoscopical examination does not admit of the slightest doubt that a pathological condition prevails also during the time in which there is freedom from attacks.

Functional, Phonetic, and Respiratory Laryngospasm.—Recently laryngospasms have been observed which do not occur, similar to the usual form, spontaneously and in paroxysms, but only upon functional activity in a certain direction of the laryngeal muscles, and, consequently, a *functional spasmus glottidis* (*phonetic* and *respiratory*) has been differentiated. In the former, the *phonetic laryngospasm*, it is a question of a spasmodic closing of the glottis upon any attempt at phonation, so that speech is impossible or extremely impaired. The laryngoscopical examination does not show any change of the glottis on respiration, but on phonation a rapid approximation of the vocal cords, which may increase to spasmodic closure, occurs.

In *functional respiratory spasmus glottidis* inspiration is suspended by spasmodic closure of the glottis, an *inspiratory dyspnœa with stridor* sets in as soon as the patient inspires, while the expiration is normal or nearly so, and phonation goes on unobstructedly. During sleep the dyspnœa disappears, in contradistinction to the condition in paralysis of the posticus; there are also cases in which the inspiratory spasm occurs but temporarily or only at the height of the inspiration after the glottis had then normally extended. It seems that an isolated spasm of the posterior crico-arytænoid muscle occurs in rare cases in which the vocal cords are fixed in complete abduction during respiration.

PARALYSES OF THE LARYNGEAL NERVES AND MUSCLES

The neuroses of the larynx, which are of the greatest practical and diagnostic importance, are the various forms of *paralysis of the motor fibres of the laryngeal nerves.*

A. MOTOR PARALYSES IN THE REGION OF THE SUPERIOR LARYNGEAL NERVE

Paralysis of the Crico-Thyreoid Muscle.—Motor paralyses in the region of the superior laryngeal nerve of the crico-thyreoid muscle, which is supplied by the external branch of the superior laryngeal nerve, are observed in paralysis of the entire superior laryngeal nerve and in paralysis of the recurrent nerve, which undoubtedly participates in some cases in the innervation of the crico-thyreoid muscle. However, absolutely indubitable cases of complete, isolated paralysis of the crico-thyreoid muscle do not exist as yet, so that the changes in the function of the vocal cords ascribed to this muscle paralysis are constructed theoretically rather than based upon the result of laryngoscopical examinations. As such there have been assumed: *Hoarseness and a lowering of the pitch of the voice*, inability to produce high notes, with unilateral paralysis of the crico-thyreoid muscle, a low position, and shortening of the affected vocal cord. All these symptoms might be explained by the physiological function of the cryco-thyreoid muscles; *they stretch the vocal cords and make them tense* upon their contraction by the change of position of the thyreoid and cricoid cartilages.

Paralysis of the Epiglottis Depressors.—Paralysis of the epiglottis detractors (of the thyreo- and aryepiglottic muscles) has also been considered as an effect of paralysis of the superior laryngeal nerve; and the effect of a paralysis of these muscles—total immotility of the epiglottis which stands erect towards the base of the tongue—has, in fact, been seen by me and others in cases in which the diagnosis of an isolated paralysis of the superior laryngeal nerve was justified in accordance with the anæsthesia of the laryngeal mucous membrane and the preservation of function of the muscles which are supplied by the recurrent laryngeal nerve. However, the question as to the innervation of these muscles is still undecided, especially as von Ziemssen

has not been able to determine any movement of the epiglottis upon irritation of the exposed superior laryngeal nerve of an executed criminal a few minutes after the death of the latter.

B. PARALYSES IN THE REGION OF THE RECURRENT NERVE

1. COMPLETE PARALYSIS OF THE RECURRENS

Bilateral Paralysis of the Recurrens.—The recurrent laryngeal nerve, as stated above, being the real motor nerve of the larynx, which consequently causes the opening and the closing of the glottis, a *paralysis of both recurrent nerves* is bound to be followed by an inability to open and close the glottis. The position of the glottis resulting therefrom is called *cadaveric position,* according to the precedence of von Ziemssen. *The vocal cords then appear slightly narrowed in the laryngoscopical picture and absolutely motionless; the arytænoid cartilage especially is motionless during respiration and phonation, and is placed forward and inward owing to the absence of any kind of muscular tonicity.* At the same time there exists absolute aphonia and the inability to cough vigorously; the forced inspiration occurs with noise, because the air current causes passive coarse vibrations of the entire relaxed soft parts of the upper and median laryngeal space (von Ziemssen). Dyspnœa does not accompany bilateral paralysis of the recurrent nerve; only in small children it may not fail to appear, owing to the smallness of the glottis respiratoria, because the effect of the adductors is not present, and besides, the relaxed vocal cords are approximated by the inspiratory air current.

Incomplete Bilateral Paralysis of the Recurrent Nerve.—In *incomplete bilateral paralysis of the recurrent laryngeal nerves,* when one vocal cord is paralyzed more than the other, a condition will be found slightly differing from the picture just described. Above all, no complete aphonia is observed in this case, but only a deep, hoarse, slightly monotonous phonation, because the one vocal cord, which is alone paretic, is still sensible, although but slightly so; the cough, on the other hand, the same as in complete bilateral paralysis, is rendered difficult, and only possible with a "profusion of air." The laryngoscopical examination shows the completely paralyzed vocal cord in cadaveric position, the other, paretic one, in sluggish motion, respectively, as has been repeatedly observed recently, in the adduction position. The explanation of this latter condition has given rise to lively controversies. It is most probable, in my opinion, that, with incomplete paralysis of the abductors and adductors, the latter predominate over the former owing to their greater muscular power and mass, and thus produce the antagonistic position (so-called median position). According to the most recent experimental investigations, we have to assume that in paralysis of the recurrent laryngeal nerve the irritability of the abductors ceases decidedly earlier than that of the adductors, a further reason for the antagonistic adduction position of the paretic vocal cord.

Unilateral Paralysis of the Recurrens.—Much more frequent than the bilateral is the *unilateral paralysis of the recurrent nerve.*

The laryngoscopical examination shows cadaveric position of the paralyzed vocal cord with displacement of the arytænoid forward and inward; upon phonation immotility of the paralyzed vocal cord and of the respective arytænoid, and, on the other hand, extension beyond the median line of the rima glottidis of the intact vocal cord and of the unaffected ary-

tænoid. The latter, with its Santorini cartilage, usually "crosses" that of the paralyzed side in such a manner that it is placed *anteriorly* (Fig. 5 *c*), rarely *posteriorly* (Fig. 5 *b*).

This compensation in the motion of the non-paralyzed vocal cord (by the effect of the adductors, principally of the crico-arytænoideus lateralis of the healthy side) causes an almost complete closure of the glottis, which naturally is in an oblique position to the paralyzed side. In this condition moderately good phonation is possible in contradistinction to the state in bilateral paralysis; only the voice is weaker, of a slightly higher pitch, owing to the excessive tension of the unaffected vocal cord, and jarring,

a *b* *c*

FIG. 5.—PARALYSIS OF THE RECURRENT NERVE.

(*a*) Inspiratory position of the glottis in right-sided paralysis of the recurrent nerve. (*b*) Phonation position (oblique position) of the glottis in right-sided paralysis of the recurrent nerve with (unusual) crossing of the arytænoid respectively Santorini's cartilages. (*c*) A case of left-sided paralysis of the recurrent nerve with usual cross position of the arytænoid (position of the arytænoid of the healthy portion forward) oblique position of the glottis.

owing to the disturbed regularity of the vibrations of the healthy vocal cord by the adjacent, sometimes even subjacent, paralyzed vocal cord. Atrophy and narrowing of the latter occurs upon longer duration of the paralysis.

To mistake unilateral for bilateral paralysis of the recurrent laryngeal nerves appears absolutely impossible according to the above statements.

2. ISOLATED PARALYSIS OF THE INDIVIDUAL MUSCLES SUPPLIED BY THE INFERIOR LARYNGEAL NERVE

The diagnosis of isolated paralysis of individual muscles or muscle groups is very easy if we represent to ourselves a disturbance of the function of the respective muscle.

a. PARALYSIS OF THE GLOTTIS-CLOSING MUSCLES—PARALYSIS OF THE ADDUCTORS

Paralysis of the adductors embraces disturbance of the function of the lateral crico-arytænoid, of the transverse arytænoid, and of the thyreo-arytænoid muscles. The laryngoscopical examination shows the glottis open upon phonation in the inspiratory position; the vocal cords leave the well-known triangular cleft between them; phonation is soundless.

Paralysis of the adductors is a very frequent manifestation, preferably in the course of hysteria—of central origin—because the reflex closure of the glottis occurs promptly—i. e., the cough, etc., ringing.

Besides complete paralysis of the adductors there is not infrequently

found paralysis of every individual muscle of the adductor group, most frequently due to hysteria or in the course of laryngitis, etc.; furthermore, in consequence of overexertion in speaking, singing, etc. The diagnosis of these isolated muscle paralyses can only be made by means of the laryngoscope; however, under consideration of the characteristic form of the glottis.

PARALYSIS OF THE LATERAL CRICO-ARYTÆNOID MUSCLE

Paralysis of the Lateral Crico-Arytænoid Muscle.—The muscle, according to its course and insertion into the muscular process of the arytænoid cartilage, draws the

Fig. 6.

Paralysis of the Lateral Crico-Arytænoid.

Fig. 7.

Paralysis of Both Internal Thyreo-Arytænoid Muscles.

Fig. 8.

Paralysis of the Internus combined with Paralysis of the Transversus.

Fig. 9.

Paralysis of the Transverse Arytænoid (Inter-Arytænoid).

arytænoid forward and at the same time turns it slightly towards the centre, so that, upon its contraction, the vocal processes approximate, although not so close as upon contraction of the thyreo-arytænoid.

If its function is suspended, the co-operation of the muscle upon closing the glottis is wanting, and the latter is slightly patulous in the region of the apices of the vocal processes ("rhomboid form" of the glottis). However, isolated paralysis of the lateral crico-arytænoid muscle occurs but very rarely. A typical case of this kind was observed in my clinic; the above figure represents the laryngoscopical finding which was then determined and which was repeatedly demonstrated (Fig. 6).

PARALYSIS OF THE (INTERNAL) THYREO-ARYTÆNOID MUSCLE

Paralysis of the Internus.—The muscle, extending from the lower half of the thyreoid cartilage, respectively the inner side of the angle of the cartilage, to the outer lateral border of the arytænoid, follows essentially the course of the inferior

thyreo-arytænoid ligament. Upon contraction of its fibres, the vocal cords are pressed from the side towards the median line; this causes the vocal cords to become relaxed, inasmuch as their points of attachment approach each other owing to the contraction of the muscles; the gross tension is thus modified by the crico-thyreoid, respectively regulated, and even more so, because it is possible that individual portions of the vocal cord may become *tense* by the effect of the internus, as the fibres of the muscle terminate in part in the elastic tissue of the vocal cord itself. Finally, the vocal cord also widens upon contraction of the "internus."

If the muscle is paralyzed, the glottis gapes, showing a marked excavation of the paralyzed narrower vocal cord. If both interni are paralyzed, as is usually the case, the glottis upon phonation appears as an oval slit extending from the vocal processes to the anterior commissure (Fig. 7). The voice is, according to the degree of paralysis, weak, not clear, or absolute aphonia is observed, especially if paralysis of the internus muscle is combined with paralysis of the transverse arytænoid. In the latter case the glottis gapes not only in its anterior ligamentous portion, but also in the cartilaginous portion, while the vocal processes, turned inward by the functionally intact lateral crico-arytænoid, project slightly between the anterior oval and the posterior triangle of the gaping glottis (Fig. 8). If this becomes associated with paralysis of the laterals, this projection of the vocal processes disappears in the open glottis, and upon phonation the latter now forms a large triangle with the base posteriorly, the same as in quiet inspiration—i. e., now the picture of *paralysis of all the adductors* appears.

PARALYSIS OF THE TRANSVERSE ARYTÆNOID (INTERARYTÆNOID)

Paralysis of the Transverse Arytænoid.—In this condition the cartilaginous portion of the glottis alone gapes upon phonation, while the anterior ligamentous portion closes normally, according to the function of the muscle which, extended between the two exterior edges of the arytænoids, upon contraction draws the arytænoid cartilage towards the median line and causes the respiratory glottis to close (Fig. 9). Paralysis of the muscle impairs phonation: the voice is hollow and without strength, because air escapes upon intonation through the patulous glottis.

b. PARALYSIS OF THE ABDUCTORS OF THE GLOTTIS

Paralysis of the posterior *crico-arytænoid muscles* occurs not very rarely, and always is an important pathological condition, because it is the function of the posterior muscle to widen the glottis completely and to furnish a sufficiently wide portal of entrance to the air upon deep inspiration.

Effect of the Posterior Crico-Arytænoid.—The muscle extends from the posterior surface of the plate of the cricoid upward and outward to the lateral border of the muscular process of the arytænoid cartilage. Its contraction turns the arytænoids in such a manner that the vocal processes move outward and upward, changing the glottis into a widely gaping triangular slit. But also that portion of the glottis which is situated behind the vocal processes becomes extended by the effect of the muscle, so that the interarytænoid incisure gradually disappears. It seems that the muscle is active even in quiet respiration because the, then observed, width of the opening of the glottis is larger than that in complete paralysis.

Bilateral Paralysis of the Posticus.—If the muscle loses its contractility, we see in the laryngoscopical picture the vocal cords approximated in *bilateral paralysis of the posterior crico-arytænoid muscle,* which can be explained also in quiet inspiration by the omission of the last-named normal function of the posterior muscles. Upon unconstrained respiration this slightly reduced width of the glottis is evidently sufficient to allow enough air to pass to satisfy the respiratory requirements; however, in all greater exertions of the body, ascending stairs, etc., in short, in every active movement making greater demands upon the respiration, dyspnœa occurs—*inspiratory dyspnœa*—while expiration takes place normally and without difficulty. The dyspnœa gradually becomes permanent and high graded; the vocal cords are approximated, except a small cleft, in adduction position, explainable by the antagonistic contracture of the adductors. Upon deep inspiration the vocal cords become still more approximate, so as to touch each other almost completely. It has been attempted to explain this latter conduct in various ways. I am forced to concur in the view of those investigators who assume that the rarefication of the air in the thorax during each inspiration causes an aspiration of the vocal cords in paralysis of the posterior muscles, because in a case of my own the vocal cords *were bent downward in a funnel-shaped manner* during each inspiration.

The impaired inspirations are accompanied by much noise because the entering inspiratory air current causes the approximated vocal cords to vibrate. *Phonation is unchanged,* because normal adduction and tension of the vocal cords is possible. The *ensemble* of the above-named symptoms of paralysis of the posticus—viz., inspiratory laryngeal dyspnœa with stridor and unimpaired expiration as well as phonation—allows us to surmise paralysis of the posticus as being very probable, which condition can easily and positively be diagnosticated by laryngoscopical examination, because this will show the vocal cords to be close together and to come still nearer upon inspiration instead of parting.

Unilateral Paralysis of the Posticus.—The picture of *unilateral paralysis of the posticus* is less characteristic. The opening of the glottis in this condition is amply sufficient for respiration; even forced inspiration usually causes only slight dyspnœa. The laryngoscopical examination shows the vocal cord of the affected side advanced towards the median line, that of the healthy side functionating normally during respiration and phonation. The symptoms of unilateral paralysis are, therefore, of a very slight nature, so that their discovery is generally accidental during a casual laryngoscopical examination. As it has been demonstrated physiologically that the irritability of the posterior muscles ceases earliest, in comparison to that of other laryngeal muscles, in the freshly extirpated larynx, we will be most liable to expect these pictures of unilateral paralysis of the posterior muscles in unilateral paralyses of the recurrent laryngeal nerve which develop gradually, and we will have to pay attention principally to the motility during phonation of the arytænoid in question, to be able to exclude *complete* paralysis of the recurrent laryngeal nerve and to assume a unilateral paralysis of the posterior muscles.

Ætiological Diagnosis.—In accordance with the explicitly described symptoms and results of investigation, it is almost always easy to establish the diagnosis of laryngeal paralyses. However, we should never be satisfied with the simple determination of a paralysis, but always search for the *cause* of the paralysis in each instance. Let me remark, in the first place, that it is not always possible to demonstrate, or to assume, anatomical changes as causes of paralysis. On the other hand, the demon-

of which are clogged with extended coagulation formations, *absence or lessening of the respiratory murmur in these portions, while the note upon percussion remains unchanged and clear,* so long as the formation of coagulations does not extend to, or originate in, the alveoli, or atelectasis arises later on in those portions of the lung which correspond to the extension of the obstructed bronchi. If the coagulations are expectorated with very violent efforts of cough, all these symptoms disappear, to recur upon the repetition of the coagulations, which is characteristic in contradistinction to other forms of bronchiostenosis. The simultaneous hæmoptysis, which sometimes precedes, at others accompanies the expectoration of the bronchial coagulations, may also be turned to good account diagnostically; the fact that a laryngeal croup or a croupous pneumonia has preceded a bronchiostenosis may in a doubtful case point to the existence of a fibrinous bronchitis. The above proves that a great many of the symptoms of fibrinous bronchitis coincide with those of bronchiostenosis.

Ætiological Points of Support.—The consideration of the ætiology furnishes but few points of support that are applicable to the diagnosis of fibrinous bronchitis. Fibrinous bronchitis has been observed in the course of various infectious diseases (tuberculosis, scarlatina, diphtheria, pneumonia, etc.), and as a direct result of the action of injurious substances upon the respiratory mucous membrane; in some cases no cause can be found. *Staphylococci* have sometimes been demonstrated in the characteristic sputum recently; it may be permissible, therefore, to assume that it is a question of staphylococcic infection in the "primary" form of fibrinous bronchitis.

BRONCHIOSTENOSIS

Bronchiostenosis.—The symptoms of this affection will, of course, occur as soon as the lumen of the bronchi is narrowed to a considerable extent for some reason or other. The pathological picture resulting therefrom is rather uniform, and the diagnosis of bronchiostenosis can be made easily and with certainty. It is self-evident that the respiration is impaired, respectively altered, the more so, the speedier the stenosis has occurred. But the symptoms will vary according to the narrowing of the respiratory canal being located above or below the bifurcation of the bronchus—i. e., therefore, according to the presence of a tracheal or a bronchial stenosis. It should be remarked, however, that narrowing of the fine bronchi cannot be counted into the last-named category, because the symptoms caused thereby are entirely different and form an integral part of certain other well-characterized pathological pictures, viz., capillary bronchitis and bronchial asthma.

It is obvious that a more or less developed occlusion of the trachea and large bronchus impairs respiration, and is bound, therefore, to cause dyspnœa and the other signs of suffocation, i. e., laboured respiration, cyanosis, and an increase of blood pressure, with irritation of the vascular nerve centres. The pulse becomes tense and large, but may also become slowed and, besides, shows the respiratory fluctuations of blood pressure most markedly in the sphygmographic picture with pronounced dyspnœa, so that, eventually, a distinct inspiratory intermittent pulse is observed. If the impaired respiration lasts for some length of time the influence of

normal inspiration, which promotes the emptying of the veins and the pulmonary circulation, diminishes, and the sequels of engorgement—dilatation of the right heart, hyperæmia of the liver, lessening of the urinary secretion, etc.—will not fail to appear. The *respiration type* is characteristic in bronchial stenosis, especially that of the *inspiratory* dyspnœa, i. e., the inspirations are principally impaired; they appear prolonged, and are executed with the aid of the auxiliary inspiratory muscles, the scaleni, sternocleidomastoid, etc., with a great exertion of energy, and as, nevertheless, sufficient air is not introduced, a displacement worth mentioning of the lower borders of the lung does not occur. On the contrary, an inspiratory retraction of the intercostal spaces, of the supraclavicular and epigastric regions becomes manifest. The number of respirations is lessened, upon the whole, because an unusually long time is taken up by the inspiratory act before the nerve fibres, which stimulate the expiratory centre reflexly, attain a condition of stimulation.

Location of the Obstruction to Respiration.—The signs described so far in regard to the diagnosis prove that an obstruction to respiration exists which chiefly impairs the inspiratory act. Its location is determined by careful *physical examination.*

Results of Percussion.—*Percussion* reveals that no *change is demonstrable of the pulmonary resonance* in spite of dyspnœa. This in itself removes from the diagnosis those causes of the dyspnœa which consist in an obstruction of the alveolar respiratory surface by fluid and inflammatory exudate, or in compression of the lung by hydrothorax, pleuritic exudates, tumours, etc. But now we must decide whether the dyspnœa is of cardiac or nervous origin, or whether it is caused by an obstruction to the passage of air in the uppermost air tracts of the larynx, trachea, and larger bronchi.

Results of Auscultation.—The results of *auscultation* will show most plainly that the first-named conditions are not present, but a stenosis of the upper portions of the respiratory tract causes the dyspnœa. Although the *vesicular murmur* is not replaced by bronchial breathing, as is the case in displacement of the air from the alveoli, *yet it is considerably weakened, or may not be audible at all, which is in remarkable contrast to the preservation of the loud percussion sound over the respective portions of the lung.* A decrease of vocal fremitus can be determined at the same time, and upon respiration a peculiar hissing and gasping, so-called stenotic sound is heard, usually audible at quite a distance.

If the diagnosis of a stenosis of the upper air passages has thus become established beyond doubt, the question suggests itself in what portion of the same is the obstruction located, a question, the solving of which presents not only diagnostic, but, above all, the greatest therapeutic interest.

Differential Diagnosis between Laryngeal and Bronchial Stenosis.—In favour of *stenosis of the larynx* are: The very marked upward and downward movement of the larynx, which often amounts to several centimetres, upon inspiration and expiration. The respiration takes place with the head bent backward (Gerhardt). Furthermore, a careful observation

alveolar walls. If larger bronchi are obstructed by secretion, the vocal fremitus may be temporarily suspended, and respiration is very apt to become dyspnœic, the type of respiration in particular an accelerated one; but here, in contradistinction to the more lasting stenosis of the air passages in catarrh of the finer bronchi, it would always be a question only of a transitory dyspnœa, which disappears upon removal of the secretion by cough.

Engorgement Symptoms.—*Cyanosis* developing with the impairment of respiration becomes a lasting one and with it the other signs of *engorgement* develop (enlargement of the liver, gastric and intestinal catarrhs, decrease of the amount of urine containing albumin, dropsy, dilatation and hypertrophy of the right ventricle), if the bronchial catarrh becomes more intense and *chronic*. These symptoms take place on account of the emptying of the veins becoming deficient, owing to impaired expiration and to the cough, and the activity of circulation, which otherwise is brought about by normal respiration, ceases, the change in volume of the lungs being impaired owing to the obstruction of the bronchi. However, engorgement symptoms are always only insignificant and never become as intense as in emphysema, unless pulmonary emphysema—which really is a quite common sequela of chronic bronchial catarrh—or peribronchitic, respectively interstitial pneumonic processes, develop, especially in consequence of syphilis.

Chronic Bronchitis.—Otherwise the symptoms of *chronic bronchial catarrh* do not differ from those just described in the acute form. But the long duration of the catarrh is the cause that it gradually extends also to the finer bronchi; that the sibilant râles increase; that emphysema and ectasis of the bronchi develop, and that the sputum becomes of a peculiar quality; sometimes the secretion is sparse, viscid, gray, translucent, coming from the finer bronchi, and expectorated after severe exertions by cough ("catarrhe sec" of Laennec), at other times it is very abundant, puriform, cellular (bronchorrhœa puriformis), or deficient in cells, serous, abundant, filiform (bronchorrhœa serosa, pituitosa), or, again, of a fœtid odour as in pulmonary gangrene (bronchitis putrida).

Ætiological Factors.—The diagnosis of bronchitis is supplemented by the observation of the ætiological factors in the individual case. The principal causes are: inhalation of dust (especially coal, iron, and stone dust, tobacco and cotton dust in factories), inhalation of irritating gases (chlorine, etc.), infections (often brought on by cold), intoxications (iodine, bromine, alcohol), and constitutional diseases, nephritis, etc. Engorgements are predisposing factors for bronchial, the same as for other catarrhs, and it is well known that bronchitis is among the most frequent symptoms of pulmonary emphysema and of cardiac affections.

If the bronchial secretion decomposes within the bronchi, due to the effect of micro-organisms, and if this decomposition continues, this condition is usually diagnosticated as a special form, *putrid bronchitis*.

PUTRID BRONCHITIS

Putrid Bronchitis.—This affection is comparatively rare, and its diagnosis causes difficulties often because the sputum shows the very same qualities as that in bronchiectasis and in pulmonary gangrene—viz., the fœtid putrid odour and the well-known separation into three layers upon standing—an uppermost green-yellowish, containing frothy globules of sputum, a middle, transparent, serous one, and the lowest with the appearance of a purely purulent sediment with white-gray plugs, first discovered by Dittrich, consisting of detritus, fat, margaric acid, and fungi.

Differential Diagnosis.—It is therefore very easy to mistake these two affections for putrid bronchitis. Its differentiation from *pulmonary gangrene* offers the least difficulties, comparatively, in which, although elastic

If no points of support are found for the presence of the affections mentioned which may cause a bronchial stenosis, and if, furthermore, no alterations of the bony structures of the thorax can be demonstrated which may produce compression (as, for instance, a gravitating abscess which originates in a carious vertebra), then we must think of those causes of bronchiostenosis which are *situated within the bronchial lumen proper* and which obstruct and narrow the same. In the first place we must consider the sputum which may be present and the condition of the same. The expectoration of bronchial casts, which we have especially considered when describing *fibrinous bronchitis*, often clears the diagnosis at once, which often may be doubtful ætiologically. It is also permissible, if the sputum points to bronchitis, and if other symptoms are present which are in favour of the presence of considerable bronchial catarrh, to think of an *inflammatory thickening of the bronchial wall*. However, this connection is rather rare, and only when all other causes for the formation of a stenosis have been excluded is it admissible to make a presumptive diagnosis in this direction. Equally uncertain is usually the diagnosis of a carcinomatous infiltration of the bronchial wall or of polypi and other neoplasms in the walls of the large air passages, unless the neoplasms are located in the trachea and can be seen upon tracheoscopical examination. At most, a provisional diagnosis could be made of carcinomatous bronchiostenosis if recurring hæmorrhagic bronchitic sputum occurs, the course of the affection is afebrile and swelling of lymph glands in the throat or in the axilla or unfounded cachexia set in. If bronchial stenosis develops rapidly after the inhalation of pungent vapours, acute œdema of the bronchial mucous membrane may be surmised. The diagnosis reaches a greater degree of certainty if, in the later stages of *syphilis*, tracheal or bronchial stenoses occur, in which case cicatricial calcareous processes of a specific nature may be looked for in the trachea and bronchi (especially often in the bifurcation, as it seems). The diagnosis of a syphilitic character of bronchiostenosis can be made with a great deal of probability if the bronchitis which causes the latter condition is accompanied by the expectoration of a muco-purulent sputum and when, at the same time, the larynx is affected syphilitically, which can be easily determined by laryngoscopical examination, or when palatal defects, glandular swellings, etc., turn the diagnosis into a certain direction. The ætiological diagnosis is easy if in *tracheotomized patients* a tracheal stenosis develops, caused by granulation proliferations due to irritation of the cannula; these proliferations may dangerously narrow the lumen of the trachea. The diagnosis is also comparatively certain if it is a question of *foreign bodies* which have entered the trachea and bronchi—for the reason that foreign bodies modify the usual picture of stenosis of the large air passages characteristically in several particulars. Inasmuch, namely, as the foreign bodies in the trachea often change their location, they may cause sudden attacks of suffocation. It may also be that in certain postures of the body the position of the foreign body may become changed, thus temporarily increasing the dyspnœa, for which reason the patients will remain in a certain posture in such cases, carefully avoiding a change of the same. Of course, the history—i. e., the statement—of the patient that a foreign body has entered the trachea or bronchi, often directs and strengthens the diagnosis. But it should not be forgotten that the entrance of a foreign body sometimes occurs unperceived, and it does not cause symptoms of stenosis until after some time, after it has expanded, and is not until then able to obstruct the air passages. It may be mentioned, finally, that a stenosis has also been observed in *hysteria* which is dependent upon a spastic contraction of the tracheal and bronchial muscular structure.

PERFORATION OF THE BRONCHIAL WALL

Perforation of the Bronchi.—By way of supplement I wish to refer to *perforation of the bronchi*. This may occur either from the invasion of aneurysms or abscesses from without into the bronchial lumen, or from within outward, owing to an erosion of the bronchial wall by ulcerations and foreign bodies in the bronchi. The diagnosis is not so difficult as it would *a priori* appear, provided that the perforation causes a communication between bronchus and œsophagus. In such cases, namely, there occurs a symptom which at once points to that event. The swallowed food

of which are clogged with extended coagulation formations, *absence or lessening of the respiratory murmur in these portions, while the note upon percussion remains unchanged and clear,* so long as the formation of coagulations does not extend to, or originate in, the alveoli, or atelectasis arises later on in those portions of the lung which correspond to the extension of the obstructed bronchi. If the coagulations are expectorated with very violent efforts of cough, all these symptoms disappear, to recur upon the repetition of the coagulations, which is characteristic in contradistinction to other forms of bronchiostenosis. The simultaneous hæmoptysis, which sometimes precedes, at others accompanies the expectoration of the bronchial coagulations, may also be turned to good account diagnostically; the fact that a laryngeal croup or a croupous pneumonia has preceded a bronchiostenosis may in a doubtful case point to the existence of a fibrinous bronchitis. The above proves that a great many of the symptoms of fibrinous bronchitis coincide with those of bronchiostenosis.

Ætiological Points of Support.—The consideration of the ætiology furnishes but few points of support that are applicable to the diagnosis of fibrinous bronchitis. Fibrinous bronchitis has been observed in the course of various infectious diseases (tuberculosis, scarlatina, diphtheria, pneumonia, etc.), and as a direct result of the action of injurious substances upon the respiratory mucous membrane; in some cases no cause can be found. *Staphylococci* have sometimes been demonstrated in the characteristic sputum recently; it may be permissible, therefore, to assume that it is a question of staphylococcic infection in the "primary" form of fibrinous bronchitis.

BRONCHIOSTENOSIS

Bronchiostenosis.—The symptoms of this affection will, of course, occur as soon as the lumen of the bronchi is narrowed to a considerable extent for some reason or other. The pathological picture resulting therefrom is rather uniform, and the diagnosis of bronchiostenosis can be made easily and with certainty. It is self-evident that the respiration is impaired, respectively altered, the more so, the speedier the stenosis has occurred. But the symptoms will vary according to the narrowing of the respiratory canal being located above or below the bifurcation of the bronchus—i. e., therefore, according to the presence of a tracheal or a bronchial stenosis. It should be remarked, however, that narrowing of the fine bronchi cannot be counted into the last-named category, because the symptoms caused thereby are entirely different and form an integral part of certain other well-characterized pathological pictures, viz., capillary bronchitis and bronchial asthma.

It is obvious that a more or less developed occlusion of the trachea and large bronchus impairs respiration, and is bound, therefore, to cause dyspnœa and the other signs of suffocation, i. e., laboured respiration, cyanosis, and an increase of blood pressure, with irritation of the vascular nerve centres. The pulse becomes tense and large, but may also become slowed and, besides, shows the respiratory fluctuations of blood pressure most markedly in the sphygmographic picture with pronounced dyspnœa, so that, eventually, a distinct inspiratory intermittent pulse is observed. If the impaired respiration lasts for some length of time the influence of

If no points of support are found for the presence of the affections mentioned which may cause a bronchial stenosis, and if, furthermore, no alterations of the bony structures of the thorax can be demonstrated which may produce compression (as, for instance, a gravitating abscess which originates in a carious vertebra), then we must think of those causes of bronchiostenosis which are *situated within the bronchial lumen proper* and which obstruct and narrow the same. In the first place we must consider the sputum which may be present and the condition of the same. The expectoration of bronchial casts, which we have especially considered when describing *fibrinous bronchitis,* often clears the diagnosis at once, which often may be doubtful ætiologically. It is also permissible, if the sputum points to bronchitis, and if other symptoms are present which are in favour of the presence of considerable bronchial catarrh, to think of an *inflammatory thickening of the bronchial wall.* However, this connection is rather rare, and only when all other causes for the formation of a stenosis have been excluded is it admissible to make a presumptive diagnosis in this direction. Equally uncertain is usually the diagnosis of a carcinomatous infiltration of the bronchial wall or of polypi and other neoplasms in the walls of the large air passages, unless the neoplasms are located in the trachea and can be seen upon tracheoscopical examination. At most, a provisional diagnosis could be made of carcinomatous bronchiostenosis if recurring hæmorrhagic bronchitic sputum occurs, the course of the affection is afebrile and swelling of lymph glands in the throat or in the axilla or unfounded cachexia set in. If bronchial stenosis develops rapidly after the inhalation of pungent vapours, acute œdema of the bronchial mucous membrane may be surmised. The diagnosis reaches a greater degree of certainty if, in the later stages of *syphilis,* tracheal or bronchial stenoses occur, in which case cicatricial calcareous processes of a specific nature may be looked for in the trachea and bronchi (especially often in the bifurcation, as it seems). The diagnosis of a syphilitic character of bronchiostenosis can be made with a great deal of probability if the bronchitis which causes the latter condition is accompanied by the expectoration of a muco-purulent sputum and when, at the same time, the larynx is affected syphilitically, which can be easily determined by laryngoscopical examination, or when palatal defects, glandular swellings, etc., turn the diagnosis into a certain direction. The ætiological diagnosis is easy if in *tracheotomized patients* a tracheal stenosis develops, caused by granulation proliferations due to irritation of the cannula; these proliferations may dangerously narrow the lumen of the trachea. The diagnosis is also comparatively certain if it is a question of *foreign bodies* which have entered the trachea and bronchi—for the reason that foreign bodies modify the usual picture of stenosis of the large air passages characteristically in several particulars. Inasmuch, namely, as the foreign bodies in the trachea often change their location, they may cause sudden attacks of suffocation. It may also be that in certain postures of the body the position of the foreign body may become changed, thus temporarily increasing the dyspnœa, for which reason the patients will remain in a certain posture in such cases, carefully avoiding a change of the same. Of course, the history—i. e., the statement—of the patient that a foreign body has entered the trachea or bronchi, often directs and strengthens the diagnosis. But it should not be forgotten that the entrance of a foreign body sometimes occurs unperceived, and it does not cause symptoms of stenosis until after some time, after it has expanded, and is not until then able to obstruct the air passages. It may be mentioned, finally, that a stenosis has also been observed in *hysteria* which is dependent upon a spastic contraction of the tracheal and bronchial muscular structure.

PERFORATION OF THE BRONCHIAL WALL

Perforation of the Bronchi.—By way of supplement I wish to refer to *perforation of the bronchi.* This may occur either from the invasion of aneurysms or abscesses from without into the bronchial lumen, or from within outward, owing to an erosion of the bronchial wall by ulcerations and foreign bodies in the bronchi. The diagnosis is not so difficult as it would *a priori* appear, provided that the perforation causes a communication between bronchus and œsophagus. In such cases, namely, there occurs a symptom which at once points to that event. The swallowed food

of the acoustic character of the stenotic sounds usually at once allows the recognition of the larynx as the locality in which it arises, but, above all, the positive finding of the laryngoscopical examination, which demonstrates œdema of the glottis, croup, etc., as the cause of the condition. (Compare also p. 77.) However, it should not be forgotten that bronchial and laryngeal stenosis may sometimes exist simultaneously.

The following symptoms point to *tracheal,* respectively *bronchial stenosis*: Aside from the negative laryngeal finding, the slight displacement of the larynx upon forced respiration, in which the head usually appears to be bent, not backward, but rather slightly forward; furthermore, the relatively insignificant diminution of the number of respirations and, finally, that the consequences of the inspiratory thinning of the air in the thorax (retraction of the thoracic wall, etc.) are often not very marked. According to the seat of the impediment to respiration above or below the bifurcation of the bronchus, the picture of the affection will vary slightly, inasmuch as in stenosis of the lumen of *one* bronchus the respective half of the thorax will remain visibly behind, while the healthy side becomes excessively expanded, and the lung falls into a condition of inflation so that the diaphragm is low and the borders of the lung appear extended in all directions. The characteristic gasping stenotic sounds, which may also be palpable, diminution of the vocal fremitus and of the vesicular murmur, with a loud percussion note, can be determined on the affected side. The voice is usually faint, but the condition of the same must under no consideration be used as a sure characteristic sign between laryngeal and bronchial stenosis; neither is the presence or absence of cough decisive.

After the diagnosis of tracheostenosis or bronchiostenosis has been established after the above-described method, it remains only to find the manner in which it has occurred, especially as the therapeutic measures depend essentially upon the decision of this question.

Ætiological Diagnosis of Bronchiostenosis.—Primarily it will have to be investigated whether compression is present—i. e., whether pressure is exerted *from the adjacent parts* upon the trachea or the main bronchi. The presence of goître, the most frequent cause of tracheostenosis, is to be considered above all; eventually also a substernal goître; tumour of the *œsophagus* may also produce tracheostenosis. But I do not advise the use of the sound in the latter organ for diagnostic purposes (except possibly with a soft rubber sound without a stylet), because a positive result will not be obtained in most cases, as an œsophageal stenosis, which may eventually be found, is, of course, produced by the same compression which causes the tracheostenosis and, on the other hand, the sounding may actually be dangerous to life, especially in *aortic aneurysm.* As to the latter, the aneurysmal distention of the aortic arch often leads to tracheostenosis and bronchiostenosis; the diagnosis is easy in some cases, impossible in others. *Mediastinal tumours* are not infrequently the cause of tracheal and bronchial stenosis, and the formation of tumours in the lung proper (pulmonary carcinoma) may also lead to the same result. It is furthermore necessary never to neglect the examination of the heart, as pericardial exudates and dilatations of the left auricle have been found in rare cases to be the cause of stenoses of the bronchi, especially of the left bronchus. *Enlargement of the bronchial lymph glands* has also often been demonstrated to be the cause of bronchiostenosis (the possibility of the latter should be considered, if tuberculosis, respectively "scrofulosis" can indubitably be demonstrated).

acteristic bronchiectatic sputum sometimes, the same as that of putrid bronchitis, separates in the well-known three layers, the lowest of which forms a thick purulent sediment. A microscopical examination of the same will reveal pus cells, epithelial cells, both having partly undergone fatty degeneration, also crystals of fatty acids, and sometimes red blood cells which, if present in large quantities, cause a more reddish appearance of the sputum, and which, in rare cases, may give rise to the formation of hæmatoidin crystals. The presence of elastic fibres or connective-tissue shreds, which are also found occasionally, proves that the wall of the distended bronchus is affected and breaking down. The following microorganisms, especially, are found in bronchiectatic sputum: Streptococci, staphylococci, pneumococci, and the bacillus coli communis, which may give rise to fever and eventually produce a septicæmic infection.

Physical Demonstration of Bronchiectatic Cavities. — Although the diagnosis of bronchiectasis can be made with a great deal of probability in accordance with the above description, and still more so, the more the periodical expectoration of very large quantities of sputum manifests itself, the diagnosis does not become positive until *cavities have been demonstrated* in the pulmonary structure, provided they are superficial enough to be accessible to physical examination. If this is the case, tympanitic or tympanitic-metallic percussion notes may be expected, the cracked-pot sound, and a change of note. Auscultation will reveal bronchial breathing, eventually with a metallic quality, moist, ringing râles, and bronchophony. Increased vocal fremitus may be looked for on palpation. All these characteristic symptoms are absent if *the cavity is filled with secretion; but all, or at least some of them, may suddenly become prominent if the contents of the cavity are discharged by a spell of coughing.* This is a very important diagnostic consideration. To show "cavity signs," the cavities must be superficial or, at least, be surrounded by consolidated tissue, the presence of which is of principal significance in this case for the bronchial breathing and the bronchophony to become audible. The increase of the vocal fremitus is also in part connected with this condition, and in part with the fact that the cavity is in direct communication with the larger bronchi, and that the vocal vibrations may be transmitted undiminished through the latter, in fact, may be intensified by reflection from the resistant walls of the cavity. It will be necessary for us to consider these conditions repeatedly.

It is also the consolidation and atrophy of the pulmonary tissue existing in the neighbourhood of the bronchiectasis which explains the deficient respiratory excursus and the flattening of the thorax in the region of the bronchiectatic cavity.

Differential Diagnosis.—It is obvious that *bronchiectatic cavities* are very apt to be confounded with *phthisical* cavities, especially as there cannot be any doubt, anatomically, that bronchiectatic factors are largely responsible for the formation of tuberculous cavities in the lungs.

In practice usually the prognostically and therapeutically important question is to be decided, whether caseous processes and tuberculosis are present in the individual case besides a bronchiectatic cavity or not. The

will be brought up with a certain regularity by attacks of cough (and that with an absolutely intact condition of the larynx). The assumption of a bronchial perforation becomes a certainty, if comparatively *much* air escapes from a sound which is introduced into the œsophagus, and if the condition is such that, as was the case in an instance of my observation, *a powerful current of air* (extinguishing a light) *suddenly escapes* from the œsophageal sound, which is gradually pushed down or slowly retracted from the stomach, *at a certain place of the œsophagus*, and this occurs with quiet breathing (in which the normal pressure fluctuations of the air in the introduced sound are scarcely perceptible). This is brought about in such a manner that the window of the sound, during the gradual upward or downward passage, reaches the perforation, thus suddenly allowing unobstructed entrance of the expiratory current into the lumen of the sound, while this is not the case above or below this place.

BRONCHIECTASIS

The diagnosis of bronchial dilatation does not offer any difficulties in a great many cases, so that the bronchiectatic cavity is found post mortem at the assumed place; however, in a still greater number of cases the pathological condition of the lung remains undiscovered, and is found at autopsy as a not diagnosticated, secondary finding, surprising those who expect bronchiectases only when the symptoms are marked. If we consider the pathological picture of that form of bronchiectasis which allows of a positive diagnosis, we shall find that the *sputum* alone is so characteristic in some cases that the diagnosis may be made from the appearance of the same, although not with absolute certainty, yet with a great deal of probability.

The Sputum in Bronchiectasis.—The sputum is muco-purulent, of a dirty, yellow-green colour, the same as in chronic bronchial catarrh, but, in contradistinction to the sputum in the latter affection, it is mostly foul-smelling, because it has decomposed. However, the odour of the sputum in bronchiectasis is usually not so intensely fœtid and acrid as in putrid bronchitis and pulmonary gangrene, because generally it is expectorated relatively early and completely, therefore does not have much time to decompose. *It is particularly characteristic of the sputum in bronchiectasis that large masses of it are expectorated at once* (*"mouthful" expectoration*). This is obviously due to the fact that the wall of the dilated portions of the bronchi has gradually become insensible to the irritation of the secretion contained therein, so that a further accumulation of sputum, without relief by cough, is possible. But so soon as a part of the accumulated sputum enters the lumen of the nonectatic bronchi which communicate with the cavities, the mucous membrane of the bronchus reacts very energetically upon the partly decomposed sputum, and the coughing continues until the latter is more or less completely expectorated. This proves that remaining quietly in the same posture of the body, mostly upon that side which is the seat of the bronchiectasis, suppresses the cough, while a change of posture produces attacks of cough which again cause large masses of sputum to be expectorated suddenly, so that the patient's mouth is entirely filled with sputum after a few efforts of coughing. The expectoration occurs periodically with long pauses intervening, during which no, or but little, catarrhal sputum is discharged. The char-

connective-tissue particles are only exceptionally found in the sputum of bronchiectasis—namely, only when the tissue surrounding the cavity becomes eroded later on, or if actual gangrene supervenes.

Gangrene of the Lung.—The latter manifests itself by the penetrating odour of the sputum, which is of a dirty-gray colour and contains the characteristic necrotic pulmonary shreds and mycotic plugs, while the elastic fibres are entirely, or nearly, absent. Besides, the diffusion of the gangrene and the destruction of the pulmonary tissue caused thereby is a quicker one, eventually very rapidly progressing, which can easily be demonstrated by physical examination.

Putrid Bronchitis—Chronic Bronchial Catarrh.—It is sometimes impossible to differentiate from chronic bronchial catarrh, respectively putrid bronchitis, certain frequently occurring forms of bronchiectasis the course of which is not accompanied by the formation of circumscribed large cavities but with widely diffused, uniform, comparativly slightly pronounced dilatation of the bronchi. Such bronchiectases usually show absolutely the clinical picture of chronic bronchial catarrh, or, upon more intense decomposition of the secretion, that of putrid bronchitis. But the surmise of the presence of bronchiectases is permissible in such cases when the râles constantly remain in a certain part of the lung; the diagnosis becomes more certain if the râles become metallic owing to interstitially pneumonically infiltrated surroundings.

Complications—Cerebral Abscess.—It is not permissible to infer from the complications which are observed in the course of a bronchiectasis, viz., articular inflammations, cerebral abscess, etc., that a bronchiectasis is present, because such metastatic processes may occur also in other affections of the lungs which are accompanied by absorption of pyogenic organisms. At any rate, the diagnosis of bronchiectasis, if it is doubtful—if, for instance, it is based solely upon the constant presence of râles at a certain locality of the lungs—gains in certainty by an intercurrent *cerebral abscess.*

A Case of Bronchiectasis and Cerebral Abscess.—I once observed such a case. A man, aged twenty-nine years, was admitted to the hospital on June 8, 1886. Four weeks previously he observed a constantly progressing weakness of the left lower extremity, which two days later advanced to the upper extremity. At the same time, during the first eight days, twitchings occurred in both extremities, which gradually became completely paralyzed. *For four years he suffered from violent cough with abundant sputum, which was fœtid, and occasionally hæmorrhagic for about a year.* The examination of the lung revealed a relatively dull percussion note posteriorly below in the left base, no bronchial breathing at this place, but coarse, large, moist râles. The analysis of the nervous symptoms pointed to a *focal affection of the right hemisphere*—hemiplegia of the extremities of the left side, of the left facial nerve, dilatation of the left pupil, left-sided disturbance of the muscular and temperature senses, and anæsthesia of the left extremities and of the left half of the face. On July 4th severe headache occurred in the right half of the head, slowing of the pulse (52 beats); three days later fever to 102.5° F., lasting about a week; increasing somnolence. On July 14th severe pains in the left knee-joint; on July 17th severe hæmidrosis [left]. On July 19th severe headache, vomiting, twitching in the extremities of the right side, flexion contracture of the left extremities, both pupils narrow, pulse greatly retarded, death. The *diagnosis* was, in view of the coarse moist râles being restricted to the left lower lobe during the entire course of the disease, *bronchiectasis of the left lower lobe*; furthermore, *cerebral abscess of the right hemisphere*, and the probable seat of the latter was supposed to be the internal capsule in the posterior portions. The *autopsy* revealed an *enormous abscess, which had destroyed the*

answer to this question is easy, since we know that the constant absence of tubercle bacilli in the sputum is against the presence of pulmonary tuberculosis. It is less of a clinico-diagnostic than of an anatomical and ætiological interest to consider whether the origin of the cavity is caused by processes of decay which affect the pulmonary parenchyma, or whether the bronchiectatic cavity is due principally to a yielding of the bronchial walls to the inspiratory and expiratory pressure, to the overpressure of the engorging secretion, or to the train of atrophic processes in the lung acting from without and to pleuritic calcareous formations; it is the history, principally, in such cases, which is to be taken into consideration.

If the cavity is located in the apex of the lung, especially if *both* apices show symptoms of catarrh and consolidation; if the expectoration of the sputum occurs in small intervals and not, as explained above, in large masses and in sudden eruptions; and if the sputum contains plenty of elastic fibres, the diagnosis of cavity caused by caseous decay of the pulmonary parenchyma is permissible. However, it should not be forgotten that bronchiectases are not so rarely also found in the upper lobe, even, although very seldom, in both apices. The fever existing in these conditions should be considered with caution from a differential diagnostic aspect, because sometimes the non-tuberculous bronchiectasis is also accompanied by fever. Neither do hypertrophy of the heart and engorgement symptoms, occurring in the course of bronchiectases in consequence of atrophy of the lungs, speak directly for bronchiectasis and against tuberculosis, because, in my experience, secondary hypertrophy of the heart is not so infrequent in tuberculosis as is usually supposed.

Sacculated Pyopneumothorax.—The differential diagnosis is more difficult, in fact generally impossible, between *bronchiectasis and sacculated empyema which has perforated into the lungs or bronchi.* The cavity symptoms, the retraction of the thorax in certain regions, the evacuation of abundant purulent fœtid sputum, which depends upon change of posture, are uniformly present in both pathological conditions. The presence of hæmatoidin and cholesterin crystals, which form upon long-lasting stagnation of purulent masses, may be made use of in the diagnosis of empyema; but, to arrive at a somewhat positive diagnosis, it is necessary that more symptoms present themselves. Especially the history should be known, i. e., the course of the disease should distinctly point to the development of a suppurative pleurisy with sudden perforation. If the pus at the same time perforates externally, the diagnosis becomes at once clear, as a rule, although outward perforation of a bronchiectatic cavity may also occur in rare cases.

Abscess of the Lungs.—It is also possible that an *abscess of the lungs* perforating into the bronchi may simulate bronchiectasis. Here, too, the observation of the history is primarily decisive regarding the diagnosis, especially preceding fibrinous or embolic pneumonia, trauma of the lungs or bronchi. Furthermore, the sputum differs from that in uncomplicated bronchiectasis or sacculated perforating pyothorax in so far as in abscess it is usually purely purulent, of a flavourless, non-fœtid odour, and constantly contains shreds of pulmonary parenchyma, while elastic fibres and

connective-tissue particles are only exceptionally found in the sputum of bronchiectasis—namely, only when the tissue surrounding the cavity becomes eroded later on, or if actual gangrene supervenes.

Gangrene of the Lung.—The latter manifests itself by the penetrating odour of the sputum, which is of a dirty-gray colour and contains the characteristic necrotic pulmonary shreds and mycotic plugs, while the elastic fibres are entirely, or nearly, absent. Besides, the diffusion of the gangrene and the destruction of the pulmonary tissue caused thereby is a quicker one, eventually very rapidly progressing, which can easily be demonstrated by physical examination.

Putrid Bronchitis—Chronic Bronchial Catarrh.—It is sometimes impossible to differentiate from chronic bronchial catarrh, respectively putrid bronchitis, certain frequently occurring forms of bronchiectasis the course of which is not accompanied by the formation of circumscribed large cavities but with widely diffused, uniform, comparativly slightly pronounced dilatation of the bronchi. Such bronchiectases usually show absolutely the clinical picture of chronic bronchial catarrh, or, upon more intense decomposition of the secretion, that of putrid bronchitis. But the surmise of the presence of bronchiectases is permissible in such cases when the râles constantly remain in a certain part of the lung; the diagnosis becomes more certain if the râles become metallic owing to interstitially pneumonically infiltrated surroundings.

Complications—Cerebral Abscess.—It is not permissible to infer from the complications which are observed in the course of a bronchiectasis, viz., articular inflammations, cerebral abscess, etc., that a bronchiectasis is present, because such metastatic processes may occur also in other affections of the lungs which are accompanied by absorption of pyogenic organisms. At any rate, the diagnosis of bronchiectasis, if it is doubtful—if, for instance, it is based solely upon the constant presence of râles at a certain locality of the lungs—gains in certainty by an intercurrent *cerebral abscess.*

A Case of Bronchiectasis and Cerebral Abscess.—I once observed such a case. A man, aged twenty-nine years, was admitted to the hospital on June 8, 1886. Four weeks previously he observed a constantly progressing weakness of the left lower extremity, which two days later advanced to the upper extremity. At the same time, during the first eight days, twitchings occurred in both extremities, which gradually became completely paralyzed. *For four years he suffered from violent cough with abundant sputum, which was fœtid, and occasionally hæmorrhagic for about a year.* The examination of the lung revealed a relatively dull percussion note posteriorly below in the left base, no bronchial breathing at this place, but coarse, large, moist râles. The analysis of the nervous symptoms pointed to a *focal affection of the right hemisphere*—hemiplegia of the extremities of the left side, of the left facial nerve, dilatation of the left pupil, left-sided disturbance of the muscular and temperature senses, and anæsthesia of the left extremities and of the left half of the face. On July 4th severe headache occurred in the right half of the head, slowing of the pulse (52 beats); three days later fever to 102.5° F., lasting about a week; increasing somnolence. On July 14th severe pains in the left knee-joint; on July 17th severe hæmidrosis [left]. On July 19th severe headache, vomiting, twitching in the extremities of the right side, flexion contracture of the left extremities, both pupils narrow, pulse greatly retarded, death. The *diagnosis* was, in view of the coarse moist râles being restricted to the left lower lobe during the entire course of the disease, *bronchiectasis of the left lower lobe*; furthermore, *cerebral abscess of the right hemisphere*, and the probable seat of the latter was supposed to be the internal capsule in the posterior portions. The *autopsy* revealed an *enormous abscess, which had destroyed the*

entire internal capsule with the exception of the anterior upper portion; the adjacent brain substance was in a condition of white softening. *The left lower lobe of the lung contained a convolution of ectatic bronchi*, which were filled with thick purulent mucus of a disagreeable odour.

Hæmorrhage of the Lungs.—Sometimes bronchiectasis is complicated, besides cerebral abscess, by *amyloid disease* of the abdominal organs and by *pulmonary hæmorrhages*. I do not, in this respect refer to the already mentioned, sparse admixture of blood to the sputum which give to the latter a meat-water-like appearance; but there also occur, in rare cases, profuse hæmoptyses owing to the bursting of dilated veins of the bronchial wall or to the erosion of arterial twigs. The occurrence of hæmoptysis, therefore, does not permit us at once to conclude in doubtful cases that tuberculosis is present and not simple bronchiectasis.

BRONCHIAL ASTHMA

Only such cases of dyspnœa as are caused by purely nervous disturbances and which anatomically are based on the absence of lesions, or only on such as affect the nervous system exclusively, should properly be classified as bronchial asthma. If the conception of this affection is restricted to this narrow limit, its diagnosis will be rendered more precise.

Symptoms of Bronchial Asthma.—The clinical picture is very characteristic: The most severe dyspnœa occurs in periodical attacks with the signs of venous engorgement and cyanosis. The patient is actually struggling for breath, and it is *expiration* principally which is obstructed and which is only accomplished with the greatest effort, so that the expiratory muscles are intensely strained, especially the abdominal muscles, which appear board-like in their rigidity. Often twice the usual time is required for expiration owing to impairment of the same, and the number of respirations *in toto* is thus not increased in spite of the dyspnœa, but, on the contrary, mostly reduced. The long-drawn-out groaning and hissing breath sounds which accompany expiration can be heard at quite a distance, while the inspiration, which expands only the upper portions of the thorax principally, although also hissing and forcible, lasts but a comparatively short time only, and is less marked in view of the painful efforts at expiration. The consequences of deficient passage of air on expiration, the most prominent cause of which may be supposed to be a spasm of the muscular structure of the small and smallest bronchi, is an acute inflation of the lung, which can be demonstrated beyond doubt by percussion. While the borders of the lung are normal, except during the time of attack, they expand very much beyond the usual limit during the paroxysm. The lower borders extend downward by several intercostal spaces, cardiac dulness is considerably reduced, the heart being covered by the inflated borders of the lungs; yet the displaced borders remain the same during inspiration and expiration or only increase minimally on inspiration. The percussion sound is altered during the asthmatic attack; it is louder and of a peculiar note, which Biermer has designated as "bandbox sound." On *auscultation* we will observe decreased vesicular breathing and sibilant râles (corresponding to the groaning and hissing which can be heard from afar), which are especially intense on expiration and are the expression of stenosis in the small and smallest bronchi. They pass into small moist râles towards the

end of the attack, when a frothy gray-white sputum is expectorated with the smallest yellow plugs, in which the microscope will reveal, besides mucous cells, pigment cells—i. e., leucocytes and alveolar epithelium (which contains hæmosiderin originating from small hæmorrhages in the depth of the lung (von Noorden), and numerous Charcot crystals (Leyden)). The reason for this more abundant secretion of mucus towards the end of the attack is to be looked for in a fluxionary hyperæmia of the bronchial mucous membrane due to vaso-motor nerve disturbance, as has become probable by the laryngoscopic determination of a congestion (Störk) of the trachea and large bronchi. The *pulse* is small but tense, in accordance with the dyspnoic condition of the blood; the heart sounds are only faintly heard, owing to the overlapping of the lungs over the heart.

These symptoms of bronchial asthma are so marked that the diagnosis does not encounter any material difficulties. The *expiratory* character of the dyspnœa should be borne in mind above all.

Differential Diagnosis.—This allows us to exclude quite a number of affections which are accompanied by *inspiratory dyspnœa*, thus various laryngeal diseases: Œdema and spasm of the glottis, spasm of the laryngeal adductors, and paralysis of the posterior crico-arytænoid muscles, furthermore, tracheal and bronchial stenoses. *Expiratory* dyspnœa is found accompanying, besides bronchial asthma, emphysema and chronic bronchial catarrh. If, then, we restrict the diagnosis of bronchial asthma, as explained above, to those cases only in which a pure neurosis prevails, the differentiation of the dyspnœa in asthma from that in emphysema and bronchial catarrh is very easy. The absolute absence of changes in the lung during the time free from attacks in the asthmatic individual removes all doubts.

Emphysema and Bronchial Asthma.—However, it is in emphysema and chronic bronchial catarrh in which there occur, besides constant impairment of respiration, which may, it is true, present greater and lesser degrees of development according to the increase or decrease of the catarrhal forces, *attacks* of dyspnœa which have the *exquisite* type of bronchial asthma, arise suddenly, are very violent, and which may disappear rapidly without any changes in the intensity of the catarrh being demonstrable physically either before or after such an attack of dyspnœa. Their origin can undoubtedly be explained in such a manner that the catarrhally irritated bronchial mucous membrane temporarily leads to spasm of the bronchial muscles, a symptom which is analogous to spasms that occur in other inflammations of mucous membranes. It is also possible that irritations, which generally do not produce a spasm of the bronchial muscles, such as a draught of cold air, the accumulation of Charcot's crystals and of mucous spirals, are the occasional cause of the occurrence of spasm.

Connection between Charcot's Crystals and Curschmann's Spirals in Bronchial Asthma.—As to the connection of the two last-named formations with asthma in particular, it cannot be denied that these constituents, as was previously mentioned, are not found exclusively in the sputum of asthmatics and occasionally are not even found in this disease. They are, therefore, not *absolutely characteristic* diagnostically of bronchial asthma; however, the crystals and spirals are only very rarely found in

the sputum of patients who do not suffer from asthma, and, on the other hand, they are never absent in sputum which is discharged in asthmatic attacks. On the contrary, it is observed that the occurrence of these formations in the sputum coincides, in regard to time, with the asthmatic disorders and the developed asthmatic paroxysms, while they are not present during the remissions. It is true, though, as has been positively determined, that the attack may temporarily fail to appear in the asthmatic, while many crystals are found in the sputum. We are, therefore, not justified, according to the above, to assume a *causal* connection between the origin of the asthmatic paroxysm and the occurrence of crystals and spirals in the bronchioles. *They should rather be considered essentially as products of the asthmatic catarrh*, although the accumulation of viscid masses of mucus and especially of crystals may occasionally be able to *produce* an asthmatic attack owing to their location for the time being in the small bronchi, and to the varying sensibility of the mucous membrane. The same holds good, possibly, for the small *fibrin coagulations* which A. Schmidt has recently demonstrated in the sputum of an asthmatic patient as a, although not constant, yet quite frequent constituent of the sputum. They are seen microscopically as threads which are composed of fine, net-like fibres and which stain red in the triacid solutions in contradistinction to mucin-containing spirals which stain green.

FIG. 10.—SPUTUM FROM A CASE OF ASTHMA, showing Curschmann spirals, Charcot-Leyden crystals, leucocytes, and numerous free eosinophile granules; unstained specimen (Jakob). (Salinger and Kalteyer.)

Spasm of the Glottis and Asthma.—Bronchial asthma is most frequently confounded with *spasm of the glottis*. Characteristic of the latter are: The *inspiratory* character of the dyspnœa, the extensive excursions of the larynx during respiration, the inspiratory retraction of the epigastrium, the absence of acute inflation of the lungs, and the short duration of the dyspnœa, which in this affection only lasts a few minutes, while it may often continue for hours in bronchial asthma.

Cardiac Asthma.—The differential diagnosis between bronchial and *cardiac* asthma has been explicitly considered in the discussion of the diagnosis of the latter affection, and it is not necessary, therefore, again to enlarge upon it.

Diaphragmatic Spasm and Bronchial Asthma.—But I wish to mention in a few words the differential diagnosis between bronchial asthma and the rare cases of purely tonic *diaphragmatic spasm*. In this affection the inspirations take place spasm-like, with exertion of all the inspiratory muscles, and the thorax remains for several seconds in a forced inspiratory position, then to relax with a certain force into the expiratory position. The epigastrium, then, is bulged out on inspiration, and the heart displaced downward.

Ætiological Diagnosis.—After the diagnosis of a bronchial asthma has been established, it remains *to find the cause of the asthma*. First, the lungs are to be examined during the attack and during the remissions; if emphysema or bronchial catarrh are found they are the soil of the asthmatic attacks. If the lungs are normal, the nose and the nasopharyngeal space should be examined. There have been

so many positive observations made recently, according to which the removal of tonsils and nasal polypi, the cauterization of the spongy tissue of the muscles, etc., caused the sudden permanent disappearance of a long-existing asthma, that there can be no doubt as to the causal connection between these changes in nose and pharynx and bronchial asthma. Not until any support is found in this regard for the ætiological diagnosis are more remote causes to be thought of, such as affection of the abdominal organs, intestinal parasites, uterine diseases, etc. The possibility of the uterine nerves inciting a bronchial asthma is proven, among other things, by the fact that these attacks sometimes occur regularly only during menstruation. It was shown in the discussion of cardiac asthma (see p. 61) that some of the pathological conditions which formerly were considered to be causes of bronchial asthma, such as lead poisoning, overfilling of the stomach, nephritis, should not always at once be regarded as such. This is permissible only if the cardiac character of an asthma, which is produced by these pathological conditions, can be excluded with absolute certainty in comparison to bronchial asthma. It is usually easy to find idiosyncrasy occasionally as a cause of the asthma—i. e., to determine that the smelling of a certain perfume, the inhalation of certain kinds of grass pollen (in hay asthma), etc.—are causes of the attack. A *direct lesion of the vagus* may also occasionally be a cause, although in very rare cases only; it is therefore necessary never to omit an examination of the throat for swelling of the lymph glands, goître, etc. With regard to the therapy it is necessary for the ætiological diagnosis also to look for the presence of certain infectious diseases and anomalies of circulation which may favour the occurrence of asthma—i. e., we have to demonstrate in the individual case of bronchial asthma whether malaria, arthritis, anæmia, etc., are present or not.

DISEASES OF THE PULMONARY TISSUE

ATELECTASIS OF THE LUNGS

When a considerable number of alveoli lose their contents of air, so that the space is not occupied by fluid or solid masses, and alveolar walls are found in juxtaposition with other alveolar walls due to the fact that the pulmonary tissue has made use of its power of elasticity and contractility, a condition is brought about which is designated *pulmonary atelectasis* or *collapse of the lung*. The consequences of this cessation of function of the alveoli for respiration are, of course, as is the case in most pulmonary diseases, a deficient supply of air, inspiratory relaxation of the thorax, particularly in its lower parts (eventually unilateral, if only one lung is affected by extended atelectasis), and superficial accelerated respiration. The air entering the lungs in such a case is insufficient under all circumstances, and accordingly cyanosis and the other symptoms of carbonic-acid intoxication will set in. The boundaries of the lung are normal, as a rule, and eventual reduction will be compensated by vicarious expansion of those alveolar parts which are not atelectatic. Only when larger portions of the lungs have become atelectatic—i. e., if the airless part is several centimetres thick and has a length of at least 5 centimetres, and is at the same time superficially situated, dulness appears on gentle percussion. So long as the contents of air have not disappeared entirely dulness will be absent. A tympanitic percussion note can be demonstrated owing to the existing reduction of tension of the alveolar walls. In pronounced dulness, accentuated vocal fremitus, bronchial breathing, and bronchophony may be ex-

pected. Crepitation is of importance, which, if it corresponds to the dull locality and is marked, proves that the alveoli at that place, in part at least, are still accessible to the inspired air current.

Stasis Phenomena.—As the change which occurs normally in the pulmonary volume is a powerful aid for the onward course of the blood *in toto*, on greater extension of the atelectasis, and with this impeded inspiration, sufficient diastoles will no longer occur, and the influx to the heart will be reduced, therefore venous engorgement is the result; but as the elasticity of the lung acts in a similar manner upon the circulation, there will occur in the *lesser circulation* also an engorgement towards the right heart. It is true that the blood is pumped through the vessels of the atelectatic parts without obstruction, but the accelerated current taking place during inspiration (especially by expansion of the left auricle and acceleration of the deflux of blood from the pulmonary veins), ceases, at least in part, owing to the expansion of thorax and lungs which has become deficient. The consequence of the engorgement of the lesser circulation is dilatation of the right heart, which is demonstrable in more developed atelectasis. The heart dulness will be found more diffuse, all the more so if, in atelectasis of the pulmonary borders, the limits of the cardiac dulness appear uncovered to a greater extent.

None of these symptoms, however, conclusively proves the presence of atelectasis. They simply show that a portion of the lungs is deprived of a part of its air, and is of no account in respiration. From what has been said so far it cannot be decided what variety of deficiency this is. But the diagnosis of atelectasis can be correctly made in most cases *if the ætiology of the case is taken into consideration at the same time, and if other pathological conditions which occur with the same symptoms are excluded.*

Ætiological Diagnosis.—Apart from congenital atelectasis, which occurs in the newborn, in which the lung is airless at birth or remains partly airless owing to insufficient efforts at respiration, or on account of occlusion of the bronchi by mucus or meconium, atelectasis represents a *sequel* of other affections. If exudates in the pleura or pericardium, accumulation of air in the pleura, pneumothorax, tumours of the lungs and mediastinum, scoliosis of the spinal column, etc., prevent the full development of the lungs in the thoracic cavity, or if the excursion of the diaphragm is restricted by ascites, large tumours, abnormal accumulation of air, atelectasis of the lungs occurs. This is very frequently brought about by the fact that occlusion of the bronchial lumen occurs owing to accumulation of large quantities of mucus in the bronchi in which the cut-off air is resorbed by the alveolar capillaries in the atelectatic regions, and the lung contracts according to its elasticity. This manner of producing atelectasis is particularly found in the capillary bronchitis of children and in enteric fever. The origin of atelectasis in the latter disease is also promoted by the general debility which sets in owing to the prolonged course of the disease. This and similar diseases causing marked debility play an important part in the ætiology of atelectasis. The debilitated patients, especially when the sensorium is clouded at the same time, remain in one position for days without moving; thus the expansion of the lungs suffers on that side on which the patients recline, and in this manner sooner or later extensive atelectasis of the lungs will develop.

Differential Diagnosis.—*Therefore, the diagnosis must take into account all of the above-mentioned factors which cause atelectasis.* If there exists no sufficient reason for the formation of the same in the ætiology of

the case, the diagnosis is a very probable one only, and a confusion with other diseases is most likely. The dulness posteriorly at the base, of course, may be caused by an exudate or by an infiltration of a different nature. Primarily the vocal fremitus over the dull region is to be investigated. If the latter is increased, an accumulation of fluid in the pleural cavity as a cause of the dulness need not be considered. It is more difficult upon first glance to exclude pneumonic infiltration, hæmorrhagic infarct, tumour of the lungs, etc., because these pathological conditions are bound to show the same physical signs as atelectasis. The differentiation of these conditions from atelectasis is in reality easier than might be expected. They are all more chronic pathological affections of the pulmonary tissue, while atelectasis represents a *transitory* condition, a change of the lungs which passes off upon the disappearance of the disease which gives rise to the atelectasis, and which can also be modified or removed by certain procedures known to the physician. If the patient is made to assume a different posture, if, for instance, in left-sided dulness, he is made to turn to the right side, and *vice versa,* and instructed to inspire deeply and frequently, the atelectatic portion of the lung is better able to expand. The separation of the adjoining alveolar walls by the inspired air current occurs with *crepitation.* In a short time, often in the course of half a day, *a previously pronounced dulness, bronchophony and bronchial breathing, can with a change in the position of the patient and upon an energetic inspiration, become replaced by clear percussion sounds and a normal respiratory murmur.* This procedure is of the greatest importance in the diagnosis, and distinguishes atelectasis of the lungs from all those pathological changes of the lungs the course of which is accompanied by similar symptoms. It is certain that hæmorrhagic sputum is in favour of hæmorrhagic infarct or pneumonia, and in the same manner the presence of fever renders the presence of pneumonia probable. But it must not be forgotten that in both these pathological conditions hæmorrhagic sputum may be absent, and on the other hand febrile affections favour the origin of atelectasis.

Circumscribed Atelectasis.—A circumscribed atelectasis comprising only a few square centimetres can, as stated before, hardly be diagnosticated; on the other hand, it is permissible at least to make a probable diagnosis of limited or commencing atelectasis, if *constant crepitation* can be heard in circumscribed areas of the lungs in patients whose affection favours, according to experience, the formation of pulmonary atelectasis. I emphasize the fact, that crepitation in such cases must be constant; for transitory crepitation disappearing after a few respirations is observed in many patients—in fact, in healthy individuals who have been lying on their back for some length of time—and is of no diagnostic importance.

CONGESTION OF THE LUNGS

Cause of Congestion.—As in marantic atelectasis, the mechanical factor plays an important *rôle* also in congestion of the lungs. Here also the most dependent part of the lungs is the seat of the affection; in the dorsal position of the patient the posterior parts of the lower lobes; if the patient lies constantly upon the side, it is the correspondingly most dependent part of the lung on this side in which the circulation of the blood is impeded.

Besides, collapse of the lungs is generally found in the same places. But one condition is particularly necessary for the occurrence of congestion, the type of passive hyperæmia and its sequelæ, the stasis and exudation of blood corpuscles and plasma into the alveoli—namely, *a considerable decrease in the active working power of the heart.* This is conditioned upon the most various pathological processes, upon grave febrile diseases, in particular the infectious diseases, most frequently in enteric fever, in the senile and in the cachectic, in long-lasting death agony, etc. The occurrence of congestion of the lungs is favoured by all factors which prevent the normal change of volume of the lungs and thus retard the circulation and especially the velocity of the blood current in the pulmonary vessels. The conditions which promote the occurrence of congestion are: ascites, meteorism, abdominal tumours, in short, factors which do not allow of a sufficient contraction of the diaphragm. In fact, even the adjoining position of an enlarged liver to the diaphragm is a reason that the action of the diaphragm on the right side takes place with more difficulty than on the left, and accordingly the congestion occurs more on the right side than on the left. In by far the majority of cases, however, congestion of the lungs is a bilateral affection.

Symptoms of Congestion.—*The symptoms* are those of deficient power of the heart and impediment to respiration—i. e., weak accelerated pulse, dilatation of the right heart, and the often-named signs of venous engorgement and carbonic-acid poisoning, symptoms which, because peculiar to the most varied pathological conditions, are, of course, of secondary diagnostic value. Upon the patient raising himself there is found posteriorly at the base of the lungs during the onset of the formation of the congestion, as long as the amount of air in the alveoli is reduced, but not yet quite driven out or absorbed, a tympanitic note or relative dulness and decreased vesicular respiration; later, dulness progressing from below upward (which is absolute in complete consolidation), bronchial breathing, bronchophony and increased vocal fremitus.

Hypostatic Pneumonia.—If the congestion, as is frequently the case, in its further course passes into inflammation, particularly if a catarrhal or slight fibrinous *pneumonia* develops, the symptoms mentioned will not be changed by this occurrence, but now fever will supervene. But it must always be considered that in simple congestion fever may be present as a consequence of the original affection. The presence of blood in the sputum is likewise not pathognomonic of the diagnosis of the formation of a *hypostatic pneumonia*, inasmuch as also in bronchial catarrh, which so usually precedes the formation of congestion, the cough causes, at least occasionally, a slightly hæmorrhagic expectoration. If the latter becomes thin and fluid, frothy, of a bright-red colour, if the râles become moist, the supervention of *pulmonary œdema* to the congestion may be diagnosticated if the other physical symptoms are in favour of an existing congestion. If, finally, the sputum is purely bloody, or red-brown, especially if chills have preceded, or not only feebleness of the heart has occurred, but, besides, a cardiac lesion can be demonstrated, upon dulness posteriorly at the base and the presence of the above-described physical signs, *hæmorrhagic infarct* of the lungs

should be thought of rather than congestion, but only when the dulness is unilateral, for the pulmonary infarct, as is well known, almost always occurs on one side, whereas congestion is bilateral. The diagnosis of hypostasis and of hypostatic pneumonia may become doubtful in various directions. However, in general it can almost always be made with certainty and correctly, if the gradual manner of its origin, the almost always noticeable bilateral occurrence of the process, the seat and, above all, the ætiology are considered, as well as the question of the presence of conditions causing its origin—that is, the diminution of the energy of the activity of the heart, and the mechanical factor which plays the principal *rôle* in the genesis of congestion of the lungs.

PULMONARY EMPHYSEMA

In contradistinction to the above-discussed conditions in vesicular *emphysema* it is a question of dilatation of the alveoli with disappearance of the interalveolar sæptum. The alveolar dilatation is a *permanent* one, and differs materially from the often-discussed transitory, acute inflation of the lungs, with which we became acquainted as a consequence of the various affections of the respiratory organs, in particular in capillary bronchitis and in bronchial asthma.

In emphysema of the lungs there is a gradual increase of volume which causes the lungs partially to cover the organs adjacent to them. Another characteristic is (apart from the disappearance of coal pigment in the emphysematous parts of the lungs, which is of significance in a pathologico-anatomical respect) *the loss of the elastic fibrous tissue of the pulmonary structure, the disappearance of the sæpta between the individual expanded alveoli and with it the respective capillary tissue*; the expansion of the vessels in the alveolar walls causes at the same time a narrowing and, finally, an obliteration of numerous vessels with fatty degeneration of the same. If we adhere to these fundamental changes in emphysema, the subsequent symptoms which are to be considered in the clinical picture of emphysema are easily comprehensible and can be utilized for the diagnosis of the affection. The two most important of these subsequent signs are: *The changes of respiration and the disturbances in the circulation of the blood.*

Changes in Respiration.—While inspiration normally always takes place with the active participation of the diaphragm and intercostal muscles, it is well known that expiration in quiet breathing takes place in such a manner that the inspiratorily expanded thorax relaxes according to its gravity, and elastic forces, especially the elasticity of the pulmonary tissue, are brought into action, inasmuch as the latter decreases the inspiratorily inflated lungs, draws the thoracic walls inward, and favours the rising of the diaphragm. Thus, if the elasticity of the pulmonary structure is materially decreased, as is the case in emphysema, *expiration* is rendered difficult and retarded; *therefore, the dyspnœa of the emphysema is principally an expiratory one.*

But inspiration also shows disturbances. It is true the reduction of the elasticity of the lungs in itself would facilitate the inspiratory expansion of the lungs; how-

ever, the deficient expiratory reduction of the lungs brings about a retarded insufficient irritation of the nerve fibres passing into the inspiratory centre. Furthermore, the tension of the elasticity of the thoracic walls, which during rest takes place normally and through the elastic tension of the lungs, and which facilitates the introduction of inspiratory movements, must suffer; more so, if the costal cartilages gradually ossify in emphysema and the *thorax becomes rigid.* Finally, the quantity of air remaining in the lungs after expiration is greater, therefore the aeration is rendered difficult and the air hunger is unavoidable. This latter is increased by the disappearance of the alveolar walls and the obliteration of the pulmonary capillaries, which cause a restriction in the absorption of oxygen. The difficulty of introducing sufficient oxygen on inspiration and to force the air out on expiration manifests itself by the following changes in the respiration, of importance for diagnosis: *The inspiration is forced*, calling into action all auxiliary inspiratory muscles at disposal. Besides the scaleni and sternocleidomastoid, which bulge out of the throat as hard, tense cords, in the higher grades of emphysema, principally the trapezii and the extensors of the spinal column come into action on inspiration; in fact, the patients firmly plant their arms, fix the shoulder girdle, and cause the pectoralis minor to co-operate in the *forced inspiration.* The principal inspiratory muscle, however—the diaphragm—is crippled in its activity, for during expiration the latter does not rise as normally but remains far down, therefore the following inspiration cannot effect a flattening of the normally arched diaphragm. Hence the effect of its contraction is *nil.* The diaphragmatico-abdominal type of respiration disappears and is replaced by the purely costal type; at the same time, the epigastric region will be drawn in on inspiration, in case special obstruction is present to the entrance of air, in particular occlusion of the bronchi by secretion. This we have had occasion to observe in other conditions with obstruction to the entrance of air. After all this has taken place, it is easily conceivable that in emphysema the thorax is in the *constant* maximal inspiratory position.

For the *expiration*, as stated above, suffers the greatest reduction in emphysema; even the forced contractions of the respiratory muscles, which the emphysematous patient executes in order to empty the alveoli more completely, are of very little use, owing to the rigidity of the thorax, although the abdominal muscles are made very tense, and the patients endeavour, by bending the body forward, to compress the intestines and force the diaphragm upward. When in coughing the lungs are pushed outward and upward in the direction of the least resistance, the pulmonary apices under such conditions reach over the clavicle in the shape of thick, round, bulging masses.

According to the above description the respiratory excursus is very insignificant. The vital capacity is reduced considerably (to 1,000 cc. and below), and the respiratory frequency is increased; at times the dyspnœa increases to asthmatic attacks, which are the consequence of a temporary spasm of the bronchial muscles, and runs a course resembling the clinical type of bronchial asthma.

Physical Diagnosis of Emphysema.—If the mere contemplation of the described respiratory changes points to an existing emphysema as a cause of the same, the diagnosis does not become certain until after further physical examination of the thorax and the lungs. *Inspection* shows a remarkable dilatation of the thorax in most cases; the sterno-vertebral diameter, particularly, has experienced considerable enlargement. The thorax is distended, especially in its upper and middle portions, while the lower parts (corresponding to the most frequent manner of the origin of the emphysema, by violent expiratory pressure movements, which act upon the pulmonary tissue, either weakened or changed through inflammatory processes) are encircled by the very tense expiratory muscles which are in-

should be thought of rather than congestion, but only when the dulness is unilateral, for the pulmonary infarct, as is well known, almost always occurs on one side, whereas congestion is bilateral. The diagnosis of hypostasis and of hypostatic pneumonia may become doubtful in various directions. However, in general it can almost always be made with certainty and correctly, if the gradual manner of its origin, the almost always noticeable bilateral occurrence of the process, the seat and, above all, the ætiology are considered, as well as the question of the presence of conditions causing its origin—that is, the diminution of the energy of the activity of the heart, and the mechanical factor which plays the principal *rôle* in the genesis of congestion of the lungs.

PULMONARY EMPHYSEMA

In contradistinction to the above-discussed conditions in vesicular *emphysema* it is a question of dilatation of the alveoli with disappearance of the interalveolar sæptum. The alveolar dilatation is a *permanent* one, and differs materially from the often-discussed transitory, acute inflation of the lungs, with which we became acquainted as a consequence of the various affections of the respiratory organs, in particular in capillary bronchitis and in bronchial asthma.

In emphysema of the lungs there is a gradual increase of volume which causes the lungs partially to cover the organs adjacent to them. Another characteristic is (apart from the disappearance of coal pigment in the emphysematous parts of the lungs, which is of significance in a pathologico-anatomical respect) *the loss of the elastic fibrous tissue of the pulmonary structure, the disappearance of the sæpta between the individual expanded alveoli and with it the respective capillary tissue*; the expansion of the vessels in the alveolar walls causes at the same time a narrowing and, finally, an obliteration of numerous vessels with fatty degeneration of the same. If we adhere to these fundamental changes in emphysema, the subsequent symptoms which are to be considered in the clinical picture of emphysema are easily comprehensible and can be utilized for the diagnosis of the affection. The two most important of these subsequent signs are: *The changes of respiration and the disturbances in the circulation of the blood.*

Changes in Respiration.—While inspiration normally always takes place with the active participation of the diaphragm and intercostal muscles, it is well known that expiration in quiet breathing takes place in such a manner that the inspiratorily expanded thorax relaxes according to its gravity, and elastic forces, especially the elasticity of the pulmonary tissue, are brought into action, inasmuch as the latter decreases the inspiratorily inflated lungs, draws the thoracic walls inward, and favours the rising of the diaphragm. Thus, if the elasticity of the pulmonary structure is materially decreased, as is the case in emphysema, *expiration* is rendered difficult and retarded; *therefore, the dyspnœa of the emphysema is principally an expiratory one.*

But inspiration also shows disturbances. It is true the reduction of the elasticity of the lungs in itself would facilitate the inspiratory expansion of the lungs; how-

ever, the deficient expiratory reduction of the lungs brings about a retarded insufficient irritation of the nerve fibres passing into the inspiratory centre. Furthermore, the tension of the elasticity of the thoracic walls, which during rest takes place normally and through the elastic tension of the lungs, and which facilitates the introduction of inspiratory movements, must suffer; more so, if the costal cartilages gradually ossify in emphysema and the *thorax becomes rigid.* Finally, the quantity of air remaining in the lungs after expiration is greater, therefore the aeration is rendered difficult and the air hunger is unavoidable. This latter is increased by the disappearance of the alveolar walls and the obliteration of the pulmonary capillaries, which cause a restriction in the absorption of oxygen. The difficulty of introducing sufficient oxygen on inspiration and to force the air out on expiration manifests itself by the following changes in the respiration, of importance for diagnosis: *The inspiration is forced*, calling into action all auxiliary inspiratory muscles at disposal. Besides the scaleni and sternocleidomastoid, which bulge out of the throat as hard, tense cords, in the higher grades of emphysema, principally the trapezii and the extensors of the spinal column come into action on inspiration; in fact, the patients firmly plant their arms, fix the shoulder girdle, and cause the pectoralis minor to co-operate in the *forced inspiration.* The prineipal inspiratory muscle, however—the diaphragm—is crippled in its activity, for during expiration the latter does not rise as normally but remains far down, therefore the following inspiration cannot effect a flattening of the normally arched diaphragm. Hence the effect of its contraction is *nil.* The diaphragmatico-abdominal type of respiration disappears and is replaced by the purely costal type; at the same time, the epigastric region will be drawn in on inspiration, in case special obstruction is present to the entrance of air, in particular occlusion of the bronchi by secretion. This we have had occasion to observe in other conditions with obstruction to the entrance of air. After all this has taken place, it is easily conceivable that in emphysema the thorax is in the *constant* maximal inspiratory position.

For the *expiration*, as stated above, suffers the greatest reduction in emphysema; even the forced contractions of the respiratory muscles, which the emphysematous patient executes in order to empty the alveoli more completely, are of very little use, owing to the rigidity of the thorax, although the abdominal muscles are made very tense, and the patients endeavour, by bending the body forward, to compress the intestines and force the diaphragm upward. When in coughing the lungs are pushed outward and upward in the direction of the least resistance, the pulmonary apices under such conditions reach over the clavicle in the shape of thick, round, bulging masses.

According to the above description the respiratory excursus is very insignificant. The vital capacity is reduced considerably (to 1,000 cc. and below), and the respiratory frequency is increased; at times the dyspnœa increases to asthmatic attacks, which are the consequence of a temporary spasm of the bronchial muscles, and runs a course resembling the clinical type of bronchial asthma.

Physical Diagnosis of Emphysema.—If the mere contemplation of the described respiratory changes points to an existing emphysema as a cause of the same, the diagnosis does not become certain until after further physical examination of the thorax and the lungs. *Inspection* shows a remarkable dilatation of the thorax in most cases; the sterno-vertebral diameter, particularly, has experienced considerable enlargement. The thorax is distended, especially in its upper and middle portions, while the lower parts (corresponding to the most frequent manner of the origin of the emphysema, by violent expiratory pressure movements, which act upon the pulmonary tissue, either weakened or changed through inflammatory processes) are encircled by the very tense expiratory muscles which are in-

serted at the lower part of the thorax like a firm, unyielding girdle. At the time of the origin of the emphysema, the forced expirations with closed glottis push the air into the upper parts of the lungs; thus the thorax gradually assumes a *barrel-shaped* form, but in my experience this is not necessarily always the case. In some instances the lateral diameter is widened from left to right, in others the form of the thorax is fairly normal, especially if the emphysema should not have commenced until in later life, at a time in which the costal cartilages were already ossified. At any rate, the characteristic *barrel-shaped chest* is absent in all cases in which an inspiratory compression, which was abnormal for certain portions of the lungs (as in vicarious emphysema), became the cause of the alveolar ectasis. The intercostal spaces are broadened, the neck appears thick owing to the hypertrophy of its auxiliary respiratory muscles.

Owing to the generally slight expansion of the thoracic wall, feeble vocal fremitus can be demonstrated upon *palpation*. The conduct of the fremitus, however, varies in the individual case. If the not too rigid thoracic wall expands well, and if no severe bronchitis is present, vocal fremitus may be normal, in fact, owing to the better conduction of the emphysematous chest—that is, less tense pulmonary tissue—it may be more marked than normal. *Percussion* will show, as the most important result, extension of the pulmonary borders. Anteriorly they extend to the eighth rib and below, posteriorly to the twelfth dorsal vertebra, not infrequently to the first or second lumbar vertebra. It is especially remarkable, and of significance in a diagnostic respect, that the borders of the lung cannot, or but little, at most a finger-breadth, be displaced on inspiration. The note is slightly changed. It is often very loud, in the posterior and lateral parts especially sonorous, with sometimes a peculiar timbre, which owing to the diminished tension of the pulmonary tissue in emphysema is midway between tympany and normal vesicular resonance (Schachtelton) [bandbox note]. The lower border of the liver is demonstrably displaced inferiorly, owing to the low position of the diaphragm, especially so in the severe grades of emphysema—i. e., it extends beyond the costal arch in the mamillary line. The spleen descends for the same reason; the superior splenic dulness begins lower. Nevertheless, in my experience, the spleen is *never palpable* in emphysema even upon most careful palpation so long as its enlargement is not due to other conditions, especially engorgement. The defective motility of the diaphragm is the cause of the fact that the inferiorly displaced spleen is not palpable on inspiration.

The results of *auscultation* are not so characteristic. The respiratory murmur is simply decreased. It is distinguished by its soft character, consequently the result of auscultation alone for the trained clinician gives the probability of the presence of an emphysema. Besides, râles are mostly always present, caused by the bronchitis accompanying the emphysema.

Circulatory Disturbances—Stasis in the Lesser and Greater Circulation.—The changes which occur in consequence of the disease in the *circulatory apparatus* are not less important for the symptom-complex and the diagnosis of emphysema. As the elastic tension of the lungs normally exert-

a favourable influence upon the lesser circulation inasmuch as the left auricle is expanded by this tension, and the deflux of the blood from the pulmonary capillaries facilitated, it is obvious that the decrease of pulmonary elasticity in emphysema must be followed by an *impediment in the lesser circulation*. On the other hand, owing to the destruction of numerous capillary areas of the lung which is caused by the anatomical changes of emphysema, the pressure in the pulmonary artery is increased. Furthermore, as expiration favours the systole of the heart and the filling of the aortic system, the consequence is that the arteries are insufficiently filled and the pulse will be small. The result of the emphysematous affection of the lung upon the circulation is, therefore, reduced blood pressure in the arterial system, obstruction to the pulmonary circulation, and the final result, a *reduced velocity of the blood current in the latter*. Furthermore, owing to the deficient action of the left heart and the engorgement in the pulmonary circulation, the deflux of the blood from the veins of the body into the right heart is difficult, and thus an engorgement develops in the veins of the greater circulation, and a decreased velocity in the blood current in the capillaries of the body.

Dilatation and Hypertrophy of the Right Heart.—This severe injury to the entire circulation is naturally followed by secondary changes in the heart—*dilatation and hypertrophy of the right heart*; but it is the latter which for some length of time corrects the faulty circulation more or less completely. However, the extension of the compensation is limited; as soon as it relaxes the signs of engorgement become evident. Therefore, it is the principal object of the diagnosis, which is not less important than the combination of the anatomical changes of the lung on account of the therapeutic measures to be taken, to observe the momentary capability of the heart in emphysema and to explain it correctly.

Difficulties in the Diagnosis of Cardiac Enlargement in Emphysema.—In contradistinction to other morbid conditions which are accompanied by dilatation and hypertrophy of the right ventricle, there are certain difficulties in emphysema in demonstrating dilatation of the heart. It is evident that the heart is considerably covered by the emphysematous borders of the lung, and furthermore, that with the descending diaphragm the same as all the organs adjoining the latter (liver and spleen) the heart, too, must become displaced. When the diaphragm in consequence of emphysema is too much flattened, the central tendon upon which the heart is situated has also descended, consequently the heart must leave its normal position. According to experience it will then assume a more horizontal position, the base sinking posteriorly to the right and the apex turning to the left and outward, and it is then entirely covered by the lungs. Accordingly, nothing can be observed of the apex beat, but in the epigastrium near the xiphoid process vigorous diffuse pulsations can be observed. They are caused by the contraction of the horizontally placed, right ventricle, which has descended and is hypertrophied. Eventually the border of the right ventricle, which becomes hardened during systole, may be palpated. On *percussion*, the displacement of the heart is observed, inasmuch as the cardiac dulness commences one to two intercostal spaces lower down. In greatly developed emphysema the lung covers the heart so completely that, although dilatation and hypertrophy of the right ventricle exists, the cardiac dulness is not only not diffuse but is decreased or entirely absent. Therefore, it is not surprising that considerable enlargement of the heart is found at the autopsy in cases in which only a small area of cardiac dulness could be demonstrated during life. In those cases in which emphysema can be positively determined, and normal cardiac dulness is present, it is permissible to con-

clude therefrom that the heart is considerably enlarged, because the lungs, although emphysematous, were not able to reduce the borders of the heart below the normal.

The diagnosis of hypertrophy of the right ventricle is aided by *auscultation*, inasmuch as the second pulmonary sound appears slightly accentuated. The sounds of the heart are very faint, often scarcely audible owing to the heart being covered by the lungs. Sometimes there appear, without the occurrence of organic valvular changes, systolic cardiac murmurs, which must be explained differently in each individual case, as accidental murmurs or as tricuspid-valve murmurs, owing to a relative tricuspid insufficiency which was brought about in the course of the dilatation of the right ventricle. If the engorgement is more developed the well-known symptoms of the same become prominent: cyanosis, venous pulsation in neck, enlargement of the liver, albuminuria, scanty urination (owing to engorgement of the kidneys), hydrothorax, ascites, anasarca, gastric and intestinal catarrh, chronic bronchitis, which latter, although generally primarily present, causes the development of emphysema, but may also be secondary to emphysema, or an existing bronchitis may be aggravated owing to the engorgement of the lungs (on account of the partial deflux of the bronchial and pulmonary veins). Cough and expectoration are not characteristic; they are merely the result of the bronchitis. The expectoration is sometimes viscid, mucous, or sometimes even purulent. Blood is rarely present. If the latter is abundant, a complicating pulmonary phthisis is to be thought of, or the presence of a hæmorrhagic infarct, the formation of which is caused by the dilatation of the right heart. The gastric and intestinal catarrh caused by the engorgement, as well as the deficient deflux of lymph, especially chyle, from the thoracic duct into the subclavian vein, the emptying of which is likewise rendered more difficult, are the principal causes for the disturbances of nutrition which will not fail to appear in the course of time in emphysema.

Differential Diagnosis.—*The characteristic form of the thorax, the type of the respiration, above all the result of percussion, the diffuse unmovable pulmonary borders, the small area of cardiac dulness, the hypertrophy of the right ventricle with the intensification of the second pulmonary sound, and the consecutive engorgement symptoms* in conjunction with the other pathological signs just described, cause the diagnosis of alveolar emphysema to be made easily and with great precision. On the other hand, the diagnosis will become more difficult when it is a question of vicarious emphysema, to which I will refer later on. At the same time it cannot be denied that a wrong diagnosis may be made if pathological conditions which have a certain resemblance are not taken into consideration. Generally it is easy to exclude them.

Acute Inflation of the Lungs.—If a patient is examined for the first time, and nothing is known regarding the anamnesis, it is quite possible that *acute inflation of the lungs*, as it occurs in capillary bronchitis and bronchial asthma, will be confounded with emphysema. As it is a question here of rapidly rising and rapidly passing conditions, the subsequent course in one direction or the other will render the diagnosis clear. In the

individual case it is to be decided whether after an asthmatic attack has ceased, or according to the relaxation of the symptoms of bronchitis, the distended pulmonary borders, which are unchanged during respiration, are replaced by the normal limits, which is not the case in emphysema with its constant alveolar ectasis. Besides, the dilatation in acute inflation is not as considerable as in fully developed emphysema—i. e., the diffusion of the pulmonary borders never passes beyond the deepest inspiratory position of the normal lung (at the lateral lower pulmonary border percuss 3 to 4 cm.), while in emphysema a further dislocation posteriorly below, even to the second lumbar vertebra, is not uncommon.

Pneumothorax.—Emphysema is scarcely to be confounded, although the possibility of a mistake is actually contended, with *pneumothorax,* particularly because the latter is unilateral almost without exception, occurs acutely, and really has not anything in common with emphysema but the dilatation of the thorax and the diminished vocal fremitus with loud tympanitic percussion sounds.

Pulmo-excessivus.—On the other hand, not infrequently, the question arises, according to my experience, whether one has to deal with emphysema or an *unusually large normal* lung. This occurs principally in the examination of applicants for life insurance, etc., or in cases in which a chronic bronchial catarrh occurs in those large lungs. The results of percussion, the most important support of the diagnosis of emphysema, are chiefly the same in emphysema and pulmo-excessivus, extension of the pulmonary borders to the eighth, posteriorly to the twelfth rib, decrease or disappearance of the cardiac dulness and of the apex beat. However, few percussion sounds are sufficient for differentiation. In *pulmo-excessivus the motility of the lower pulmonary border is normal*—i. e., the lower border of the clear percussion is displaced two finger-breadths inferiorly on percussion over the right mamillary line in consequence of deep inspiratory movements. Besides, the second pulmonary sound is not accentuated. The feebleness and softness of the vesicular breathing as well as the signs of difficult breathing are absent, and, as a rule, no catarrhal râles can be heard. Even if bronchial râles are present, besides the extension of the pulmonary borders, emphysema may positively be excluded as soon as the lower lateral border of the lung shows the normal expansion of 3 to 4 cm.

Non-compensated Cardiac Defects.—Not infrequently I have had difficulty in the differentiation of emphysema, and that in its later stages, from a *mitral defect,* or especially from an *idiopathic hypertrophy of the heart in the stage of relaxation.* The characteristic form of the thorax not being always developed in emphysema, and as the signs of insufficiency of the heart, engorgement symptoms, especially development of *hydropericardium and hydrothorax,* also occur in emphysema, furthermore, systolic cardiac murmurs set in in the later stages of the emphysema, and, on the other hand, chronic bronchitis is found in both conditions, owing to the engorgement of the blood in the lungs, the diagnosis of emphysema is deprived of its best objective supports, for it is obvious that the hydrothorax prevents the exact determination of the pulmonary borders, and the cardiac borders appear diffused by the complicating hydropericardium. It is true, it should

be expected that a change in the position of the body will make the low position of the pulmonary borders easily demonstrable; however, even a physician who has done much examining will have learned that this manœuvre will in many cases not give the indubitable results, be it that the fluid of the hydrothorax is in part sacculated, or be it that the compressed lung is insufficiently filled with air on inspiration. In such a case it is best to administer digitalis, 5 to 6 times, 0.1 pulv. fol. digitalis per day. In the majority of cases, the disappearance of the hydropericardium and of the hydrothorax is brought about in this manner, with improvement of the activity of the heart, and now the diagnosis of emphysema will present no further serious difficulties.

Of course, both, lesions of the heart and emphysema, may be present simultaneously; for that both affections exclude one another has been proved to be incorrect. This holds good for idiopathic hypertrophy of the heart as well as for defects of the cardiac valves, and also, as will be specially enlarged upon, for tuberculosis, which in my experience is not infrequently associated with emphysema.

As easy as it is to diagnosticate well-marked emphysema, so difficult is it often to recognise *slight degrees of development* of the affection. It should be made a rule to refrain from any diagnosis—in fact, from a probable diagnosis of emphysema if the pulmonary borders are not situated low down, beyond the shadow of doubt, and, which is the main point, if they do not remain unchanged on deepest inspiration or are not displaced for at least one finger-breadth.

Vicarious Emphysema.—The diagnosis of *vicarious emphysema* requires special discussion. It develops in all instances in which single portions of the lung become affected, and now an increase of air pressure occurs in those parts which are still accessible to the entrance of air, both as well in inspiration as expiration, if cough, pressure movements, etc., play a part at the same time. Everything that compresses the lung and prevents its development may become the cause of this form of emphysema—pleuritis, pericarditis, scoliosis of the vertebral column, interstitial pneumonia, especially atrophy of the lungs after pleuritis, etc. The diagnosis is to be based on the ætiology in the first place; if one of the above-named pathological conditions prevails which leads to vicarious emphysema and mostly concerns one half of the thorax, attention should be paid to the healthy side. Not infrequently a displacement inferiorly of the lower border of the lung is then found, and, above all, in case the lung is atrophied and airless on one side an extension of the median border of the healthy lung, which is characterized by sonorous and low percussion sounds, beyond the centre of the sternum towards the affected side.

It is generally easy in such cases to demonstrate the displacement of the mediastinum and to follow the anterior median emphysematous border of the lung in its entire course by means of percussion. If vicarious emphysema develops on the affected side in the unobstructed areas of the lung, it may equalize the decrease which is caused by compression or atrophy of the lung, in so far as the borders of the affected lung eventually do not appear materially reduced. Vicarious emphysema almost always occurs in the anterior borders of the lungs during the death agony, if it is a question of patients in whom towards the end of life the lower posterior portions of the lung cannot be fully expanded, but inspiration still occurs with such energy that the anterior median borders are dilated vicariously; this may cause the cardiac dulness to become smaller, and thus the formation of this border emphysema will be indicated diagnostically.

Senile Emphysema.—Two other pathological conditions which injudiciously have also been named emphysema are to be strictly differentiated from the alveolar emphysema—the *interlobular* and *subpleural emphysema* and the *senile emphysema*. The latter affection (*atrophy of the lungs*), the same as vesicular pulmonary em-

physema, is accompanied by the disappearance of the interalveolar tissue with devastation of the capillaries and destruction of the alveoli; however, dilatation of the lungs is not present. On the contrary, the volume of the lungs is small, the position of the diaphragm high, the borders of the lung narrow, movable on inspiration, the cardiac dulness is isolated to a greater extent, the spinal column shows the kyphotic senile curvature, the thorax laterally appears flattened—conditions which are the opposite of those in vesicular emphysema. It is therefore impossible to confound these conditions. The affection corresponds to alveolar emphysema only in the dyspnœa caused by the decreased respiratory surface and the cyanosis, which is easy to explain by the destruction of large areas of pulmonary capillaries. The dilatation and hypertrophy of the right heart, which is usually present, *is absent* in this affection, obviously because an involution of the heart advances equally with the involution of the lungs.

Subpleural and Mediastinal Emphysema.—In the so-called *interlobular* and *subpleural emphysema* air enters the interlobular and subpleural connective tissue owing to tearing of the alveolar walls. From the hilus of the lungs the air enters the *mediastinal connective tissue* and the subcutaneous tissue of the throat, trunk, etc. Interlobular emphysema is generally rare, and, as a rule, offers more pathological-anatomical than clinical interest. It is caused mainly by strong pressure movements, violent attacks of coughing, also in consequence of contusions of the thorax and lungs, ulcerating processes of the lungs, etc.

The following symptoms point to the development of a subpleural respectively mediastinal emphysema: cutaneous emphysema in the throat and trunk, disappearance of the cardiac dulness, but, instead, tympanitic percussion sounds in the region of the heart, which, in contradistinction to percussion in pneumopericardium, do not change their position upon change of the posture of the patient. This manifestation is caused by the *accumulation of air in the anterior mediastinum*, upon which, when the accumulation of air assumes considerable dimensions, the visible pulsation of the heart disappears and the intercostal spaces become indistinct. Besides, a diagnostic symptom occurs on auscultation, a fine crepitation, which is synchronous with the action of the heart, the importance and constancy of which has lately been pointed out by F. Müller. If there exists subpleural emphysema besides the mediastinal, it appears that the lower border of the lung is forced down thereby—that is, the emphysematous dilatation of the subpleural tissue causes a descent of the clear percussion sound towards the costal arch. The respiratory sounds appear weakened, or indistinct if previously bronchial, in those parts of the lung in which the condition is present, above which the subpleural emphysema is established in the form of smaller or larger air vesicles. Besides the above-named symptoms, choking attacks and swelling of the jugular veins have been observed as a consequence of the compression of the heart and of the large vessels in the thorax. Accordingly, the diagnosis of subpleural, respectively mediastinal, emphysema can be made with some certainty. However, only when the above symptoms are very pronounced, and if an ætiological factor for the origin of a subpleural emphysema is undoubtedly present.

ŒDEMA OF THE LUNGS

Symptoms and Signs.—The diagnosis of œdema of the lungs—i. e., the exudation of fluid blood into the alveoli—is not difficult, because the symptoms which announce its presence are very pronounced. The exudation of fluid into the alveoli may be so abundant that dulness, bronchial breathing, increased pectoral fremitus, the signs of complete absence of air from the alveoli occur. However, it is rare that such a degree in the development in pulmonary œdema can be determined. In by far the majority of the cases accumulation of blood is limited, and, in respect to percussion, no change at all can be demonstrated, or, upon pronounced serous saturation of the alveolar wall, and of the interstitial tissue, a change of the normal pulmonary note

into a tympanitic sound may be elicited. Auscultation proves the presence of large quantities of fluid in the air cells by the occurrence of *moist* râles, which are fine or coarse, according to the diffusion of the fluid into the smaller or larger bronchi. *The sputum is particularly characteristic,* it is copious, frothy, thin, fluid, yellowish or pink coloured, with a plentiful admixture of blood corpuscles, of a dark-red tinge, or, if the admixture is plentiful the sputum is prune-juice-like, as is especially observed in inflammatory œdema in the course of croupous pneumonia. It is obvious that the saturation of the alveoli with blood impedes the respiration. Dyspnœa, cyanosis, and the signs of carbonic-acid intoxication which refer to the nervous system, are, therefore, certain consequences in severer grades of pulmonary œdema.

Diagnosis regarding Ætiology and Pathogenesis.—The diagnosis of pulmonary œdema, according to the above, is generally certain so long as it is not a question of very slight degrees of the affection, or the œdema does not develop until, in the death agony, the expectoration stops, and the tracheal râles prevent the finer differentiation of the moist râles upon auscultation. However, the scientifically trained diagnostician cannot possibly be satisfied with the simple determination of the presence of an œdema of the lungs. It is his duty to attempt to *decide the origin of the pulmonary œdema* in the individual case. This question has as yet not been fully determined. For a time it appeared as if the genesis of pulmonary œdema were sufficiently elucidated by the epoch-making paper of Cohnheim and Welch. According to them, unilateral relaxation of the left ventricle was to be the proper efficient cause of pulmonary œdema, inasmuch as an engorgement in the pulmonary circulation occurs with exudation of blood into the lungs on continued action of the right heart. However, more recent experimental and particularly clinical experiences do not speak in favour of the general applicability of the Welch theory of the origin of pulmonary œdema in man. So much is certain in my opinion, that the process is not a uniform one, that rather various conditions may lead to transudation of fluid blood into the alveoli, and, accordingly, various forms of pulmonary œdema must be assumed. Two principal forms of pulmonary œdema must under all circumstances be differentiated owing to the facts which are experimentally and clinically demonstrated: *inflammatory pulmonary œdema* and the *œdema due to engorgement.*

Inflammatory Pulmonary Œdema.—As to the first form, *inflammatory pulmonary œdema*, it is found in inflammatory processes of the lungs as an expression of a moderate degree of inflammation, because in contradistinction to the full development of the process as a rule no abundant corpuscular elements exude from the vessels, and the inflammatory exudation is often limited to the exudation of blood.

[illegible] in the *neighbourhood* of pronounced pneumonias and inflammatory [illegible] processes in the lungs tumours, infarcts, etc. The œdematous infiltration of the lungs under these circumstances is, as Cohnheim expressed it so correctly, [illegible] of the inflammatory process, which has its centre in the [illegible] portions of the lung. In other cases the serous saturation of the [illegible] product of the inflammatory process in the lungs; it is a ques-

tion here of the *initial stages* of an inflammation which leads *rapidly* either to cure or to death (in which, previous to the abundant extravasation of blood corpuscles, mostly fluid only passes from the vessels), or that the inflammatory infection is so insignificant that it ends with the transudation of fluid. The last-named cases, *serous pneumonias*, are rare, it is true, but there can be no doubt as to their occurrence.

For the diagnosis of inflammatory œdema it is above all important that this variety of pulmonary œdema takes its course with fever and the usually strong pulse, and that in the majority of cases the main part of the inflammation is undoubtedly pre-eminent by distinct pneumonic signs besides the pulmonary œdema. Recently Sahli has proved by autopsy findings that this inflammatory character of œdema is much more frequent than has been so far assumed.

Stasis Œdema.—The second form of pulmonary œdema, *engorgement œdema,* is the result of circulatory disturbances in the lungs, which, according to the origin of the engorgement, and œdematous transudations at other places, is characterized by the fact that the venous deflux from the lungs is considerably impeded, while the arterial influx goes on, and thus a congestion of the capillaries and a transudation of blood from them take place.

If *both* ventricles of the heart become paretic simultaneously, the necessary consequence would be a slowing of the circulation in the lungs, but never a pulmonary œdema—i. e., a transudation of fluid from the capillaries. If the latter is to occur, the arterial influx in the pulmonary vascular system must not diminish too much, for the amount of the latter is as characteristic of the origin of œdema as the other factor, viz., the resistance in the venous system. Welch, therefore, was perfectly correct when, to produce experimental pulmonary œdema, he tried to cause unilateral paralysis of the left ventricle with intact function of the right heart, and he actually succeeded in producing in a rabbit artificial pulmonary œdema by crushing the left ventricle. However, it cannot be denied that certain difficulties exist in explaining the occurrence of pulmonary œdema at the bedside with this theory. It is undoubtedly the case that the occurrence of a *strong tense pulse,* as is actually observed sometimes, although not always, during the development of œdema, speaks against the validity of Welch's theory in such cases; and, furthermore, at the first glance it is difficult to understand how a high-grade paralysis of the left ventricle, corresponding with the experiment (which may occur in the rabbit's heart, but in the dog's heart cannot be produced artificially), is to occur *isolatedly* with intact action of the right ventricle in the human heart. But it must not be forgotten that, according to experimental experience, the deficient oxygen supply appears to act unequally upon both sides of the heart, exerts, especially upon the left ventricle, a more paralyzing influence, and, furthermore that, upon beginning relaxation of the entire heart, the deficient filling of the arteries creates an abnormal resistance posteriorly in the left auricle and in the pulmonary veins. This causes an increased pressure, not only in the last-named districts, but with the deficient tone of the pulmonary arteries, also in the capillaries, and in the pulmonary arterial circulation. But to this condition the right heart reacts with greater activity, so that it continues to contract, relatively vigorously, while the left has already become relaxed. It is therefore conceivable that pulmonary œdema is apt to develop in conditions in which the left ventricle has been overtaxed unilaterally (as, for instance, in atrophy of the kidneys), as soon as the increased activity of the left ventricle relaxes; but the same must also be the case in general cardiac insufficiency as a result of affections of the heart, and especially also in the death agony. It does not appear strange to me that it is not generally the last occurrence in the last-named conditions. The occurrence of œdema in such cases depends solely upon the ability of the right ventricle to react upon the begin-

ning engorgement. If the right ventricle (as is mostly the case in the final agony) is not able any longer to contract fully, no œdema will occur, because, as we have just stated, to produce the same not only resistance in the venous system must be present *but also the arterial influx must not diminish materially. Vice versa* on the occurrence of resistance in the pulmonary circulation the compensating power of the right ventricle may assume such dimensions in other cases that the resistance is overcome, and the blood which now enters the left ventricle in greater amount stimulates the latter to greater activity; therefore, œdema does not occur here either.

But not only paresis but *spasm of the heart* may increase, according to the reasons enlarged upon in cardiac asthma, the pressure in the pulmonary arteries and capillaries and produce pulmonary œdema. Grossmann has recently proved this sufficiently by experiments, inasmuch as spasms of the cardiac musculature caused artificially by injections of muscarine into animals, occurred in a higher degree in the left ventricle than in the right, and were accompanied by an increased pressure in the left auricle and the pulmonary artery and decreased pressure in the aorta. Clinical experience also favours the view that in some cases of rapidly occurring pulmonary œdema the cause is to be looked for in spasm of the heart. I have seen pulmonary œdema supervene upon angina pectoris which alternated with muscular spasms in other parts of the body.

Differential Diagnosis of Stasis Œdema.—In contradistinction to inflammatory pulmonary œdema, *engorgement œdema can be diagnosticated when fever is absent. Furthermore, the second pulmonary sound must be accentuated under all circumstances, and the pulse small.* The engorgement of pulmonary œdema, however, can in the individual case be decided upon with a degree of certainty from the anamnestic data.

"Hydræmic" Pulmonary Œdema.—Until recently *hydræmia* also was considered as a cause of pulmonary œdema. This was supposed to explain its relatively frequent occurrence in renal disease, and also the occurrence of pulmonary œdema in cachectic conditions of various kinds. However, the theory of hydræmic dropsy could not hold its own on experimental investigation, according to which theory the diffusibility of the blood, which is deficient in albumin, was to explain the occurrence of the transudation. For Cohnheim has plainly shown that hydræmia *does not,* as such, produce œdema, but that in consequence of long-lasting hydræmia the vascular walls become more permeable and the energy of the heart appears weakened. If, therefore, hydræmia, as such, does not produce pulmonary œdema, yet it produces an undoubted predisposition to it—i. e., all the above-mentioned causes of pulmonary œdema will be more apt to cause the occurrence of the pulmonary œdema in hydræmics than in other patients. This holds good for engorgement œdema, as well as for the inflammatory form, because, owing to the hydræmic injury to the vascular walls, inflammatory irritations which affect the lungs are less easily overcome, and cause a far greater transudation of blood than under ordinary circumstances.

INFLAMMATORY ALVEOLAR INFILTRATION—PNEUMONIA

FIBRINOUS—CROUPOUS PNEUMONIA

As is well known, "croupous" pneumonia is one of the diseases which is very easy of diagnosis. Especially is this so when the lung is considerably infiltrated. But since we know that croupous pneumonia is an infectious disease, the meaning of infiltration of the lung and pneumonia is not the same. It is necessary, therefore, in discussing the diagnosis of pneumonia, to separate cases in which the infiltration is pronounced and demonstrable by percussion and auscultation from those in which the localization of the pneumonic process in the lungs is not so pronounced on account of other pathological manifestations. The former is the case in the majority of instances, and, therefore, we will deal with this class for the present, while the latter will be discussed later on.

Sputum.—The most important symptom is the expectoration of the *rusty sputum*. Only in rare cases is there *no* sputum expectorated. The sputum is characteristic; it is viscid, adhering tenaciously to the receptacle into which it is expectorated, glassy and translucent; the blood and mucous particles are intimately intermingled. The colour of the sputum is yellowish or brownish-red. The sputum when placed in water shows the dichomatous ramifications of the finer bronchi. Later, during the period of resolution, the colour of the sputum is more yellowish, and rarely, if crisis is delayed, it becomes green. When the exudate is composed of inflammatory œdematous fluid the suspicious prune-juice expectoration appears. When the process becomes gangrenous the expectoration has a putrid odour. The microscopical examination of the expectoration shows red blood corpuscles, which are swollen and degenerated, also mucus, epithelial cells, and *micro-organisms*.

Micro-Organisms in Sputum.—Regarding the micro-organisms, it is well known that in 1883 Friedländer considered capsular bacilli as the specific cause of pneumonia. However, it soon became evident that this organism was of ætiological significance in but very few cases of primary and secondary pneumonia. In the great majority of cases the sputum contains the pneumococcus lanceolatus capsulatus, which was discovered in 1884 by A. Fränkel. This is the actual causative factor of fibrinous pneumonia, and this has since been demonstrated by many observers, particularly by Weichselbaum.

Fränkel's pneumococcus appears, as a rule, in pairs (the diplococcus pneumoniæ), tapering towards the polar ends (the diplococcus lanceolatus), and is surrounded by an oval capsule. It is easily stained by aniline dyes, and distinctly stained by Gram's method in contradistinction to Friedländer's pneumobacillus, which does not stain by Gram's method. According to experience, Fränkel's pneumococci are found in greater abundance and are more virulent the more recent the inflammation in that portion of the lung from which they emanate. Further investigations have shown that pneumococci are *by no means found alone* in croupous pneumonia. They can be demonstrated in pleurisy, endocarditis (compare p. 7), peritonitis, cerebro-spinal meningitis, etc.—that is, in all the various inflammations that are of an infectious character. Fränkel's pneumococcus is therefore considered to be a widely disseminated organism, and is a causative factor in many inflammations (mostly purulent) in the various organs of the human body. Upon its entrance into the lungs it

causes pneumonia, and, although not exclusively so, but certainly in most cases, is the cause of pneumonia. Although it is without doubt that the pneumococcus is found in the sputum of the healthy, yet it is no proof against its virulent character, which is, on the contrary, enormous, as is evidenced by the inoculation of animals with the bacteria. This fact only shows that these extremely virulent bacteria are not able to remain in the lungs under ordinary circumstances and multiply; but to do so, certain auxiliary factors are requisite, and it will be the object of future investigations to determine this. Recently pneumococci have also been demonstrated in the *blood* of some patients suffering from pneumonia. These were cases which ran a fatal course, and therefore the supposition is probably justifiable that a (pneumococcic) septicæmia developed which caused or favoured the fatal termination (see septicopyæmia).

Other micro-organisms have also been found to be causative factors of pneumonia—namely, *streptococci* and *staphylococci*, and various *bacilli*. As a rule, only one of the above-named organisms is found in pneumonic areas; sometimes, however, several may be present (mixed infection). According to the organism causing the infection, it could be presumed that the clinical picture would vary; in fact, the distinctly streptococcic pneumonias generally do not occur with lobar but with *cellular lobular* infiltrations (Finkler), but there are exceptions to this rule. It is of greater importance that in pneumonia due to this cause resolution is, as a rule, greatly delayed, which apparently also holds good in those cases in which there is a mixed infection of streptococci and diplococci.

The diagnosis of pneumonia can, as a rule, be made without hesitation upon the above-mentioned appearance of the sputum, especially its tinction, its viscidity, etc., even in cases in which no physically demonstrable change in the lung is present. However, the *infiltration* of the lungs can be easily and positively demonstrated in by far the majority of the cases by means of physical examination, sometimes even on the first day.

Slight Infiltration of the Lung.—Only in very rare cases, in which the infiltration is centrally located, or remains restricted to a small portion of the lungs (less than 5 cm. in circumference), days are spent in vain looking for the characteristic percussory and auscultatory changes. Under such circumstances the seat of the pneumonic infiltration can be best discovered by *comparative auscultation of the voice at different parts of the thorax*. I have often succeeded, if there was no other trace of dulness or bronchial breathing, in demonstrating by the determination of the beginning *bronchophony* the location at which the characteristic signs of infiltration become evident within the course of a few days. Regarding the seat of pneumonia, I wish to lay particular stress upon the fact that, in my experience, it frequently develops in the axilla, and that here bronchophony first becomes audible. [The *most frequent seat* of the pneumonic infiltration is the right base, next in frequency is the left base, both bases, and the apices less frequently.]

Percussion.—Upon further development of the infiltrate the following physical signs are found: *tympanitic percussion sound* during the stage of engorgement and resolution; *dulness*, which may be absolute, accompanied, however, by less resistance than when there is an exudation of fluid into the pleural cavity. Exceptionally a *cracked-pot sound* is obtained over the infiltrated portions of the lung. This is due to the fact that upon percussion the air escapes from the relaxed tissues, or the bronchi, with a hissing sound. *A change in the height of the pitch upon opening and closing the mouth* is rarely found. In the latter case it is almost always the result of infiltration into the upper lobes, because the tympanitic sound over the principal bronchus is audible through the infiltrated pulmonary tissue, and becomes higher upon opening and lower upon closing the mouth

(Williams's tracheal sounds). The change in the pitch of the sound has also been observed in infiltration of the lower lobes; in some cases a note of a metallic quality is obtained, which cannot be satisfactorily explained. *The limits of the dulness* are sometimes distinct, and occasionally not, according to the anatomical condition of the infiltrated area. Sometimes it corresponds almost exactly with the outlines of the respective lobe of the lung affected. If the left lower lobe is the seat of the infiltration, the tympanitic gastric sound may be transmitted through the area of consolidation, and may lead to an error in diagnosis. On the other hand, the dulness rarely extends beyond the upper border of the semilunar space [Traube] (which generally corresponds in extent to that part of the pleural sac, the left pleural sinus, which is anterior to the border of the spleen) in contradistinction to the dulness caused by left-sided pleuritic exudations.

Auscultation.—On *auscultation*, during the beginning of infiltration and in the stage of resolution, crepitant râles are heard. (*Crepitatio indux respectively redux*). It is of importance that crepitant râles occur almost exclusively during inspiration; only exceptionally was I able to determine them during expiration. The *respiratory murmur is bronchial* at the height of the stage of infiltration, generally loudest over the localities of greatest dulness—owing to the greater reflection of the infiltrated walls of the bronchi, which thus have advanced relatively nearer the thoracic wall. Eventually the râles may have a ringing sound.

Vocal Fremitus.—*The examination of the voice by palpation* shows *increased vocal fremitus*, however, not at all as regularly as is generally assumed. The increase of vocal fremitus is absent if the main bronchus is occluded by secretion, or if, in very marked infiltration, the tension of the thorax becomes excessive, and the conditions for the transmission of the sound waves thus become more unfavourable. Auscultation of the voice shows *bronchophony*, very rarely *ægophony*; the latter when the bronchi accidentally become so compressed by the hepatized and distended lung, so that the sound waves are sometimes able to pass the closely approximated walls of the bronchi, and sometimes cannot do so.

Inspection.—Upon *inspection* the relaxation of the affected half of the thorax during respiration, and the increase of the respiratory frequency, appears remarkable. *Mensuration* shows an increase in circumference in the affected half of the thorax from 1 to 2 cm., but the increased circumference is materially smaller than in a massive pleural exudate.

Other Symptoms of Pneumonia.—All the other symptoms peculiar to pneumonia, except the above-named, are of minor importance: the *fever* commencing with chills or vomiting, the *stitches in the side*, the *accelerated respiration*, etc. Special stress is to be laid upon the following: the number of respirations is almost always more than 30 to the minute, sometimes even 100 and above. It is generally influenced but little by the fever (Gerhardt), and is in great disproportion to the pulse, so that instead of the usual ratio of 1 to 5, 1 respiration occurs in the time of 3 or only 2 pulse beats. The *fever*, which is almost never absent, but of a considerable height, commences, as a rule, with a single *chill*. Repeated chills are rare. Only exceptionally there is no indication of the chill. The temperature rises quickly to 104° F. to 105° F. and above, and falls by crisis (in the majority of cases during the night)

with perspiration, very rarely on the first or second day, most frequently towards the end of the first week. The crisis is sometimes preceded by an excessive rise of temperature (precritical rise). The fall of temperature by crisis is usually followed for some time by subnormal temperature; in other cases the temperature, which has become normal, is interrupted by another rise, or by repeated exacerbations (*pneumonia with intermittent fever*). This intermittent course of the fever is caused either by the abrupt progress of the inflammation or, in malarial districts, by the infection with malarial toxines—i. e., by the effect of the plasmodia. The condition of the *pulse*, especially its frequency, fluctuates very much in the individual case; but on the whole, with a temperature of 105° F. the frequency of the pulse is generally about 120—in contradistinction to enteric fever, in which the pulse is relatively retarded. Comparatively, a considerable increase in the frequency of the pulse occurs with increasing weakness of the heart. The pulse becomes soft, small, and irregular. The weakness of the heart generally develops quite gradually; rarely it occurs suddenly, especially during the time of crisis we should be prepared for collapse. The *cough* is superficial, painful, occurs in *short* paroxysms, and has thus something characteristic in comparison with other diseases which are accompanied by cough. However, it may be entirely absent, especially in the aged [and in the alcoholic]. Upon the *skin*, *herpes*, especially herpes labialis, are often observed, besides the flushed skin and eventually cyanosis. Herpes is of diagnostic significance, in so far as it does not occur as regularly in any other infectious disease. Not infrequently *jaundice* is found; this is of significance in a diagnostic respect, because icterus does not accompany any other febrile disease as frequently as pneumonia. Therefore, supported by long years of experience, I can only give the rule, in case icterus is associated with high fever, always to examine the lungs in the first place, and to exclude pneumonia as the cause of the icterus only when the examination of the chest gives an absolutely negative result, and if the characteristic appearance of the sputum is lacking.

The *spleen* corresponding to the infectious nature of croupous pneumonia may become *enlarged*. This, however, I maintain, according to my experience, contrary to the statements of others, is not of frequent occurrence. It is true, I assume an enlargement of the spleen to be positive only when the organ is palpable, because the spleen, except in a few rare cases, can be palpated if it is considerably enlarged, and if a relaxation of the abdominal walls is brought about to as great an extent as possible, and, on the other hand, percussion of the spleen yields a doubtful result in many cases. It has been observed frequently that the enlargement of the organ increases with or after the crisis. The cause of the *post-critical enlargement of the spleen*, which was first found by Gerhardt, but according to my experience is infrequent, should perhaps be looked for in a deposition in the spleen of morphological elements of the blood which were destroyed during the course of the disease. Therefore the splenic enlargement is to be considered as "spodogenous"; an early defervescence appears to favour the occurrence of post-critical enlargement of the spleen (Matthes).

The *urine* does not show changes which are characteristic of pneumonia, although in this disease, particularly, the most numerous urine analyses have been made. A greater *excretion of urates*, the *sedimentum lateritium*, especially during the crisis, is noticeable above all, which is caused by various factors—namely, the great diaphoresis, and the great destruction of leucocytes during and shortly after the crisis, because in this instance abundant quantities of uric acid are formed from the nuclein of the leucocytes especially. The excretion of urea during resolution is remarkable, as is also the alkaline reaction of the urine one or two days after the crisis (F. Pick). Comparatively frequently, a considerable excretion of albumoses has likewise been demonstrated, as well as a *decrease of the urinary chlorides* during the height of the fever and their reappearance with the termination of the inflammation. *Albuminuria* can be demonstrated relatively frequently during the course of the disease (in about 50 per cent of the cases), which is oftener than in any other infectious disease. In my opinion this is almost always caused by irritation of the kidneys produced by the pneumonic toxines, which are more or less affected by the same. Accordingly, in some of the cases a simple transitory albuminuria occurs, in others the elimination of tube casts and the appearance of hæmaturia; in short, in these cases the complete

picture of acute nephritis develops, the symptoms of which may eventually exist for months after the termination of the pneumonia. The albuminuria is the result of cardiac feebleness only in rare instances.

An *acute leucocytosis* is demonstrable in the *blood* of patients suffering from pneumonia (more pronounced than in any other infectious disease)—i. e., the white blood cells appear considerably increased in the peripheral circulation on microscopical examination. Besides, a considerable *oligæmia* develops in these cases, because the quantity of the total blood decreases according to the area of pneumonic infiltration. *Pneumococci* can be demonstrated in the blood in some cases, as stated above.

The diagnosis of fully developed pneumonia is very easy. However, daily experience at the bedside teaches that cases occur of well-marked pneumonia which present great diagnostic difficulties during their entire course, and that doubts may arise, at least during the first days, regarding the presence of inflammation of the lungs in a considerable number of cases. This happens particularly when the sputum does not contain blood for some time, which occurs but rarely, or when the general infection predominates in the pathological picture (for instance, in drunkards in the form of delirium tremens), or when the pneumonic infiltration is centrally located and does not advance towards the periphery. In such cases the diagnosis is actually impossible; sometimes it may at least be possible to make a provisional diagnosis upon the character of the cough, the increase in the respiratory frequency, the herpes, the initial chill, the high fever, and the marked critical fall of the temperature.

Abortive Types of Pneumonia.—In pneumonia as well as in other infectious diseases there occur abortive, rudimentary, and masked forms. Kühn has called attention to the latter form. These are to be taken into consideration, if during an *epidemic of pneumonia* febrile affections occur in the course of which neither bloody sputum nor changes in the lung occur, but in which one of the accessory symptoms becomes prominent, as herpes, initial chills followed by fever, which may eventually become recurrent, great exhaustion, which contrasts with the slight objective demonstrable symptoms, etc. In some cases the brain is especially affected by the pneumonic toxines and reacts by giving rise to epileptic or apoplectic attacks accompanied by fever, "typhoid," and meningitic symptoms. Kühn has also observed neuralgias—regular masked pneumonia—caused by pneumonic infection. I again emphasize the fact that the diagnosis of such rudimentary and masked pneumonias can only be made provisionally if any other explanation of the pathological symptoms can be excluded, and the simultaneous epidemic occurrence of pneumonia is beyond doubt.

Differential Diagnosis—Pneumonia and Pleurisy.—*If the physical examination gives a positive result, but the sputum does not contain any blood* (if there is any sputum at all), as a rule, pneumonia and pleurisy *are to be differentiated.* Common to both processes are: dulness, bronchial breathing, diminished vocal fremitus, which is often present in pneumonia, bronchophony, and ægophony, which is very rare in pneumonia, and other signs.

The following symptoms in particular are indicative of *pneumonia*: lesser resistance of the dulness, intensification of vocal fremitus, the presence of bronchophony and bronchial breathing, especially in the region of the *lower* portions of the dull area, the absence of dulness in the semi-

lunar space and the absence of displacement of the adjacent organs, the crepitant râle (which is found very rarely and only in pleurisy in the retracted portions of the lung which are situated above the exudate), the initial chill, the crisis, and the usually rapid disappearance of the dulness. The question whether a pleural exudate or a pneumonic infiltration prevails is of daily occurrence at the bedside, so that we will have to refer again to the differential diagnosis of these diseases when discussing the diagnosis of pleurisy (see p. 159).

Hæmorrhagic Infarct.—*If dulness can be demonstrated and if the sputum contains blood, hæmorrhagic infarct* and *pulmonary œdema* are to be considered, especially in regard to the diagnosis. The confounding of pneumonia with *pulmonary infarct* can be avoided if the usually afebrile course of this disease is observed, as well as the less thorough admixture of blood in the sputum, and, above all, the source of the embolism. Common to both processes are the signs of consolidation of the pulmonary tissue, the bronchial breathing, metallic râles, etc. Both processes are generally preceded with a chill.

Œdema of the Lungs.—It is less easy to confound pneumonia and *pulmonary œdema,* because consolidation with its sequelæ occurs only exceptionally in the latter, and then bilaterally, the crepitant râle is of less uniform character, and the sputum though bloody is thin and frothy. However, it must not be forgotten that not rarely both processes may be combined. Either an engorgement œdema develops, owing to the deficient oxygen supply and the weakened action of the heart, or an inflammatory œdema occurs in the neighbourhood of the pneumonic infiltration. There exists, besides, as stated before, a serous pneumonia in which the exudate is essentially thin (see p. 118).

Tuberculous and Pneumonic Infiltration.—*Phthisic processes, which are accompanied with hæmoptysis and infiltration,* may simulate croupous pneumonia, especially when the previous course of the disease is not known, and the tubercular consolidation progresses rapidly. In these cases, the observation of the characteristic viscid, rusty condition of the pneumonic sputum, above all, protects from error; besides, tubercle bacilli may be demonstrated in the sputum, and also the *ensemble* of the symptoms of pulmonary phthisis. The diagnosis might be difficult in rare cases, in which an acute croupous infiltration supervenes upon the tubercular disease, and the former complicates the local symptoms of pulmonary phthisis.

The differential diagnosis between croupous pneumonia *and other forms of pulmonary inflammation* will be best explained when discussing the latter.

BRONCHO-PNEUMONIA—CATARRHAL PNEUMONIA

Ætiological Criteria of Value in Diagnosis.—Catarrhal pneumonia can be distinctly separated clinically from croupous pneumonia, less by the physical signs than by its typical origin and its course. It develops from bronchitis when the latter descends, generally by means of atelectasis or by aspiration of the secretions which cause the inflammation. It may also be produced by substances which accidentally gain entrance into the respiratory tract (deglutition, foreign-body pneumonia). Particulary *infectious* bronchial catarrhs in the course of influenza, measles, whooping-cough, diphtheria, scarlatina, enteric fever, and others, frequently develop into broncho-

pneumonia. In at least 50 per cent of the cases of the latter affection the inflammation is caused by Fränkel's diplococcus, the same as in fibrinous pneumonia. The smallness of the bronchi and the greater sensibility of the alveolar walls and their epithelial covering in *infantile* lungs are the reasons why this form of pneumonia occurs particularly in children. On the other hand, the *senile* are predisposed to catarrhal pneumonia because the reflex excitability of the mucous membrane of the respiratory tract diminishes in old individuals, thus facilitating the occlusion of the bronchi by secretion and the downward progress of the inflammation. All these factors should be taken into account when considering the variety of pneumonia, because, as a rule, it is impossible to make a diagnosis from the physical signs alone.

Results of Physical Examination.—For a positive diagnosis it is necessary under all circumstances that the signs of consolidation be present. In lobular affections, a circumscribed *relative dulness, which is usually bilateral, radiating along the spinal column,* or at least tympanitic percussion sounds are to be expected. Besides crepitant râles, bronchophony and bronchial breathing, metallic râles, and increased pectoral fremitus over the consolidated portions of the lung, can be determined. However, these symptoms may be absent in many cases, but in spite of this we are often compelled, in order to arrive at a correct conclusion in the individual case, especially as to its diagnosis, to be satisfied with a probable diagnosis. *The latter is permissible if the body temperature reaches or exceeds 104° F. with a demonstrable bronchitis,* which does not, as a rule, occur in simple bronchitis. Furthermore, *if severe pain is complained of during the cough, and if there is a disproportion between the degree of bronchitis and the cyanosis and high respiratory frequency.*

Differential Diagnosis.—From this it is obvious that the diagnosis of catarrhal pneumonia is often very difficult; that frequently we succeed in making but a provisional diagnosis, and it is nearly always necessary to decide the question by differential diagnosis.

Croupous Pneumonia.—If it is a question of *croupous or catarrhal pneumonia,* bilateral symmetrical dulness in the posterior inferior portion of the thorax is in favour of the croupous form, as is also a short, critical course of the fever; while in catarrhal pneumonia there is generally a prolonged fever which terminates by lysis. Croupous pneumonia is further characterized by the fact that it generally attacks persons in apparently the best of health in the form of an acutely occurring infectious disease; catarrhal pneumonia, on the other hand, is preceded by bronchitis, which has developed in the majority of cases after a previously existing infectious disease. The appearance of the sputum is generally without value from a diagnostic standpoint, because it is usually absent in the pneumonias of children and in the aged; if it is present, it is mostly muco-purulent in catarrhal pneumonia, rusty and containing blood in croupous pneumonia. Exceptions occur in both these instances.

Miliary Tuberculosis.—It is more difficult to *differentiate catarrhal pneumonia from acute miliary tuberculosis.* The differential diagnosis is impossible so long as no dulness can be ascertained in pneumonia. Dysp-

nœa, cyanosis, diffuse capillary bronchitis, occur in both. The localization of the process in the apices of the lungs, the presence of tubercle bacilli in the sputum (which is very rare in acute miliary tuberculosis), and the demonstration of chorioidal tubercles in the eye facilitate the diagnosis.

Atelectasis.—The pulmonary atelectasis which generally precedes catarrhal pneumonia differs from the latter by its transitory character, inasmuch as in atelectasis dulness and the auscultatory signs connected with it are modified by the position of the patient, or may be made to disappear entirely. Pulmonary atelectasis in itself does not cause fever. If it is observed during this affection it is caused by an accompanying bronchitis. If the temperature is 104° F. and above broncho-pneumonia is to be assumed in a doubtful case.

The differentiation of catarrhal pneumonia from pleurisy, as well as the supposition of a pleuritis complicating the former, require the same diagnostic considerations that have been described when discussing the differential diagnosis of croupous pneumonia and pleurisy.

INTERSTITIAL PNEUMONIA

The diagnosis of interstitial pneumonia is almost always of minor clinical interest. Usually it is to be considered only as a complication of other pulmonary diseases. It is an accompanying condition of many inflammatory processes of the respiratory tract. It is seen in bronchitis and pleurisy, ulcerating, gangrenous, caseous processes and new growths in the lung. More rarely it is seen in croupous and catarrhal pneumonia affecting only the surface of the alveolar walls. Interstitial pneumonia occurs as a more independent disease in consequence of the inhalation of iron, coal, or stone dust, etc., and in the course of syphilis. This specification of the ætiological conditions of interstitial pneumonia shows that its diagnosis always requires the consideration of its cause, and the determination of other affections of the respiratory tract occurring simultaneously.

Atrophy of the Lungs—Consequences.—The characteristic feature of interstitial pneumonia is *atrophy of the lungs* affecting smaller or larger portions. If the latter is the case it is not difficult to determine it. The thorax is drawn in at those places where the lungs have contracted. If the atrophy affects larger portions of one lung the retraction of the thorax becomes manifest by the approximation of the ribs, curvature of the spine, the low position of the shoulder, and the projection of the scapulæ. Respiration is impeded on the affected side, the circumference of one half of the thorax appears considerably reduced in comparison with the other. The capacity of the lungs is diminished. In atrophy of the left lung the changes in the cardiac region are most noticeable: displacement of the apex beat to the left and to a higher intercostal space, pulsation in the cardiac region more diffuse owing to the pulmonary tissue normally over the heart and large vessels having retracted. In the second left intercostal space, external to the origin of the pulmonary artery, a systolic bulging and

diastolic thrill of short duration become visible and palpable. The latter is an expression of the closure of the pulmonary valves, which appears on auscultation as an accentuated valve sound in comparison to the second aortic sound. Of course, the picture is completely changed in unilateral atrophy of the right lung, as in this case the heart may be displaced to the right, and the cardiac dulness appear diminished owing to emphysema of the left lung.

Percussion shows more or less pronounced dulness according to the degree of hyperplasia of the connective tissue. If the atrophy is limited to the apices of the lung, or very much developed at this point, they will also be shrunken. Besides, symptoms of (phthisic and bronchiectatic) pulmonary cavities in the upper lobe can usually be determined. If the atrophy affects the lower portions of the lungs, the lower borders, as well as the borders of the abdominal organs adjacent to the diaphragm (liver, stomach, and spleen), are displaced upward. The pulmonary borders thus displaced are movable on respiration unless pleuritic adhesions accompany the atrophic process, as is usually the case. Compensatory emphysema develops in the non-atrophied lung. The unaffected lung reaches into the area occupied by the atrophied pulmonic tissue, due to compensatory emphysema; it is possible, as a rule, to determine the borders of both lungs by percussion on account of the unilateral infiltration and atrophy—that is, by means of linear percussion, a displacement of the anterior mediastinum may be determined, the anterior median pulmonary border of the non-atrophied emphysematous lung may be differentiated from the atrophied lung by the clear percussion sound of the former which reaches to the opposite sternal border and even beyond it.

This mediastinal displacement of the pulmonary borders is particularly marked in left-sided atrophy of the lungs.

On *auscultation,* the following signs, which are characteristic of the disappearance of air from the alveoli, are found: feeble respiration, bronchial breathing, bronchophony, ringing râles, and in some cases, pronounced evidences of cavity (metallic note, etc.). The vocal fremitus is intensified or decreased according to the conditions which hold good for the other processes of infiltration. The sputum is not characteristic, with the exception that in interstitial pneumonias due to inhalation of dust, various kinds of dust may be found. This is, of course, not a pathognomonic symptom. Upon the development of bronchiectasis the sputum becomes characteristic of this affection. Blood is, in my experience, rarely found in the sputum, and in doubtful cases points decidedly in favour of tuberculosis.

Circulatory Disturbances, etc.—Stress is to be laid upon the obstruction to the circulation, which is a sequel of interstitial pneumonia of importance from the standpoint of diagnosis. Large areas of the pulmonary circulation are obliterated in interstitial pneumonia more so than in any other form of pneumonia. To this fact, and also to the deficient respiratory area, are to be ascribed the general symptoms of engorgement, cyanosis, dilatation and hypertrophy of the right ventricle, and the accentuation of the second pulmonary sound. The uncovering of the heart, which results from the contraction of the lung, is always to be considered when judging the size of the heart. Fever is absent, or but slight. If it is present to a greater extent,

it indicates other pulmonary processes occurring simultaneously with interstitial pneumonia—as a rule, tuberculosis of the lungs.

If the above symptom-complex is fully developed, the diagnosis of interstitial pneumonia is not difficult. It is different, however, if only circumscribed areas of infiltration and atrophy are present. In these cases the diagnosis is sometimes absolutely impossible, at other times only a provisional diagnosis can be made. The deepening of the supraclavicular and infraclavicular spaces, etc., corresponding to atrophied portions of the lungs, is of diagnostic importance. However, these local atrophies are usually, as stated above, of minor diagnostic importance in the main affection of the respiratory organs which accompany these atrophic processes, and are principally of significance only in the entire clinical picture and for the prognosis of the individual case.

CASEOUS PNEUMONIA, CHRONIC PULMONARY TUBERCULOSIS, PHTHISIS PULMONUM

The designation phthisis pulmonum embraces the destructive affections of the lung which show a progressive character and which are connected with the colonization and action of the *tubercle bacilli* in the lung. This process [1] manifests itself anatomically as catarrh of the apices and tubercular granulation, which is followed by tubercular peribronchitis and (caseous) broncho-pneumonia, resulting in the formation of cavities. These different anatomical stages of tuberculosis of the lungs are also observed in the clinical picture as readily distinguishable, developmental stages of the affection, easily ascertained by diagnosis. The *presence of tubercle bacilli in the sputum* is pathognomonic of all stages, and their demonstration is easy and forms the best support for a diagnosis of pulmonary phthisis of a chronic or subacute course.

Diagnostic Value of Tubercle Bacilli.—The elaborate investigations of R. Koch, in 1882, which demonstrated that tuberculosis is produced by a micro-organism, the tubercle bacillus, has entirely changed our methods of diagnosis, thus rendering the diagnosis more positive, perhaps, than that of any other disease. The property of the tubercle bacilli to retain the carbol fuchsin stain after being treated with nitric acid renders their recognition easy and positive. While the cells, nuclei, and other bacteria are discoloured upon the treatment of the preparation with nitric acid, the tubercle bacilli alone remain stained and can be easily recognised in the discoloured, or later counterstained preparation.

It is very easy, in by far the majority of cases, to demonstrate tubercle bacilli in the sputum of the patients. In rare cases it is difficult, owing to the bacilli being expectorated only at times and in small numbers. This is especially the case in the first stage of the affection, when it is often necessary to make twenty or more preparations before a positive decision can be arrived at. However, the diagnosis of pulmonary tuberculosis did not present any material difficulties before the discovery of Koch's bacillus, although it was not so positive as it is to-day. The diagnosis was then made solely from the character and the course of the anatomical changes taking place in the lungs and the general symptoms accompanying them. Although at present the finding of tubercle bacilli in the sputum takes the lion's share in the diagnosis of pulmonary tuberculosis, yet the most careful physical examination of the thorax is necessary under all circumstances, for, by this means, the first indications of the disease can be recognised, and their demonstration leads to the search in the sputum for the bacilli, thus enabling the physician to consider the case as suspicious from the beginning. Furthermore, the finding of tubercle bacilli in the sputum by no means decides the question as to the stage of the tubercular process, nor can a

[1] I refer to Rindfleisch's description in his text-book. Sixth edition, p. 447.

conclusion be drawn regarding the probable course of the disease, the threatening dangers, and the measures to be employed in combating the process. Aside from this, it is the object of every diagnosis to obtain as true a picture as possible of the *anatomico-organic changes* of every disease by a thorough clinical examination.

However, the diagnosis of tuberculosis without the demonstration of tubercle bacilli in the sputum cannot be made with as much certainty, for when tubercle bacilli are found, tuberculosis exists. In the usual staining methods, tubercle bacilli can only be confounded with the bacillus of leprosy and the smegma bacillus, which are as difficult to decolourize by acids as the tubercle bacilli. But the bacillus of leprosy occurs in larger colonies and cannot be transmitted to animals. This is also the case with the smegma bacilli, which also differ from tubercle bacilli in that when they are stained red with carbol fuchsin they gradually turn blue when treated with a saturated alcoholic solution of methylene blue, while the tubercle bacilli remain red. Other bacilli, besides the tubercle bacilli, are found in the majority of cases of pulmonary phthisis. Usually a mixed infection with streptococci, rarely with pneumococci and staphylococci, etc., is present. This fact is of particular importance because these micro-organisms are responsible for the fever of the patients, and render the prognosis much more unfavourable.

The introduction of the *tuberculin* into the therapy of tuberculosis by R. Koch seemed to open up a new means of diagnosis. The subcutaneous injections of tuberculin showed at once that tuberculous patients react locally (at the area of tubercular infection) and, as a rule, in a remarkably specific manner. It was hoped, therefore, that tuberculin would be an unerring means for the discovery of a tuberculous process in the body in general, but experiments which have been made the world over have shown that its diagnostic value is only a limited one. Its application is undeniably attended with danger, so that at present it is very little used as a means of diagnosis. However, in doubtful cases, I consider it advisable to make a trial injection of 0.005–0.01, and a positive or negative reaction will permit, though not a positive diagnosis, yet a provisional conclusion as to the presence or absence of tuberculosis.

I. DIAGNOSIS OF THE FIRST STAGE OF PULMONARY TUBERCULOSIS

Catarrh of the apices with tubercular granulation is considered as the first stage in an anatomical respect. This first stage of the affection manifests itself clinically by generally very pronounced symptoms which can be utilized in the diagnosis.

Not infrequently the pathological process is ushered in by a *general weakening* of the organism, by anæmia, diabetes mellitus, and poor nutrition in general, regardless whether this is the consequence of unsuitable nourishment or chronic gastric and intestinal catarrh. A further predisposition to tuberculosis is caused by local weakening of the pulmonary tissue due to inflammatory processes; but in acute and superficial processes this tendency is not nearly so marked, least of all in croupous pneumonia, in which, according to the experience of others as well as my own, it is almost never followed by tuberculosis. If no local inflammation of the parenchyma of the pulmonary tissue which changes its location, precedes, *the apex of the lung* is the first seat of the tubercular affection according to numerous observations. This is evidently caused by the fact that the inspired bacilli are driven, according to Hanau, farther into the apices by forced expiratory movements, if bronchial catarrh causes severe coughing.

Changes in the Apices.—*A catarrh which is localized in the pulmonary apices is under all circumstances very suspicious of incipient tuberculosis.* This catarrh is *nearly always,* as is proved by the more recent sputum examinations, *the sign of the entrance of the bacilli into the organism,* and with the catarrh *slight dulness in the supraclavicular region* can in the majority of cases be demonstrated. There are, however, exceptions to this

rule, such as, catarrh of the apices without demonstrable bacilli, without change in the percussion note, and infiltration of the apices. However, it is well to consider all these conditions as exceptions, and to exclude the diagnosis of incipient tuberculosis in any case only after repeated examinations.

Percussion and Auscultation.—Pronounced *dulness* over the apex, especially if the dulness is *unilateral*, which can be demonstrated in the infraclavicular space or the supraspinous region, is very significant of tuberculosis. Great precautions are to be taken, if one apex gives a slightly different note from the other, for such slight differences sometimes occur in non-consolidated apices. Especially have I found this the case in emphysema. Wrong conclusions are best avoided by percussing with the patient in a *sitting posture*. Bedridden patients should be made to sit up with the legs hanging over the edge of the bed. The examining physician should place himself exactly in the median line behind the patient and percuss so that he compares corresponding sides of the supraclavicular spaces, while the patient bends his head straightforward without deviating to the right or left. If a slight difference is determined between the left and right apex, or if both apices (rarely the case) show a uniform impairment of the normal percussion note, then the extent of the pulmonary note above the clavicle is to be determined. If this does not extend for a distance of 3 cm. above the clavicle it is to be considered abnormal, and this is also the case if upon linear percussion it can be determined that the borders of the apices are not uniform in extent. It is still more important from a diagnostic standpoint if *auscultation shows a deviation from the normal, thus confirming percussion*. The slightest change in the respiratory sound is sufficient, such as jerky respiration, prolonged expiration, weak, intensified, or coarse vesicular breathing, indistinct respiratory murmur. If the latter is of a bronchial character, or if crepitant râles are found, even if these are isolated and not of a metallic character, the slightest impairment of resonance at the apices is of great diagnostic significance.

Initial Hæmoptysis.—Tuberculosis commences in some cases with hæmoptysis, which occurs in apparently healthy individuals. *It has become more and more certain during recent years that these apparently spontaneous hæmorrhages are not the cause but a symptom of incipient tuberculosis.*

It is true, it is possible that in some cases hæmorrhage of the lungs causes the development of tuberculosis—namely, when actual trauma occurs in a previously healthy lung and leads to hæmoptysis. The blood which is found in the lungs could in this case become the medium for the organism which causes inflammation as well as for tubercle bacilli, and the latter might possibly enter the injured tissues more easily than in healthy tissue. This, however, is a very rare occurrence. *The reverse is almost always the case*—i. e., *hæmoptysis is the result of an existing though latent tuberculosis.* The tubercular granulations involve the walls of the branches of the pulmonary arteries at their point of entrance into the pulmonary acini, thus affecting the resistance of the vascular walls, resulting in rupture of the smallest vessels, and hæmorrhage into the bronchioles occurs, the walls of which are also involved in the tubercular process; in other cases the hæmorrhage is also caused by the rupture of a small vessel (which may be aneurysmal) in the wall of a small cavity, which has remained latent, and which occupies the apex of the lungs. If an acute inflammatory process supervenes upon hæmoptysis, occurring in an apparently healthy individual, it may be assumed that some of the contents of this cavity are aspirated with the blood, thus producing diffuse lobular inflammatory areas. Bäumler has recently called attention to this form of dissemination of the tubercular process which sets in rapidly and which almost always results in death in the course of one or two weeks. It occurs in apparently healthy individuals suddenly, or in quiescent tuberculosis after physical exertion which is accompanied with deep inspiratory acts, and is marked by blood-spitting, high fever, and diffuse

catarrhal symptoms in the lungs, which are followed in a few days by isolated crepitant râles, tympanitic percussion sounds, and increasing dyspnœa. This form of *acute broncho-pneumonia, which is caused by aspiration of blood and the contents of cavities*, differs from acute miliary tuberculosis by the rapidity of its course, greater dyspnœa, the occurrence of physical signs in the lungs, the hæmoptysis, and the greater frequency of tubercle bacilli in the hæmorrhagic sputum.

In such cases of apparently spontaneous hæmoptysis there can generally be demonstrated auscultatory and percussory deviations at the apices; but I wish to emphasize particularly that these deviations may sometimes be absolutely wanting.

In all these initial symptoms of tuberculosis the presence of *fever* will mostly be found. Exceptions to this rule occur, however, according to my experience, but they are rare, and are frequently due to the fact that the temperature is only taken once, or taken in the axilla. Very high fever occurs in cases in which the above-described lobular aspiration pneumonia follows hæmoptysis.

The diagnosis of incipient tuberculosis is supported by the *determination of an hereditary predisposition*; furthermore, by the above-mentioned debility, delicate structure of the body, weak development of the musculature, and insufficient development of the thorax. The development of pulmonary tuberculosis is also indicated by "scrofulous" glandular swellings in the neck, tuberculous affections of the bones and joints, or periproctitis with the formation of a fistula in the rectum.

Combination of Tuberculosis and Pleurisy.—*Pulmonary tuberculosis is, relatively speaking, often ushered in by pleuritis.* Here, also, the rule holds good, which is in accordance with the view held by me for a number of years, that the pleuritis is not the cause of the tuberculosis, but *vice versa*, the pleura is secondarily affected. In such cases an incipient and insidious tuberculosis, which gave no evidence of its existence, is complicated by an apparently spontaneous pleuritis, in the course of which, or after its "cure," the tuberculosis of the apex becomes prominent and without doubt.

That pleurisy is frequently of tubercular origin is also proved by the fact that tubercle bacilli appear in the sputum in such patients after the injection of tuberculin.

Of course, I do not mean to deny that pleuritis may be the primary, and *tuberculosis the secondary affection*, and that the predominance of the pleurisy materially favours the progress of tuberculosis.

The cause of this fact is not at all evident. According to experience, the lung on the opposite side of the area of pleurisy is usually the seat of the tubercular affection. It must be considered in such cases, that the inhalation of dust containing bacilli into the apices of the lungs is favoured by the great intensity of the inspiratory current in the previously healthy lung which is not retracted by pleuritic adhesions. It must be assumed, however, that it is a question of individuals hereditarily predisposed, or whose constitutions are debilitated, which so frequently is the case in connection with the predominating pleuritis.

In this stage, as well as in the other stages, the diagnosis becomes positive only by the demonstration of tubercle bacilli in the sputum, which may

be found when the physical signs are very slight, or absent, and even then in large numbers.

II. DIAGNOSIS OF THE SECOND STAGE OF PULMONARY TUBERCULOSIS

This stage is characterized by *tuberculous peribronchitis* and *caseous broncho-pneumonia.* Clinically this stage is generally combined with the third, the formation of cavities. Cases occur, however, in which the patients die before a material softening of the infiltration and elimination of the cheesy areas take place, in which induration, development of connective tissue and atrophy predominate. On the other hand, in some cases the cheesy infiltration and cavernous disintegration take a very rapid course (*phthisis florida, galloping consumption*), so that death occurs in the course of weeks or a few months. This is accompanied by fever and emaciation. All these anatomical changes can usually be found in one lung, but, in spite of this, it is clinically correct to differentiate tuberculous infiltrations with commencing disintegration diagnostically from the final stage of the tubercular process—the formation of larger cavities. Accordingly, we will have to discuss the diagnosis of tuberculous broncho-pneumonic infiltration.

Percussion furnishes the most important proof that such infiltration has taken place. Dulness, progressing from the apex downward, shows that air in the alveoli is gradually disappearing, intensified pectoral fremitus, bronchophony, ringing râles and bronchial breathing rendering the acoustic signs more transmissible. The catarrhal symptoms are in this stage also usually restricted to the superior portions of the lungs, corresponding to the changes on percussion, or they are more developed in these places than in the lower portions of the lungs. *Vice versa,* in cases in which gelatinous caseous pneumonias arise by aspiration of tuberculous masses (A. Fränkel and Troje) the infiltrated areas are found in the lower portions, a complication which is accompanied by remittent fever, expectoration of a brass-coloured or rusty sputum containing few tubercle bacilli, and rapid loss of strength. If interstitial development of connective tissue is marked, the upper portions of the lungs, which are usually thus affected, and the corresponding portions of thorax appear relaxed, according to the degree of sclerosis and atrophy of the pulmonary tissue. If the atrophy extends over a great part of one lung the other lung is often found to be in a state of compensatory emphysema, so that the clear sound of the latter extends farther than the median line, advancing beyond the opposite sternal border, and differs distinctly from the dull sound of the opposite or atrophied lung. The infiltrated portions of the lungs do not expand on inspiration, particularly so upon unilateral prevalence of the process.

Phthisoid Chest.—*The thorax appears flat,* the intercostal spaces are sunken, the weakness of the musculature, especially of the serratus anticus, makes the scapula project wing-like. The angulus Ludovici becomes very prominent, the neck is usually remarkably long, an *ensemble* of changes of the thoracic structure which is embraced under the name of the "*paralytic*" *phthisic thorax.* [Expiratory shape of chest.]

Secondary Phenomena.—The diagnosis in this stage is supported by various accessory symptoms, which are produced either by anatomical changes in the lungs or by the constitutional deterioration which accompanies tuberculosis. Among the former are the occasionally occurring *systolic murmur heard over the subclavian artery* below the clavicle, which can be explained by stenosis of this vessel due to atrophy of the apex, and the *unilateral paralysis of the vocal cords* produced on the right side by traction of the right recurrent laryngeal nerve which enters behind the subclavian. To the left it is usually the result of compression of the nerve due to enlarged bronchial glands at the aortic arch. *Pleuritic friction*, due to pleuritis over the infiltrated portions of the lungs, or tubercular nodules developing in the pleura, complete the picture of chronic progressive tuberculosis of the lungs.

Among the *subsequent general symptoms* may be mentioned the pallor, which in very pronounced, atrophic processes is combined with cyanosis, the tendency to perspire, especially at night, marked emaciation, and lassitude. Occasional complications are certain changes in the skin: pityriasis (tabescentium) and lichen scrofulosorum.

Fever.—The bodily temperature is almost always above normal, although in some cases there are long-continued afebrile periods. The fever is usually very high during the evening, the typus inversus is sometimes seen, and may exist permanently. In fact, as I have observed, it may occur with a certain degree of constancy in several members of the same family.

The *size of the heart is not, as a rule, altered*, because it adapts itself to the decreasing amount of blood, and thus the *hypertrophy of the right ventricle*, which might be expected owing to the circulatory disturbances in the lung, does not develop. But, according to my experience, there are occasional cases in which the right-sided hypertrophy of the heart is well marked, and can be demonstrated by percussion and by the accentuation of the second pulmonary sound.

Symptoms of Engorgement.—Among the symptoms of engorgement may be particularly mentioned the splenic tumour, engorgement of the liver (fatty liver is much more frequent), and engorgement of the kidney. The engorgement of the kidney is responsible, as a rule, for the decreased secretion of urine and the albuminuria which are seen during the course of phthisis. The heart may become dislocated in more pronounced atrophy of the lungs.

Sputum.—The *sputum* in this stage is muco-purulent. Upon the development of caseous tuberculous pneumonia it becomes greenish or rusty, glassy, similar to the sputum in fibrinous pneumonia. It contains desquamated alveolar epithelium, leucocytes, and various micro-organisms, streptococci, staphylococci, and pneumococci, but only the finding of tubercle bacilli is pathognomonic.

III. DIAGNOSIS OF THE THIRD STAGE OF PULMONARY TUBERCULOSIS

Condition of the Sputum.—This stage is characterized by ulceration of the bronchial walls, extensive reduction of the caseous masses, and the formation of *cavities*. Clinically this is shown at first by the occurrence of particles of pulmonary tissues, *especially elastic fibres*, in the sputum, which, however, are often found early, long before the physical symptoms of cavity formation are pronounced. Their demonstration does not present great difficulty, and is generally successful without the previous addition of hot caustic potash to the sputum. The macroscopical condition of the sputum also becomes characteristic; it appears *nummular, lumpy*, and *airless*. When expectorated into water it sinks quickly (*"sputum globosum fundum petens"*).

The rounded shape of the individual masses of sputum is explained by the fact that they have been in cavities until expectorated, an explanation which is not acceptable. The form of the sputum is due alone to the great cohesion of its parts.

The sputum is *airless* because it has been at the bottom of a cavity for some time, and later entering the bronchus was expectorated at once, and therefore did not become mixed with air. The sputum is sometimes hæmorrhagic. In some instances it is only a question of admixture of blood in streaks, at other times pure blood in small or large quantities is expectorated. This *expectoration of blood* has to be distinguished from initial hæmoptysis and may be the cause of rapid death if it is due to the erosion of large vessels.

Percussion.—If the presence of cavities in the lungs appears to be probable in the just-described condition of the sputum, this becomes a certainty by the results of *percussion* and *auscultation.* It is true they are not of the same significance as formerly, since we know that the demonstration of tubercle bacilli in the sputum allows of a much surer basis for the diagnosis of tuberculous infection, and that the presence of cavities may be taken for granted in a longer duration of the process, consequently it is immaterial whether cavities can be demonstrated by physical diagnosis or not. However, the evidence of their existence obtained by percussion and auscultation still remains an important feature of physical diagnosis, especially because thereby the size and position of the cavities, as well as their communication with a bronchus, can be approximately determined in these cases.

Signs of a Cavity.—The physical signs of cavity formation are as follows: The usual seat of cavity formation is at the apex of the upper lobes; here, especially in the infraclavicular space, the thoracic wall appears to be sunken. Percussion shows a *tympanitic* sound, which is, however, only caused by a cavity when it is at least the size of a walnut, and when directly adjacent to the thoracic wall, or only separated from the same by condensed airless tissue. The walls of the cavity should not be very tense and must be able to reflect the sound. The distinctness of the tympanitic sound depends upon the size of the quantity of air in the cavity—i. e., upon the size of the cavity, and the amount of fluid which is in the cavity besides the air.

Change of Sound.—Not rarely, but by no means in the majority of the cases, in fact not even regularly when large cavities occupy the apex, can so-called *change of the sound* be determined in the tympanitic note over the cavity—i. e., a higher or lower pitch according to manipulations which are made upon the patient. The high note occurs comparatively most frequently upon opening of the mouth, the lower upon closing (Wintrich's sign). In other cases the sound of the note changes in alternately raising and lowering the patient (erect and recumbent posture) (Gerhardt's sign), usually in such a manner that the note becomes higher upon sitting up, while the reverse is rarely the case. A combination of both signs can sometimes be determined—i. e., the occurrence of Wintrich's sign in an exclusive sitting position, in other cases again in the recumbent position of the patient ("interrupted" Wintrich's sign). Finally, it is possible to discover a slight change in the pitch by the respiration (Friedreich's sign). Almost all these percussion phenomena can also be observed in other conditions of the lung, and therefore they are not absolutely positive signs of the presence of cavities. *Positive proofs* of the presence of cav-

ities seem to be the lowering of the pitch of the note upon the patient assuming the erect posture, and the interrupted change in pitch. However, just these two signs upon percussion are very rarely observed in a distinct manner.

If Wintrich's sign upon opening and closing the mouth in the respiratory interval (during which it is usually elicited to prevent the eventual effect of Friedreich's change of note) is absent temporarily, it may be brought about eventually, as recently shown by Rumpf, in such a manner that the examination is made during the inspiratory period exclusively, best in the course of two successive inspirations, and that in such a manner that the percussion of the patient with closed mouth and nose is done during one inspiration, and the percussion with open mouth and protruding tongue during the following inspiration ("inspiratory change of pitch").

Cracked-Pot Note.—The occurrence of the *cracked-pot sound* is less applicable to the diagnosis, because it occurs in various pathological changes of the lungs, irrespective of the possibility to elicit it in some healthy individuals with thin thoracic walls, by percussion during loud talking. It is true, however, that this phenomenon is most distinctly and most frequently pronounced in cavities, and in such as are adjacent to the thoracic wall and communicate with the bronchus.

Metallic Percussion Note.—However, a cracked-pot sound points to the presence of a larger cavity in the thorax, if it is accompanied by a metallic note.

The latter, distinguished by its quality, by the prominence of very high notes besides the underlying note, is a sure sign that the percussion causes uniformly reflected sound waves, which are contained in large cavities—at least 6 cc. The respective cavities must not only be large to cause the metallic note, or the metallic after-sound (i. e., the slow ringing out of the high note after the deep underlying note has rapidly disappeared, "amphoric" note in the proper sense), but they must be near the surface also, and, above all, *their walls should be smooth, uniformly thickened, flexible, and their opening must not be too wide.* The metallic percussion sound is not loud generally, and often does not become distinct until the ear is placed upon the percussed area of the thorax (auscultatory percussion), and if the finger is not used, but a hard elastic rod, for instance, the handle of the percussion hammer ("rod pleximeter percussion"). If the cavity communicates freely with the bronchus and the oral cavity, the metallic percussion sound is heard louder with the mouth open, because the oral cavity thus acts as a sounding board and the note proper to this cavity, which is of a higher pitch when the mouth is open, serves to intensify the high cavity sounds corresponding to the same. *If a metallic sound can be produced by means of percussion, it is a positive proof of the presence of a larger cavity in the thorax*; then it remains only questionable whether this hollow space is a cavity or whether it is formed by a pneumothorax. We shall fully enlarge upon the decision of this question later on, when discussing the differential diagnosis of phthisical cavities and other pathological conditions in the thorax.

Metallic and other Auscultatory Signs.—The same as the percussion sound, so the respiratory murmur, too, may be characterized by a metallic note, respectively after-sound ("*amphoric*" *breathing*). The presumptions for its origin are the same as for the occurrence of the metallic percussion note. The voice, upon auscultation, also assumes a metallic timbre, and the *râles* arising in the large cavities or in their neighbourhood may likewise have a *metallic* quality. If, under these circumstances, isolated bubbles burst in the fluid of the cavities, they, too, may have metallic resonance and create the impression of a *ringing drop falling into the cavity* ("*tintement métallique*") [*gutta cadens*].

With the above description we are in a position to apply the *auscultatory signs* which point to the presence of a cavity. The last-named metallic manifestations are not at all frequent; usually only *bronchial breathing* is heard on auscultation. However, the latter is only present under certain conditions—namely, between the cavity and the thoracic walls there should not be much air-containing tissue, because otherwise the bronchial breathing prevailing in the cavity is masked by the vesicular respiration. Besides, the bronchus which leads to the cavity must not be obstructed by secretion. Of course, the bronchial respiratory sound is not especially characteristic of the presence of cavities, because bronchial breathing also occurs upon infiltration of the lung in the second stage of phthisis. Neither does the occurrence of metallic râles, or of so-called broncho-cavernous respiration, the increase of the vocal fremitus, etc., speak in itself for the foundation of a cavity. Therefore, so long as a metallic note does not accompany the phenomena of percussion and auscultation, the diagnosis of a phthisical cavity should be made with caution, and, even if this is the case, care should be exercised in the diagnosis, because in this case it may be a question of large cavities, and the decision is to be rendered *whether a pulmonary cavity or, rather, a pneumothorax is present.* This differential diagnosis may cause considerable difficulty in some cases.

Differential Diagnosis between Pulmonary Cavities and Pneumothorax.—Mistaking one condition for the other is possible especially when it is a question of *sacculated pneumothorax.* The symptoms caused by the latter are absolutely the same as in large cavities, and, according to the description, must be the same in so far as they refer to the conditions of auscultation and percussion. A differentiation of both affections is therefore actually impossible in some instances. The best differentio-diagnostic factor is the condition of the intercostal spaces over the respective parts of the thorax. *They are retracted in by far the majority of cases in cavities,* and *bulging in pneumothorax.* Besides, the *pectoral fremitus appears intensified over a cavity,* and *diminished over a pneumothorax, the same as bronchophony,* because, in my opinion, in pneumothorax the transmission is rendered difficult owing to the excessively tense thoracic wall, and because the bronchus is more remote from the thoracic wall. As soon as the pneumothorax assumes larger dimensions, or even affects the entire pleural cavity, mistakes are scarcely possible. The uniform distention of the thorax, the displacement of the adjacent organs, especially of the heart and liver, the changed dulness in the lower portions upon change of posture and the orthopnœa, but above all the *succussion sound, which appears in exceedingly rare cases in cavities—in a practice of many years I have never observed it in cavities*—establish the diagnosis of pneumothorax, while the physical signs connected with the metallic note, furthermore the cracked-pot sound and the change of pitch, are found equally in both conditions. It is true the latter signs are found much more frequently in cavities than in pneumothorax, so that the demonstration of the cracked-pot sound especially is very much in favour of the presence of a cavity, the same as profuse loud râles in the neighbourhood of the cavity, which seem to be quite close to the ear, when deciding the question as to whether pneumothorax or cavities are present, speak decidedly in favour of the presence of the latter.

Secondary Signs of the Third Stage.—Besides the above-described physico-diagnostic changes the following symptoms of this stage of phthisis are found: Increased signs of debility of the organism which also were prominent in the second stage, *progressive emaciation* (it is true there are exceptions to this rule in which the patient is well nourished in contradistinction to the pulmonary condition), bed sores, thrombosis of the crural vein with œdema of the respective extremity, or general œdema (due to *marasmus, secondary nephritis,* or *amyloid degeneration*). Analogous

to the disintegration of the *tuberculous products of inflammation in the lung, tuberculous ulcers form in the intestine and larynx.* More rarely they are found in the pharynx, *tuberculous rectal fistulæ*; the latter, however, may also develop in the first stage of the disease. The rapid loss of strength is promoted particularly by the fever, the insomnia, and furthermore, by gastric catarrh and profuse diarrhœas, which latter may be supervened by intestinal hæmorrhages originating from the ulcers. The *urine* of the patients shows, besides albumin depending upon eventual nephritis and the never-absent tubercle bacilli in complicating tuberculosis of the urinary tract, the *diazo reaction.* The latter is found constantly in cases of florid and steadily progressing chronic phthisis, so that their demonstration is generally of bad prognostic import.

In a relatively small number of cases the organism is flooded with the tubercular toxine issuing from the localized tuberculous area in the lung. In these cases we observe the clinical picture of *acute miliary tuberculosis,* the diagnosis of which will be discussed in the chapter on infectious diseases.

EMBOLISM OF THE PULMONARY ARTERY, HÆMORRHAGIC INFARCT

Ætiological Diagnosis.—*To produce embolism of the pulmonary artery a source for the formation of thrombi is required, the detachment of which from the locality of thrombosis is followed by the introduction of fibrin plugs into the pulmonary artery.* The locality, therefore, at which coagulation of the blood occurs should be looked for posteriorly from the branching off of the pulmonary artery from the right ventricle, consequently in the latter, in the right auricle, or in the veins of the periphery. Aside from valvular defects of the right heart in which coagulations may form immediately upon the valves which have become roughened, dilatations of the right heart are to be considered above all, especially such as develop after emphysema and mitral defects. As soon as the dilated heart relaxes in its compensatory activity and the relaxed myocardium is not able to give the proper propulsion to the blood, fibrin coagulations occur between the trabeculæ carneæ, or in the appendix of the right auricle, which are occasionally carried away by the circulation to the lungs. The conditions for the formation of thrombi in the right heart, without reference to the rare tricuspid-valve and pulmonary-artery defects, are most frequently present in mitral defects, but of course also, though less frequently, in all other valvular malformation and in degeneration of the myocardium generally. If the location of the thrombosis is outside of the heart in the venous system, the most frequent source of the emboli carried from the vena cava to the right heart and the lung, should be considered as coming from eventual inflammatory areas in the periphery or from marantic thrombosis in the crural vein and in the internal spermatic veins. The above-named ætiological factors should be considered primarily in every diagnosis of embolism of the pulmonary artery. If there is no good reason for the assumption of such sources of embolism, the diagnosis of embolism of the pulmonary artery rests on a very weak foundation and had better not be made at all.

The diagnosis, however, is usually easy. It is based upon the above-named ætiological factors, and furthermore, upon various marked symptoms which differ according to the embolism occurring in the trunk of the pulmonary artery or in a single smaller branch of the same.

Diagnosis of Occlusion of the Trunk and the Large Branches of the Pulmonary Artery.—*Embolism of the trunk or one of the main branches of the pulmonary artery* is characterized by rapidly occurrring dyspnœa, which is followed by death from suffocation, "pulmonary apoplexy," if large vascular districts are cut off from the blood supply. It is obvious, if the right heart can no longer empty its contents into the pulmonary artery, that a rapidly increasing dilatation, with diffusion of the cardiac dulness, acute cyanosis, smallness of the pulse, and mostly unconsciousness, owing to the engorgement in the cerebral veins and the insufficient supply of arterial blood to the brain, are the necessary consequences. But even if these symptoms can be determined by the physician himself (usually he only ascertains from the friends or family that dyspnœa, cyanosis, and unconsciousness preceded the sudden death), the greatest caution should be exercised in the diagnosis of embolism of the trunk of the pulmonary artery.

In this respect I have too often seen, in spite of the coincidence of the above symptoms, that no obturation of the pulmonary artery was found post mortem, and the cause of the rapidly fatal catastrophe remained obscure. The most probable cause of death in such cases is sudden paralysis of the heart. The consequences of paralysis naturally must be similar to those of embolism of the pulmonary artery—i. e., dyspnœa, cyanosis, acute dilatation of the entire heart, insufficient filling of the arterial system, and interruption of the cerebral circulation. Sudden death occurs as the result of this rapid relaxation of the heart.

Diagnosis of Embolism of the Smaller Arterial Twigs of the Pulmonary Artery and Hæmorrhagic Infarct.—In *obstruction of the smaller branches of the pulmonary artery*, which is followed by infarction of the respective vascular districts, the pathological symptoms are much milder and the diagnosis can be established much easier and with more certainty. Sometimes, not at all constantly, a *chill* designates the onset of pulmonary embolism. At the same time *cyanosis* and *dyspnœa* occur with increased respiratory frequency, the degree of which usually depends upon the size of the embolized portion of the lung in which the exchange of gases cannot take place. Gradually the organism accommodates itself to the diminished oxygen supply, and the difficulty of breathing becomes manifest only in greater demands upon the lungs produced by muscular exertion, in which case the patient complains also of palpitation of the heart.

Whether *fever* is connected with the embolic infarction or not depends, in my opinion, primarily upon the nature of the *embolus* introduced into the lung. If the latter originates, as is usual, in patients with adipose hearts or old cardiac defects, the course of the embolism is *usually afebrile*. If, however, the source of the embolus is due to acute endocarditis, or the cause lies in a peripheral inflammatory area in which the thrombosis has developed, the fever might increase owing to the embolism. In the second place, it should be considered that the circulation of the blood is obstructed in the infarcted portion of the lung so that the inflammatory germs are more apt to find lodgment, and consequently fever occurs. The occurrence of pleurisy at the base of the infarct, which is usually the case, points principally to the probability of the process having taken place in the above-named manner.

Blood in the Sputum.—The most remarkable of the objective symptoms is the condition of the *sputum*. This consists of pure *blood* which

is not, as a rule, bright-red but *dark* and *blackish*. It is frequently mixed with mucus, but not so intimately as in pneumonia. This hæmorrhagic condition of the sputum lasts for days and weeks.

This *hæmorrhagic* sputum is found on various occasions; besides occurring in pneumonia, it is found in neoplasms of the lung, in bronchitis, especially in croup of the bronchi, in perforation of aneurysms, the many forms of hæmorrhagic diathesis, etc. Confounding the hæmorrhagic sputum of the affection with which we are dealing, is possible above all with that occurring in the hæmoptysis of phthisical patients. In this case the simultaneous presence of tubercle bacilli in the sputum and the concentration of the pathological symptoms in the pulmonary apices are principally determining.

The diagnosis of hæmorrhagic infarct can never be established from the condition of the sputum alone. It becomes certain only upon the presence of the above-named diagnostic points, and particularly also by positive results obtained by physical examination of the lungs—namely, by the demonstration of circumscribed dulness (most frequently in the lower lobes, especially the right), of circumscribed bronchial breathing, ringing râles, and the other symptoms of consolidation. Also if the infarct has extended peripherally to the pleura, pleuritic friction sounds are not infrequently heard. Finally, where the coagulation becomes lodged, unless the obstruction of the respective branch of the pulmonary artery is not complete, a systolic, high-pitched whistling heart murmur can be heard, and eventually a thrill may be palpable. However, the determination of all these symptoms is not sufficient to establish a positive diagnosis of an embolus in the pulmonary artery. I will again emphasize the fact that, to accomplish this, it is necessary in the first place to demonstrate the source of the formation of the thrombi. Finally, it may be mentioned that there are cases of embolism in which no formation of infarcts occurs and which cannot, therefore, be diagnosticated, probably owing to sufficient collateral circulation between the pulmonary and bronchial arteries.

If the coagulation which leads to embolism is purulent or septically infected, there develops not a simple infarct of the lung but a *metastatic abscess*. If under such circumstances it is a question of septicæmic processes, there arise, as a rule, numerous small foci in the lungs which almost always escape the diagnosis and which are not discovered until the autopsy is made, as secondary findings. If, on the other hand, owing to the importation of the larger suppurating emboli into the pulmonary artery a large purulent area develops, this is known as a *pulmonary abscess*. This may also occur in a different manner, and its diagnosis is sometimes difficult, so that special discussion of pulmonary abscess is required.

PULMONARY ABSCESS

If the embolus which enters the lungs contains pus-producing cocci, especially in pyæmic processes, suppuration develops at the place in which the embolus becomes lodged. This is also the case if an inflammation in the lungs, either croupous, catarrhal, or a deglutition pneumonia, is accompanied with an intense effect of the cocci, and if the latter are in a position fully to display their property of dissolving albuminous substances. In such cases large purulent areas develop in the lungs. The pulmonary

abscess is, relatively speaking, frequently observed following influenza pneumonias.

Condition of the Sputum.—These ætiological factors must always be considered in the diagnosis of pulmonary abscess, for by observing them the differential diagnosis of this process is decidedly facilitated. The diagnosis of pulmonary abscess is often difficult, and can only be established with certainty when the symptoms are distinctly pronounced. The most important criterion is the condition of the *sputum*, which has the appearance of *pure pus*. If the expectoration is profuse, a serous upper layer separates from the cellular sediment. According to the amount of *pulmonary substance lost*, larger or smaller quantities of *parenchymatous shreds, elastic fibres*, single or stratified, fatty cells (fat partly in crystals), *cholesterin plates*, and *hæmatoidin crystals* are found in the sputum, and finally, as the most important constituents, the various *pus cocci* (staphylococcus aureus and albus, Friedländer's pneumobacillus) and other non-specific bacteria.

Development of Cavities.—The disintegration of the pulmonary tissue causes, according to its extent, the origin of a *demonstrable cavity* with the thoroughly explained symptoms characteristic of the physical demonstration of pulmonary cavities: tympanitic sound, metallic râles, amphoric respiration, etc., symptoms which may gradually disappear with the healing of the abscess, unless the loss of tissue is too great.

The diagnosis is supported by the course of the *fever*, which is usually accompanied with chills the same as in other suppurative processes.

Differential Diagnosis.—The *differential diagnosis* lies between pulmonary abscess and *empyema which has perforated into the lungs, phthisis with cavity formation, bronchiectasis*, and *pulmonary gangrene*. The first two conditions are usually easier to distinguish from pulmonary abscess than the latter. If pus has perforated into the lungs or bronchus from an abscess of the liver, the pleural cavity, gravitation abscess from the spinal column, etc., this condition manifests itself not only by various pathological manifestations which point to the origin of the expectorated pus, but above all by the fact that in such cases shreds of pulmonary tissue are absent from the sputum (except immediately after the perforation has taken place).

Phthisical Cavities.—The condition of the sputum is also decisive in the majority of cases of *phthisical cavities*. The sputum in these cases rarely has the appearance of yellow-green pus and so profuse an admixture of elastic fibres in alveolar order as is the case in pulmonary abscess. Above all, however, the sputum expectorated is not as massy as in abscess, and of special significance is the presence of tubercle bacilli in the sputum from tubercular cavities. The genesis of the infection is of importance in all cases—the origin of the abscess (from a certain source of embolism, pneumonia, or, after the entrance of a foreign body into the lungs) on the one hand, and on the other the relatively slow development of *phthisical cavities*. The seat of the abscess is not of importance in the differential diagnosis, because it is known that it occurs preferably in the upper lobes of the lungs, the same as tubercular cavities.

Bronchiectatic Cavities.—The differential diagnosis between pulmonary abscess and *bronchiectatic cavities* is sometimes more difficult. The sputum in the latter case has more or less the well-known fœtid odour; the elastic fibres are usually entirely absent, or at least they are not as abundant and not in such long rows in alveolar order as in abscess (compare p. 101).

Pulmonary Gangrene.—The latter is still more the case in the sputum of *pulmonary gangrene*, in which the presence of elastic fibres can be determined but rarely, for the latter are dissolved in gangrene by the peptonizing effect of a specific ferment. Exceptions may occur, however. In my experience it is possible that in pulmonary gangrene, if it is rapidly progressing, particles appear in the sputum which present the unchanged fibrinous structure of the pulmonary alveoli upon microscopic examination. Sometimes pulmonary abscess passes into pulmonary gangrene. The sputum in pulmonary gangrene has a penetrating odour, is of a dirty gray colour—in short, is characterized by very peculiar properties (by the occurrence of Dittrich's plugs, leptothrix threads which stain blue upon the addition of iodine). The course of the disease also is materially different from that of pulmonary abscess, as will be seen under the discussion of Gangrene of the Lungs.

GANGRENE OF THE LUNGS

This affection occurs much more frequently than does abscess of the lungs. Its diagnosis usually offers less difficulty than does that of abscess, because the symptoms of gangrene are very marked. The mortification of portions of the parenchyma, which is usually accompanied by putrid disintegration, nearly always causes the appearance in the sputum of larger or smaller gangrenous shreds of lung tissue, which have a very fœtid odour, a diagnostic substratum which may be considered almost pathognomonic of this affection.

Condition of the Sputum.—The *odour of the sputum* under such conditions is sometimes *cadaverously penetrating, exceedingly putrid*, or, sometimes, *mawkishly sweet*. The odour is especially intense during the time of expectoration. It is advisable, therefore, in doubtful cases, always to use a fresh receptacle to receive the sputum which is to be used for purposes of diagnosis. The diagnosis is impossible in cases in which no gangrenous sputum is expectorated. It is absolutely wrong, according to my experience, to make a diagnosis of pulmonary gangrene basing it merely upon a cadaverous fœtor of the expired air with simultaneous changes in the lungs which make the presence of gangrene possible. These diagnoses turn out to be wrong at the autopsy in more cases than those in which they are right. The consistence of the sputum is usually that of a *thin fluid*, the *colour dirty green, gray, or brown*, according to the admixture of more or less blood or blood pigment from the destroyed red blood corpuscles. It is especially characteristic that very soon after the expectoration of the sputum *three layers* are formed, to which fact Traube was the first to call attention. The upper layer is frothy, the central serous, both containing only small amounts of mucus. The lowest layer, the sediment proper, is from a diagnostic standpoint the most important part of the sputum. It

consists of detritus, fat globules, fatty acid needles and especially masses of villous shreds, up to 1 cm. in size, black or blackish-gray in colour, which can be recognised on microscopical examination as remnants of the dead pulmonary tissue from the alveolar order of the fibres. The alveolar structure, however, usually contains *no elastic fibres*, but only a striped foundation because (according to investigations made by Filehne some time ago on patients in my clinic) the sputum contains a peptonizing ferment which dissolves the elastic fibres.

If *well-preserved elastic fibres* occur in the gangrenous sputum—as occurs occasionally, which I emphasize particularly—this can be explained in such a way that their expectoration occurs very rapidly, before their chemical dissolution takes place in the gangrenous portions of the lung.

In the sputum of patients, suffering from pulmonary gangrene, there are, furthermore, found triple phosphate- and hæmotidin crystals, disintegrated blood corpuscles and pigment flakes, which form the principal constituents of the ominous plugs which are known since Dittrich's discovery of the gangrenous sputum. It cannot be decided with certainty whether certain micro-organisms are pathognomonic. A certain form of leptothrix, which was first described by von Leyden and Jaffé, seems to be of diagnostic importance. Upon addition of iodine the micro-organism stains between violet and blue. A micrococcus, which recently has been cultivated by Hirschler and Terray, appears also to be of diagnostic significance. This coccus develops an intense fœtid odour when grown on nutritive culture media. The following *chemical* putrefaction substances are demonstrable in the sputum in pulmonary gangrene: fatty acids, ammonia, phenol, indol, skatol, etc.

Symptoms.—In view of these properties of the sputum which are of such diagnostic importance, the remaining clinical manifestations of pulmonary gangrene are of minor significance. They consist in fever, loss of strength, dyspepsia, etc. Only the local changes in the lungs, which can be determined by percussion and auscultation, are to be considered beside the pathognomonic condition of the sputum, to supplement the diagnosis and to determine the extension and localization of the gangrene. The signs of rapidly progressing infiltration appear to be more prominent than the symptoms of cavity formation in the diffuse form of the disease, in the circumscribed form the latter predominate. It is especially characteristic of extensive infiltrations affecting an entire lobe and occurring in a few days, sometimes hours, as I observed in one patient. Extensive dulness is then followed in a few hours by a tympanitic note, which is associated with metallic râles and the other cavity symptoms.

The *differential diagnosis* between pulmonary gangrene, putrid bronchitis, and bronchiectasis (in which conditions fœtid sputum also is expectorated) may be difficult as long as no pulmonary shreds can be demonstrated in the sputum. If these diseases have existed for some length of time, and if portions of pulmonary tissue are now found in the sputum, this proves that the putrid disintegration of the contents of the bronchi or [illegible] has passed into the adjacent pulmonary structure—

i. e., a secondary pulmonary gangrene has supervened upon these less pernicious processes.

Ætiological Points.—This diagnosis is supported, if we succeed in tracing the genesis of the pulmonary gangrene in detail, especially in determining whether, besides the above-named diseases, pneumonic infiltrations preceded, in particular deglutition pneumonias, phthisis, abscess, or trauma affecting the lung with or without injury to the thoracic wall. Or, finally, if a source can be found for the importation of emboli, which may be considered a more certain direct cause of pulmonary gangrene if putrid disintegration can be demonstrated in the peripheral primary focus of the thrombus. Severe *general disturbances of nutrition*, the long duration of infectious diseases, particularly the severe disturbances of metabolism seen in *diabetes mellitus*, undoubtedly also favour the occurrence of pulmonary gangrene.

SYPHILIS OF THE LUNGS

Syphilis of the lungs manifests itself, the same as in other organs, in the form of diffuse inflammatory processes of a fibrous character, or in the form of gummatous nodes, especially in the mediastinal glands; but if the positive recognition of anatomical changes as being syphilitic are doubtful, this is still more the case for the *clinical* diagnosis of pulmonary syphilis, which is nothing more than a supposititious diagnosis, especially as it is beyond doubt that mixed forms of syphilis and phthisis occur. The marked presence of an advanced stage of lues, the predominance of atrophic processes of the lungs, intense dyspnœa, which is disproportionate to the chronic inflammatory changes of the lungs (caused by stenoses of the bronchi), but, above all, the absence of tubercle bacilli in the sputum after repeated examination for the same, would indicate in a doubtful case the syphilitic character of the pulmonary affection, particularly if an antisyphilitic treatment arrests the further progress of the disease, but even then a *positive* diagnosis of syphilis is not admissible.

NEOPLASMS OF THE LUNGS

The same difficulty is experienced in the diagnosis of pulmonary neoplasms. Here also the ætiology—i. e., the demonstration of the tumefaction, of sarcoma and carcinoma in other places of the body—present the best support for the diagnosis. It is difficult under all circumstances to diagnosticate primary neoplasms of the lungs with certainty. In fact, it is only possible in exceptional cases, if it is a question of small solitary or extremely small nodules permeating the entire lung (carcinosis pulm. miliaris). The symptom-picture of *carcinoma of the lungs* sometimes simulates chronic pneumonia, at other times a mediastinal tumour—i. e., a locally limited tumour in the thoracic cavity (see the following chapter). If, in this picture, hæmoptysis (sometimes in the form of raspberry jelly-like sputum, which, however, occurs also in other affections according to the experience of others as well as my own), hæmorrhagic pleurisy, metastatic enlargement of lymph glands in the axilla, eventually remittent fever and a remarkable progressing cachexia, occur, carcinoma of the lungs may be assumed with a considerable degree of probability. But the diagnosis does not become certain until we succeed in demonstrating microscopically carcinomatous elements in the sputum, or with the aid of exploratory puncture, or if the carcinoma, the course of which so far was under the above-named pathological picture, perforates the thoracic wall. However, both occurrences are very rare. The diagnosis of primary carcinoma of the lungs, therefore, is almost always very difficult, and even the most experienced diagnostician is liable to error if he attempts to establish the presence of pulmonary carcinoma from the mere *ensemble* of the pathological symptoms without the above-named direct points of support.

Echinococcus of the Lungs.—A similar point of view is to be taken in the diagnosis of echinococcus of the lungs. A positive diagnosis is only possible in these cases, if cysts, portions of the cystic wall, or the contents of the cyst with echinococcic hooklets are expectorated. An approximately positive diagnosis cannot be

upper part of the sternum bulged, and slight dulness appeared to the right, posteriorly and above. *The vocal fremitus was intensified anteriorly and to the right below the clavicle, and had entirely disappeared from the right nipple downward.* The heart could be felt in the sixth intercostal space beyond the left mammary line.

Attacks of asthma and dysphagia occurred in the further course of the disease, the dyspnœa leading towards the end of life to bronchial stridor, which appeared over the sternum at the height of the third intercostal space. The cutaneous veins over the upper portion of the right half of the thorax were distended and tortuous. At the same time there was cyanosis, *œdema of the right side of the face and the right hand*, later also in the region of the right ankle. No marked difference could be determined between the left and right radial pulse, nor a change in the pupils or in the fundus of the eyes. *But nevertheless*, by reason of the symptom-complex described, the diagnosis of *mediastinal tumour with right exudative pleurisy could be made with certainty*, especially because a gradual extension of the dulness to the left side could be determined, so that it extended a few centimetres beyond the left border of the sternum into the left portion of the thoracic cavity. During the last days of life the apex of a stone-hard tumour, one finger-breadth in width, appeared over the manubrium sterni in the jugular fossa, and could be distinctly palpated. With these facts the character of the mediastinal tumour could be determined with some certainty. Death occurred with severe attacks of suffocation and profuse hæmoptysis two months after the admission of the patient.

The *autopsy* (Rindfleisch) showed an enormous mediastinal sarcoma of a breadth of 27.5 cm. and 17.5 cm. high. This tumour occupied the greater part of the right portion of the thorax and a small portion of the left. A portion of the right cavity was not occupied by the tumour and was filled with a yellowish, slightly cloudy fluid. Exactly at the height of the sixth rib, above the accumulation of fluid, a hard tumour commenced which closed the pleural cavity transversely at this place, at least anteriorly, while posteriorly, behind the tumour, the hand could be introduced to about the middle of the scapula. The tumour extended upward beyond the manubrium sterni compressing the trachea from right to left. The descending aorta had remained unchanged, the aortic arch was only slightly compressed. On the other hand, the right innominate vein was entirely compressed by the tumour, the heart was displaced to the left and downward, and not hypertrophied. The right lung was displaced backward, very much compressed, and adherent to the tumour. The upper lobe of the left lung was firmly adherent to the extent of 3 to 5 cm., this area being absolutely airless. The height of the tumour on the left side in the mammary line was 10 cm.

Auscultatory Changes in Mediastinal Tumours.—The *respiratory murmur* is usually lessened as in the case described, but it remains vesicular, and at the places of greatest compression slightly bronchial. *Stridor and ægophony* may occur in the course of the disease *owing to compression of the trachea or bronchi.*

Pressure Signs on the Part of the Circulation.—The result of the laryngoscopical examination is of diagnostic importance in many cases, because it reveals stenosis of the trachea produced by the tumour; or paralysis of one or both vocal cords may be determined, which is caused by the pressure of the mediastinal tumour upon the vagus or recurrent laryngeal nerve. The impediment to respiration and the decreased oxygenation of the blood caused thereby produce venous discoloration in the capillaries of the skin, a more or less pronounced *cyanosis*, as the sequence of mediastinal tumour, and this cyanosis is still further increased by the *pressure of the tumour upon the large venous trunks*. They do not offer sufficient resistance to the growth of the tumour, consequently they are compressed, and engorgement occurs with its consequences in the affected venous region.

Consequences of Venous Compression.—If a tumour proliferates in the *lower portion of the posterior* mediastinal space anteriorly and to the right, the thoracic portion of the inferior vena cava may suffer from pressure. This will cause œdema of the abdomen and of the lower extremities. It may develop to a high degree, and, as in one of my cases, be the only symptom of venous engorgement for some time. According to the position of the inferior vena cava there would be present, besides the pressure of the latter, a pressure upon the *phrenic nerve* which lies immediately behind the vein, which causes singultus and impairment of the activity of the diaphragm in respiration. If the tumour in the posterior part of the mediastinal space grows more posteriorly, or towards the left, compression will be exerted upon the œsophagus, the left phrenic nerve, and both vagi.

If the pressure of the tumour exerts itself more upon the *central district* of the diaphragmatic space it affects the *superior vena cava*, causing *swelling of the face and both upper extremities*, as well as engorgement of the brain, which is manifested by headache, tinnitus, vertigo, etc. But this occlusion of the vena cava may be compensated by the well-known anastomoses between the superior and inferior vena cava, causing great distention of the superficial veins of the thorax and abdominal wall. Usually not the superior and inferior venæ cavæ but the *innominate vein of the right or left side* will become obstructed when pressed upon by the tumour. In such cases, owing to the less impeded deflux of blood from the affected common jugular vein and subclavian vein, a characteristic *unilateral œdema of the throat, face, and one arm occurs*, also *unilateral swelling of the veins of the thorax and of the abdominal wall*, owing to the impediment to the deflux of the blood from the internal mammary into the innominate. Deviations from this picture occur if, according to the location of the tumour, only the subclavian of one side or the azygos or hemiazygos are affected by the pressure of the tumour.

Results of Arterial Compression.—The large *arterial trunks* offer considerably more resistance to the pressure of the neoplasm than the venous trunks; in fact, the above-mentioned case proves that the heart easily overcomes the obstruction and need not become hypertrophied, even if tumour masses undoubtedly encroach upon the lumen of the organ. If the stenosis is considerable and affects the subclavian, left carotid, or innominate arteries, an enfeeblement of the pulse upon the affected side may result.

Pressure of the Tumour upon the Heart, etc.—The heart proper is also affected by the pressure of the tumour. The diastole is impaired, and the deflux of the blood and filling of the arteries are thus impeded. A dislocation of the organ also occurs, usually with displacement of the apex beat towards the left and downward. The same as over the diaphragm the heart is displaced by the tumour, so will large neoplasms also displace the organs downward adjacent to the lower surface of the diaphragm, viz., *liver* and *spleen*. This is demonstrated by palpation.

Pressure on the Œsophagus.—Besides the respiratory organs, the heart and the vessels located in the mediastinal space, the other organs situated there may be compressed by the growing neoplasm, particularly the *œsophagus*, the compression of which impairs or renders deglutition impossible.

Pressure on the Nerves.—Above all it may press on the *vagus*, recurrent, phrenic, and sympathetic fibres. If the vagus is affected, besides the already mentioned asthmatic attacks, dysphagia occurs due to arrest of the food or difficulty in swallowing.[1] Vomiting is also observed in mediastinal

[1] Of course it is provided in these cases that the lumen of the œsophagus proper is not compressed by the tumour.

tumours and may be ascribed to disturbances in the innervation of the vagus, particularly upon irritations of centripetal vagus fibres. (It is well known that bilateral section of the vagus prevents the motions of vomiting.) A remarkable slowing of the pulse or acceleration may be due to compression of the vagi. Better supports for the diagnosis are given by the compression of the *recurrent laryngeal nerve* with its well-known result—paralysis of one or both vocal cords. It appears also that sometimes a spasm of the adductors may temporarily be caused by the pressure upon the recurrent laryngeal nerve. Finally, the *sympathetic fibres* may also be affected by the compression and very remarkable symptoms may follow, especially inequality of the pupils. The unilateral dilatation is caused by irritation of the fibres which enter the sympathetic nerve from the spinal cord through the superior dorsal nerves.

The various subjective symptoms which are caused by the growing neoplasm, should not be taken into consideration in establishing the diagnosis. However, the *metastatic swelling of peripheral lymph glands* in the throat, axilla, etc., are of diagnostic significance.

The above-described characteristics of mediastinal tumours—namely, *dyspnœa, bulging of the thorax, the irregular borders, and the steady growth of the borders of the dulness, the disappearance of the respiratory murmur, the displacement of the heart and of the abdominal organs, the marked symptoms of engorgement in the venous system, and of the pressure upon the arteries and nerves situated in the mediastinum and upon the œsophagus and the bronchi*, allow of the diagnosis of mediastinal tumour with more or less certainty. The latter becomes positive when the growing tumour passes the borders of the thorax and becomes palpable, either because it produces erosion of the ribs and perforates externally, or *because it appears over the clavicles or over the incisura semilunaris of the manubrium sterni in the jugular fossa in the neck*. However, it is always necessary before the final establishment of the diagnosis to take into consideration such diseases as cause similar symptoms in order to give greater security to the possibility of a mediastinal tumour by excluding them.

Differential Diagnosis.—So long as the mediastinal tumour is of such small dimensions that it produces no dulness, the diagnosis can only be surmised, and it is of very little value to discuss the question whether the doubtful symptoms of compression are caused by distended tuberculous bronchial glands, syphilitic changes with cicatrices, a latent aneurysm, or a commencing mediastinal tumour. To decide whether one of the latter is present, only becomes possible when dulness begins in the anterior or posterior parts of the thoracic walls with supervening symptoms of lessened space in the thoracic cavity. This, it is true, is the case in various affections of the thoracic organs.

Mistaking pleurisy and pericarditis for the condition can be best avoided if the steady, *slow* diffusion of the dulness *and the progressive compression of various organs* situated in the mediastinum are sufficiently observed. I wish to emphasize in particular also as a symptom indicating mediastinal tumour, *the irregularity of the margins of the dulness.*

The observation of this sign exclusively, enabled me in a case recently observed in my clinic to make the diagnosis of mediastinal tumour correctly. The course of the latter was very suggestive of an extensive left-sided pleural exudate (determined by puncture) with considerable dyspnœa. The cardiac dulness passing into the left-sided dulness caused by the pleural exudate, extended beyond the right border of the sternum. The extreme marginal borders of this dull area which extended to the right were not regular, however. It was possible to determine inferiorly a dull area *about 5 cm. long which extended beyond the marginal line in a tongue-shaped manner to the right.* This caused a diagnosis of a simple pleural exudate with displacement of the heart to the right, to be replaced by the diagnosis of mediastinal tumour.

Pericardial Exudate.—The differential diagnosis is still more facilitated by the observation of the following finer characteristic signs: The *pericardial exudate* causes the apex beat to become localized within the cardiac dulness, slightly palpable, and may be brought into prominence by the bending forward of the patient. This also causes the borders of the dulness to increase. Dulness caused by a mediastinal tumour, on the other hand, does not vary in size upon change of posture of the body. Furthermore, the heart, which is displaced by the tumour, always beats with its apex against the most extreme border of the cardiac dulness, if its dulness can be outlined from that of the tumour. The heart beats disappear entirely in rare cases, in which the mass of tumour extends between the heart and the thoracic walls. However, in this case, in contradistinction to the conduct of the heart beat in pericardial exudates, it will always remain unobserved upon the patient bending forward.

Pleural Effusion.—The differentiation of the mediastinal tumour from *pleuritic exudates* may be more difficult. Common to both are dyspnœa, the dislocation of the heart and of the neighbouring organs generally, the bulging of the affected half of the thorax, the engorgement œdema, which eventually develops unilaterally, etc. But irregular borders of the dull area extending in the *upper* portions of the lungs which eventually reach the other side without the dulness affecting the lower portion of the thorax, are *against* pleuritic effusion, also the fact that in the area of dulness, although it is absolute, the vocal fremitus has not disappeared. However, all such characteristic symptoms become secondary to the result of the exploratory puncture, which clears up the situation at once. It is important in general for the differential diagnosis between mediastinal tumour and pericardial and pleural exudates that in the latter conditions one or the other pressure symptoms (paralysis of the recurrent laryngeal nerve, unilateral engorgement œdema, dysphagia, etc.) may occur, this, however, is rarely the case, and the pressure symptoms never appear so developed, nor are they so prominent as in mediastinal tumours.

Malignant Disease of the Pleura.—Usually no serious difficulties are encountered in distinguishing mediastinal tumours from pleural exudations, yet they are present to a considerable extent in the differentiation from *pleural neoplasms.* The diagnosis is especially complicated when the new formation originates in the costal pleura, if it does not perforate externally, and if the tumour masses assume larger dimensions. In the latter case they cause the thoracic wall to bulge, they compress the lung, the vena cava, the œsophagus, etc., and they may further proliferate into the mediastinal space. A differentiation of both pathological conditions appears impossible, therefore, but it should not be forgotten that upon new formations in the pleura, exudation of fluid in the pleural sac will follow, which is not always the case

with mediastinal tumours. A negative result will be obtained upon exploratory puncture with the Pravaz syringe, which is commonly used, if the puncture is made directly into the tumour mass. But if the long needle is employed, we will reach through the hard mass of the tumour, which in such cases can be plainly felt, into the fluid, which, in this case, is usually bloody, and may be aspirated. That the differential diagnosis may be established correctly in this manner, is proved by a case of pleural sarcoma which was recently observed by me, and the course of which will be reported in detail in the discussion of the diagnosis of pleural malignant disease.

Aneurysm of the Aorta.—Of frequent occurrence is the confusion of mediastinal tumours with *aneurysms of the ascending aorta and of the aortic arch.* Common to both is the lessening of space in the thorax, the dyspnœa, the dulness over the sternum, its gradual extension, the compression of the surrounding parts, especially of the œsophagus, the bronchi, the veins and nerves, and the dislocation of the heart. This shows that the symptom-picture is the same in all its essential points—obviously because the symptoms of an intrathoracic tumour filled with fluid cannot be any different from those of a solid tumour in regard to displacement and compression of the adjacent organs, nor can the result of percussion be different. Symptoms of differentiation may be expected from the occurrence of *murmurs*, which are synchronous with the systole and diastole of the heart, and from the *pulsation of the tumour.* These factors actually prove to be firm diagnostic points in the majority of cases.

As regards *pulsation*, it is characteristic of aneurysm. It is true a mediastinal tumour may also pulsate, but it is of rare occurrence and may be absent in the largest tumours, while pulsation is almost never lacking in large aneurysms. It is caused in mediastinal tumours by the transmission of the vibration to the tumour through the aorta, which is situated below the latter, or by the heart which elevates the tumour. It is characteristic that in mediastinal tumours, the same as in other pulsating solid tumours, only a raising and lowering, very rarely an expansion from left to right, can be observed. Besides, in these cases, we never find the uniform general and gradual extension of the pulsating tumour as in the aneurysmal sac. The differential diagnosis, however, in so far as it is based upon pulsation, always meets with great difficulties. These are also encountered in the diagnostic utilization of *vascular murmurs.* It is true that here also the occurrence of loud murmurs at once indicates the presence of aneurysm, but they may even, if the latter is of considerable extent, be entirely absent, according to experience, and, on the other hand, vascular murmurs may be heard also when mediastinal tumours are present. The latter is the case when stenosis of the large vascular trunks arises, owing to compression by the tumour, but it may, as proved by the autopsy report of the above-detailed case, be rather pronounced without the occurrence of murmurs. At any rate they are systolic, while in aneurysm *diastolic* murmurs may also be audible. Inequality of the radial pulses is against mediastinal tumour, early occurrence of pressure symptoms more in favour of the same, particularly if their occurrence renders a considerable degree of compression probable (like the buttonhole stenosis of the trachea), or an effect of the compression towards the right, like an exclusively right-sided or bilateral paralysis of the vocal cords. The diagnosis of the mediastinal tumour in comparison to an aneurysm of the aortic arch becomes quite feasible if an *unilateral* and *right-sided œdema of the face and the arms sets in* (caused by pressure upon the right innominate vein), because the aneurysm may occasion compression of the left, but not a compression of the right, innominate vein, according to the situation of the aortic arch. It is not necessary to explain further that the diagnosis of mediastinal tumour becomes plainer by the occurrence of metastatic glandular enlargements in the neck and in the axilla, or by the apex of the hard tumour becoming palpable in the jugular fossa.

Diagnosis of the Nature of Mediastinal Tumours.—Finally, the question to be considered is, as to the *character of the diagnosticated mediastinal tumour.* The most frequent tumours are sarcomata and carcinomata; cysts and lipomata, etc., are rarely found. The rapid growth, the metastatic glandular swellings, cachexia, tumours in other parts of the body, indicate a malignant character—sarcomata by the youthful age. The anatomical diagnosis is doubtful in by far the majority of cases, and even surmises are not in place to avoid disappointments.

Mediastinal Hæmorrhage and Abscess.—However, in all cases *mediastinal hæmorrhages* and *abscesses* should be excluded as much as possible before positively diagnosticating a mediastinal tumour. The first-named mediastinal affections should only be thought of, if the *ætiology* points to them with some degree of certainty. An abscess especially becomes probable if trauma has been inflicted upon the thorax, if suppuration, with or without caries, can be detected in the neighbourhood, if pyæmia is present, if a pulmonary abscess or empyema has existed until then or could be diagnosticated, and now pressure symptoms and dulness become prominent in the mediastinal space in the pathological picture.

Mediastinal hæmorrhage should practically be considered last, owing to the rarity of extensive blood extravasations in the mediastinal space. They should be thought of when the intramediastinal pressure symptoms develop rapidly, when a trauma preceded, or when a general (scorbutic, etc.) tendency to hæmorrhages can be positively demonstrated.

In conclusion of our diagnostic discussion, I wish to emphasize *that the definite diagnosis of the mediastinal tumour is always to be preceded by an exploratory puncture,* except when the seat and character of the tumour in question render the presence of an aneurysm possible. Exploratory puncture has recently been recommended and employed in the latter case also to decide the question whether aneurysm or mediastinal tumour is present.

DISEASES OF THE PLEURA

PLEURITIS

The diagnosis of pleuritis is based *solely on the results of the physical examination.* The subjective symptoms of which the patient complains, even the pain upon breathing, and also the general symptoms, especially the fever, are not sufficient for a diagnosis, at least they should be only secondary factors in judging the nature of the disease. The physical signs vary in the individual cases according to the inflammation of the pleura being accompanied by the deposition of a fluid exudate or not, and therefore the diagnosis of dry pleurisy and pleurisy with effusion should be discussed separately.

Pleuritis Sicca.—The patient usually lies upon the back or on the unaffected side; the respiratory excursus is less marked upon the affected than upon the healthy side. Upon palpation we find distinct friction due to roughening of the pleural surfaces. *Percussion* shows nothing abnormal,

at most a deficient movability of the pulmonary borders, either because the greater respiratory excursions are limited on the affected side, owing to the pain, or because adhesions occur between the pleural surfaces and the displacement of the lungs in the complementary space becomes reduced. *Auscultation* furnishes the main criterion for the existence of pleuritis sicca. It may yield a *friction sound* which may vary in degree and intensity, but at the same time presents certain essential qualities.

Friction Sound.—The friction sound occurs in intermissions slightly varying in acoustic intensity, it is not continuous, and is almost always divided between the inspiratory and expiratory phases, not restricted to inspiration alone as are crepitant râles. Furthermore, it creates the impression of being very superficial, arising in the immediate neighbourhood of the ear, and upon a sensitive ear it always causes a more or less disagreeable, and, as a rule, grating sound. It is advisable to place the ear firmly against the thoracic wall when auscultating, and have the patient take as deep inspirations as possible. The sound is generally heard more distinctly when a stethoscope is employed. We are often in doubt, in spite of these characteristic symptoms, whether we have to deal with a pleuritic friction or other similar sounding changes in the respiratory murmur. Of course, this is not the case if the friction sound is grating or rubbing in character. If the latter, however, is *very soft*, or *vice versa* more *crackling*, the determination of the character of the sound may often become difficult.

Differentiation from Dry Râles.—The coarse friction sound may eventually be confounded with dry râles, particularly because they may also be observed on palpation, as is well known. Above all, the friction sound occurs at *intervals*, and it must be observed whether the disappearance of the sound or a change in vocal fremitus is caused by violent attacks of coughing. If the latter is the case, it will point to the origin of the râles to bronchial secretion, while the pleuritic friction is not altered thereby in its intensity, and above all, not in its extension. The decision becomes more difficult, of course, if dry râles and pleuritic friction sounds are both present. Here the observation of the variableness of the sounds produced in different areas of the thoracic wall upon coughing is helpful. If, on the other hand, the râle *is soft, more uniform and fine*, it is very frequently mistaken for crepitation.

Crepitation.—The fact that the latter with few exceptions is restricted to inspiration and occurs in a "shower," permits a positive decision in the majority of cases. Both of these sounds, crepitation and pleuritic friction, may become weaker or disappear entirely during a prolonged examination, to reappear after some time of rest. Pleuritic friction should also be surmised if the above sound is concentrated upon a circumscribed area of the thoracic wall, and it becomes quite certain if the rhythm of the sound shows certain, although insignificant, irregularities, and also affects expiration. *All the above-named characteristic symptoms are not sufficient in rare cases for even a very practised ear*, so that the diagnosis has to be left undecided. If the friction sound occurs in the neighbourhood of the heart it is possible that it is not only caused or modified by respiration, but also by the action of the heart, which we explained when discussing pleuro-pericardial friction (see p. 44).

A sound similar to pleuritic friction may also occur in *miliary tuberculosis of the pleura*, to which fact Jürgensen was the first to call attention. I am able to confirm this occurrence of a sound produced by the displacement of the pleural surfaces which have become uneven owing to the formation of tubercles, and I consider it permissible to make use of the pleural friction in the diagnosis of miliary tuberculosis, although I do not think it possible to distinguish upon auscultation this sound produced by touch from a delicate friction sound produced by pleuritis.

In the presence of the friction sound all other symptoms of dry pleuritis are of little significance. For instance, whether the pain, which is actually present in the majority of cases, becomes more or less pronounced, or whether cough accompanies the disease or not. It is undoubtedly true

that cough may be absent in some cases. While the principal feature in pleuritis sicca is the friction sound, the latter is of minor importance in the diagnosis of

PLEURITIS EXSUDATIVA (PLEURISY WITH EFFUSION)

The patient lies mostly upon the affected side, which thus permits the healthy lung to extend fully without difficulty. In spite of this, however, with large exudates respiration becomes impeded. If the patient is in the dorsal position we see that the affected half of the thorax expands to a less extent on respiration, first in the lower, later, upon extension of the exudate, also in the upper portion of the lung. The affected half of the thorax is distended visibly, and on measurement shows an enlargement of several centimetres discounting about 1 cm. on the right side, especially in the region of the hypochondrium. The adjacent organs become displaced by the exudate, the heart beat can be demonstrated to be displaced to the opposite side, the diaphragm and the organs adjacent to it are dislocated downward, which can be easily determined by palpation and percussion of the liver and spleen.

As to the palpation of these organs, the downwardly displaced *liver* can easily be felt in its changed position. *The spleen, however, which has been forced downward, cannot,* according to my experience, although we may succeed in palpating a moderately enlarged spleen (by 1 to 2 cm.). The reason for this is that the enlarged spleen approaches the palpating hand upon inspiration, which is not the case in dislocation of this organ, for in the latter case the downwardly distended diaphragm remains immovable on inspiration.

Pulsating Pleurisy.—In rare cases there may occur extensive systolic pulsation, which is transmitted from the heart and diffused over the affected half of the thorax, *pleuritis pulsans.* It may be that the cause of this phenomenon is due to inflammatory serous relaxation of the intercostal muscles with great tension of the exudate (Traube-Keppler) so that strong movements of the heart are transmitted through the fluid and transferred to the thoracic wall. The phenomenon is by far most frequently found in left-sided pleurisy. It is especially observed in sacculated exudates and upon a purulent condition of the same. If the pulsation does not extend over a larger portion of the thorax, but is restricted to a limited bulged area which tends to perforation, a pulsating aneurysm may be simulated (see p. 160).

Percussion.—*Percussion* yields the most important signs. *The sound when the exudate is abundant is absolutely flat, and the percussing finger experiences the feeling of great resistance.* Of course, it depends upon the size of the exudate how far upward the dulness extends. The upper limit of the fluid is generally higher posteriorly than anteriorly, owing to the fact that the fluid sinks to a greater degree posteriorly upon the patient assuming the dorsal decubitus. Deviations occur in every direction, however, from this sinking of the upper border of the dulness from the back forward. Sometimes it is equally as high anteriorly as posteriorly. In other cases the course of the upper margin of the dulness is not straight but is interrupted by elevations in the axillary line. The cause of this is beyond doubt the conduct of the patient during the formation of the exudate, viz., whether the accumulation of fluid occurred while the patient was walking about or while he was constantly lying on his back or side.

The varying course of the upper margin of the dull area is determined in the second place by an occasional occurrence of fibrinous adhesions between the pleural surfaces (at the upper border of the fluid) which obstruct the extension of the dulness in these areas. *The borders of the dulness usually do not change in particular upon the change of posture, in contradistinction to the fluid in transudates*; at least they do not change momentarily but only after the change in posture has been maintained for some time. It appears that the cause of this difficulty in respiratory movement in the area of dulness is also due to adhesion of the compressed portions of the lung to the costal pleura, so that a rapid or complete extension of the airless portions of the lung cannot take place.

Tympanitic sounds are found *above the dull area* which is caused by the accumulation of fluid, and in its uppermost margins also by airless portions of the lung, owing to the retraction and decreased tension of the lungs. The sound is *lower* in other cases, also owing to the diminished tension of the pulmonary parenchyma. *The cracked-pot sound appears* in rare cases in the region of the tympanitic area, which can be explained by the fact that the air put into vibration by percussion leaves the bronchi, which are stenosed by the exudate, in short expiratory gasps. Still more rarely do we observe upon percussion of the infraclavicular region Williams's tracheal note, and that only if the extensive exudation which reaches high up still leaves the compressed upper lobe in contact with the thoracic wall.

What has been said holds good for medium-sized exudates. The smallest (less than 300 cc.) do not show any demonstrable dulness, slightly larger ones are plainly recognisable, especially upon light percussion. The demonstration of such small exudates is most successful, as taught by Gerhardt, if at first the lower border of the lung is exactly marked out on both sides from the nipples to the posterior scapular line while the patient is sitting and is then made to assume the lateral position (upon the affected side) supported by the extended elbow. If percussion is now repeated there appears in the axillary line of the affected side, over the original border line, a dull area caused by the lateral accumulation of the fluid, which is usually more movable in smaller exudates. Very massive exudates may eventually show absolute flatness up to the clavicle. The thoracic wall of the affected and also of the healthy side becomes considerably distended. The adjacent organs appear very much displaced. The anterior mediastinum in particular is laterally displaced so that the margin of the dulness extends to the sternal margin of the opposite side.

Vocal Fremitus.—The *vocal fremitus* is *entirely lost* or *diminished in* these dull areas. It may be intensified above the border of the fluid if compressed pulmonary tissue is adjacent to the thoracic wall; but the vocal fremitus is also not infrequently present or intensified within the region of the dulness, when band-like adhesions between lung and costal pleura transmit the vibrations of the voice, and if the tension of the thoracic wall is not too high. In such cases it is permissible even to make use of those islands of intensified vocal vibrations within the area of dulness for the diagnosis of a sacculation and formation of a pouch in the pleural space,

especially if exploratory puncture at various places yields different kinds of fluids—purulent and serous (Gerhardt).

Auscultation will show a different condition of the respiratory murmur according to the size of the exudate—i. e., in considerable masses of exudates *absolute disappearance of the same* or *bronchial breathing,* the latter increasing in intensity towards the upper border of the exudate, will occur. If the lung is not completely compressed by the exudate the bronchial character does not become prominent. Then we hear indistinct breathing or feeble breath sounds. The bronchial breathing shows a metallic character in very rare cases, which as yet cannot be satisfactorily explained.

Ægophony.—The condition of the *auscultated voice* is of diagnostic importance. It is lessened in the dull areas. Bronchophony can be heard at other places also only when compressed pulmonary tissue is adjacent to the thoracic wall, therefore upward, or if the lung is prevented from retracting by bands of adhesions. *Ægophony* is observed more frequently than bronchophony, but only in medium-sized exudates, usually in the neighbourhood of the scapula. The *cause* of this sign is partial compression of the small bronchi, which causes the sound waves of the voice to pass the neighbouring walls without obstruction. Ægophony is most frequently, although not exclusively, met with in pleurisy (rarely in pneumonic infiltrations or in normal thoracic conditions in infancy), so that it should be accorded a certain diagnostic significance. Finally, it may be mentioned that sometimes a *friction sound* might be audible at places in which the inflamed pleural surfaces are in contact, therefore, at the upper border of the dulness; it may also take place in exudative pleuritis. However, this occurs in fewer cases than theory would seem to indicate. The friction sounds occur with more frequency in the later course of pleuritis at the time of resorption of the exudate.

All the other phenomena which cannot be demonstrated by a physical examination should not be made use of in the diagnosis. *Fever, acceleration of pulse, pressure in the gastric region,* etc., are sometimes present, and in other cases absent. They change in intensity and they may be caused by many other pathological processes, so that it is best not to claim any diagnostic value for them. More in immediate connection with the mechanical consequences of a pleuritic exudation, and therefore of greater significance diagnostically, are *small pulse, cyanosis,* and *diminished urinary secretion.* They are the result of the impaired diastole of the heart which is diminished in space in the thoracic cavity. This obstructs the return of blood to the heart, the filling of the arteries becomes deficient, and the blood in the capillaries flows under greater pressure, but slower. The amount of urinary secretion is in part also determined by the exudation in the pleura; it becomes less upon rapid growth of the exudate and much urine is voided at the time of resorption.

Differential Diagnosis.—Pleuritis can usually be diagnosticated with great certainty by reason of the above-described results of physical examination. However, it is usually necessary to consider a number of affections which cause similar symptoms, and exclude them before a positive diagnosis of pleuritis is made.

So long as no dulness is demonstrable, the diagnosis of pleuritis sicca is restricted to the presence of friction sounds. But as the latter some-

times disappear entirely in the course of pleuritis, and at times are not audible in spite of the inflammation of the pleural surfaces, it is possible to mistake pleuritis sicca for other pathological conditions which simulate pleural pains. This is especially the case if the pains occur unilaterally in the thorax, become intensified by inspiration or by sneezing, pressure, cough, etc., thus impairing respiration and making it superficial. Not infrequently we are in doubt whether *muscular rheumatism* or pleuritis sicca is present. Here, above all, manual examination of the muscles is of significance, also the question of painfulness of their fibres upon compression between the fingers, the marked increase of the pain upon motion. Faradization of the muscles may also be employed as a means of diagnosis, because this will result without exception in at least a transitory lessening of the muscular pains. Confounding it with *costal affections,* periostitis and caries, should not occur, because in these cases the restriction of the pains to the ribs and the local examination of the same will at once clear up the diagnosis. The differentiation from *intercostal neuralgia* is more difficult, in fact impossible in some cases, especially because the pleuritic pain may become localized exactly corresponding to the course of the intercostal nerves, and also because the restriction of the same to certain points is not wanting. Besides, the diffusion of the pain over several intercostal spaces, the close connection of the pain with respiration, the anodal application of the constant current which has no effect upon the pain would in such cases decide in favour of the pain being of pleuritic origin. Sometimes the pain in pleuritis is restricted principally to the terminal branches of the lower intercostal nerves in the epigastrium or umbilical region, and then it may be confounded with *gastralgia, ulcer of the stomach,* or even *peritonitis.* If the above-mentioned affections which are accompanied by pain in the thorax can be excluded in cases in which no pleuritic friction sound is audible, a provisional diagnosis of pleuritis sicca may be made. However, such diagnoses are of no value so long as a friction sound cannot be heard occasionally. Friction sounds, however, may be confounded with mucous râles, crepitant râles, etc., as stated above. But in by far the majority of cases it will be possible to distinguish friction sounds from similar acoustic phenomena, as above explained, especially because many years' experience with auscultation renders the ear very acute in detecting friction sounds.

Pleurisy with Effusion.—*Pleurisy combined with exudation* presents eventually no less difficulties in the diagnosis. More or less all those affections are to be considered in this case which show dulness upon percussion of the thorax. Most frequently we are confronted by the question, *Infiltration or exudation?* The usually stated rule that intensified vocal fremitus points to pneumonia and diminished vocal fremitus to pleurisy, is insufficient in the majority of cases, in my experience. The pectoral fremitus is found diminished very frequently, not only in cases of infiltration (when obstruction of the bronchi or simultaneous pleuritis does not permit the theoretically demanded intensification of the same to take place), but also *vice versa,* as we have seen, unchanged in pleuritis or even intensified in rare cases. The following points should be taken into consideration in the differential diagnosis:

PNEUMONIA	PLEURITIS
1. *The affection begins* (at least in the croupous forms of pneumonia) with chills.	1. *Onset* of the affection with moderate or high fever, almost exclusively without chills.
2. Dulness irregular, generally corresponding to the area of the lobes; if the greatest intensity of the dulness is superiorly, this points directly to pneumonia; the same is the case if dulness is absent towards the spinal cord. The dulness in uncomplicated pneumonia is *almost never absolute.*	2. *Dulness* increasing in intensity from above downward and usually decreasing from behind and above forward and downward. In left-sided pleuritis special attention should be paid to dulness in the pleural sinus (in the so-called semilunar space), which is almost without exception absent in pneumonia. Dulness *absolute, offering great resistance on percussion.*
3. If the *pectoral fremitus is accentuated,* it is in favour of *pneumonia,* if diminished, not against it; vigorous coughing may cause an intensified appearance of the previously weakened pectoral fremitus.	3. *Pectoral fremitus* in by far the majority of cases, diminished or *suspended* over the areas of the most intense dulness.
4. *Bronchial breathing,* usually loudest over the areas of greatest dulness. *Bronchophony,* loud at the areas of bronchial breathing, very rarely ægophony. *Crepitant râles,* long lasting, widely diffused.	4. *Bronchial breathing,* principally at the upper border of the dulness, weakest at the areas of greatest dulness, appearing to come from a distance if at all audible. *Bronchophony* less than in pneumonia. *Ægophony frequent.* *Crepitant râles* rare, transitorily in the upper portions of the dulness at which eventually friction sounds.
5. *Displacement* of heart and liver only in excessive extension of the pneumonia and even then only insignificant; neither is the *circumference of the thorax* increased at all, or, as a rule, but very little.	5. *Symptoms of displacement* very marked upon larger exudates. *The circumference of the affected half of the thorax* constantly increased, often considerably so—i. e., by several centimetres.
6. *Sputum* almost always present and rusty.	6. *Sputum,* if present at all, catarrhal.
7. High *fever,* with critical defervescence, herpes, etc., their occurrence and course not being constant, are of not much significance diagnostically, especially because combinations of pleuritis and pneumonia are quite common.	7. *The course of the fever* not characteristic; the fever is usually moderately high, rarely entirely absent; defervescence of the fever gradual, at any rate not critical. Duration of the disease generally much longer than that of pneumonia.

It should be mentioned that both affections, pneumonia (lobar and lobular) and exudative pleuritis, frequently occur combined. If pneumonia has become chronic and bronchiectasis exists at the same time, it is possible, if the large bronchi and the cavities are filled with sputum, that the dulness, diminished vocal fremitus, and the respiratory sounds may simulate sacculated pleurisy, especially because exploratory puncture reveals purulent masses from the cavities. However, the repeated examination of the dull area at various times, the sudden expectoration of large masses of sputum, and eventually abrupt changes of the percussion and auscultation, will clear up the diagnosis.

Mediastinal and Pleural Tumours.—The differential diagnosis between *mediastinal,* respectively *pleural tumours* and exudative pleuritis, is much more difficult, and has been enlarged upon under the discussion of the above-named tumours. It

may be emphasized briefly that in pleuritis the vocal fremitus is diminished, the dull area occupies the lower portions and is restricted to one side, while in tumours when they grow from below upward the dulness extends slowly upward and in an irregular manner on the same side, or eventually, contrary to the condition in pleuritis, extends with tongue-like projections to the upper side to the upper border of the dull area. Besides, the symptoms of compression of nerves, vessels, and œsophagus point decidedly more in favour of mediastinal tumour than they do of pleuritis. However, the diagnosis becomes certain only on exploratory puncture, which should always be made in such cases. If no fluid is aspirated this does not mean that pleuritis may not be present, because not infrequently negative results are obtained on puncture owing to the fact that the needle does not penetrate the costal pleura which has been thickened in consequence of inflammation, or because numerous fibrin flocculi in the exudate prevent the aspiration of fluid. The diagnosis becomes possible in such cases only after repeated exploratory puncture, or after the employment of a larger needle.

Aneurysm.—*Pulsating pleurisy* may be mistaken for an aneurysm if the pulsating pleuritic exudate causes the thoracic wall to bulge out as a circumscribed area. In favour of the latter are the seat of the tumour (usually in the lower portions of the thorax), the absence of vascular murmurs over the tumour, and the increase of size of the latter upon strong expiratory movements. In favour of aneurysm are the well-known changes of the pulse, very pronounced symptoms of compression of intrathoracic vessels and nerves, vascular murmurs, etc.

Peripleuritic Abscesses.—Large *peripleuritic abscesses* situated outside of the costal pleura are very apt to simulate pleuritis. Dulness, respiratory murmurs which appear to be distant, diminished vocal fremitus, and narrowing of the intercostal spaces are common to both conditions. However, the symptoms of compression of the lungs or displacement of the adjacent organs are absent in peripleuritic abscesses in spite of very diffuse dulness. The bulging of the intercostal spaces is essentially restricted to the extent of the dulness, and the fluctuation becomes particularly distinct lower down. In several cases of my observation it extended far into the abdominal cavity. If the abscesses open, all doubt is cleared up, for a sound introduced into the abscess cavity does not enter the pleural cavity as is the case in empyema, and besides, no pneumothorax is caused by the operation. Accumulations of pus below the diaphragm, "*hypophrenic abscesses*," which usually contain, besides pus, air (subphrenic pyopneumothorax), which, however, may contain pus only, are difficult to differentiate from small pleuritic exudates. The lower border of the lungs becomes displaced upon deep inspiration in contradistinction to the condition in pleuritic exudate, and the fluid evacuated by exploratory puncture may contain, besides pus, particles of food, fæces, etc., according to the origin of the hypophrenic abscess. If the fluid comes from some locality below the diaphragm it rapidly drops from the trocar upon inspiration, and if it does not come from that locality it drops upon expiration (Gerhardt). The diagnosis is supported by the demonstration of perihepatitic friction and other peritonitic symptoms.

Tumours of the Liver and Spleen.—The differential diagnosis between *pleuritis and enlargement of the liver and spleen* is of very little practical significance. The enlargement of these organs, except in abscess and echinococcus, is directed downward, and their lower portions and borders are usually easily recognisable as belonging to an enlarged organ. The characteristic symptoms between pleuritis and the two above-named affections of the liver and spleen are generally more of theoretical than of practical value. They will be discussed in the chapter on Affections of the Liver and Spleen.

Hæmothorax.—The differentiation of *hydrothorax* and *hæmothorax* from pleuritis is very often a subject of diagnostic consideration in practice. In hæmothorax the ætiology (trauma, aneurysm, pulmonary gangrene, etc.), the sudden onset, and the simultaneous symptom-complex of internal hæmorrhage (pallor of the skin, etc.), are to be principally considered. The diagnosis becomes certain in this case also by exploratory

puncture. If this reveals blood in the syringe it would not constitute proof of the presence of hæmothorax, because the fluid which is aspirated with the Pravaz syringe is not infrequently purely hæmorrhagic, while exploratory puncture made at another place may produce serum which is not blood-tinged. This fact can only be explained by the supposition that the point of the needle penetrated a blood-vessel of the thoracic wall or of the pleura, causing the aspiration of blood. If we obtain pure blood also in the second or third exploratory puncture which has been done at another point, the diagnosis of hæmothorax becomes established. If, on the other hand, the fluid is only slightly tinged and of a reddish or brown colour, it is a question of hæmorrhagic pleuritis due to scurvy, general hæmorrhagic diathesis, tuberculosis, or carcinoma of the pleura. In the last-named affection the extracted fluid is almost pure blood, dark red to black in colour (compare p. 168).

Hydrothorax.—In many cases it is very difficult to decide whether pleuritis or *hydrothorax* is present. The ætiology (engorgement, Bright's disease, cachexia), the absence of fever, of displacement symptoms, of the friction sound, and of the intercostal bulging, the bilateral occurrence of the fluid, the straight course of the border line of the fluid, the great tendency of the dulness to change with the posture of the patient, almost always admit of a sure diagnosis of hydrothorax. The specific gravity of the fluid in hydrothorax is less (10.15 and below) than that of the pleuritic exudate. There are cases, however, in which the conditions are more complicated. The differentiation is not easy when the dulness is unilateral. This does not occur as infrequently in hydrothorax as is supposed. The cause for this is either that the patient is constantly, or almost constantly, lying on one side, which favours the occurrence of hydrothorax in this half of the thorax, or that the pleural surfaces are coalescent on one side. The latter is to be assumed if a percussory displacement of the lower borders of the lung cannot be demonstrated on deep inspiration on that side which is free from exudate. It is quite obvious that the vocal fremitus is diminished, bronchial breathing and ægophony are audible—in short, most of the physical symptoms are the same in hydrothorax as in pleuritis.

Condition of the Exudate.—If the presence of a pleuritic exudate has been diagnosticated in the above-described explicit manner *the further question suggests itself whether the condition of the same is serous, purulent, hæmorrhagic, or ichorous.* There is no doubt that we are able in most cases to draw conclusions as to the nature of the exudate from the ætiology and accompanying symptoms. A purulent pleurisy should be thought of, for instance, in great remissions of the fever, intercurrent chills, and severe general condition of the patient. Furthermore, if the pleurisy develops in the course of pneumonia, influenza, or pyæmia, if circumscribed œdema of the thoracic wall appears, if the whispering voice is not observed on the affected side, etc. However, all these factors may be absent and yet pyothorax be present. In general all such diagnostic considerations regarding the special composition of the exudate which are derived from clinical symptoms, are nothing but provisional diagnoses. Every physician of experience will admit that in spite of the most careful

consideration of the diagnostic rules in question, he is not infrequently surprised at the result of the *exploratory puncture.* The latter is to be made in every case in which there is a question regarding the character of the pleural exudate. Since the exploratory puncture has been recognised to be an operation entirely devoid of danger and has become a general diagnostic aid, all the diagnostic considerations as to the character of the pleuritic exudate, which were formerly necessary and which proved insufficient in many cases, have become of little consequence. The exploratory puncture gives a quick and certain result. It is only necessary to select dependent places, for in sero-purulent exudates the masses of pus usually sink and upon puncture of the upper portions only a cloudy serous fluid will be aspirated.

The *bacteriological examination of pleuritic exudates* has shown that *serofibrinous exudates,* even if they are due to tuberculosis, *are almost always free from micro-organisms.* Only in isolated cases do they contain *pneumococci, staphylococci, tubercle bacilli,* and even *streptococci* without a purulent condition of the exudate taking place. The so-called idiopathic pleuritis depends almost always upon tuberculosis, as has been proved by animal inoculations. In such cases it is very rarely a question of *rheumatic pleuritides,* that is, such as are caused by the noxa of acute articular rheumatism. If, at the same time, few pyogenic micro-organisms are present in the clear pleural exudate, suppuration will generally occur later. *Purulent exudates* in contradistinction to the serous show large numbers of organisms. Sometimes only one variety is present in empyema, at other times there are several. Streptococci are found most frequently—in about 50 per cent of the cases—less frequently pneumococci. The latter predominate in the empyemata of infancy. Mixed forms, pneumococci and streptococci, tubercle bacilli and streptococci, etc., have often been demonstrated. Rarely also typhoid bacilli. Aside from the demonstration of tubercle bacilli in the exudate all these findings of bacteria are not of much diagnostic significance, at least up to the present time. It is by no means clear in every case how the micro-organism enters the pleural cavity.

PNEUMOTHORAX

The diagnosis of pneumothorax—the accumulation of air in the pleural sac—is one of the most positive diagnoses existing, because it is based upon very marked physical changes in the thorax. In some cases a sudden attack of dyspnœa and cyanosis calls attention to the onset of pneumothorax, in others it occurs unobserved. However, the presence of pneumothorax under no circumstances escapes careful examination, except in the case of a small sacculated pneumothorax, and it is even then a diagnostic error to overlook the condition.

The affected half of the thorax[1] is distended, the intercostal spaces

[1] Pneumothorax is generally unilateral. I have observed bilateral pneumothorax only once, which was caused by the perforation of two phthisical cavities of both upper lobes. Strange to say, the patient lived at least six hours after its occurrence.

appear narrow or bulging, the adjacent organs, heart, liver, and spleen are displaced towards the opposite side. The patient *lies* on the affected side, respiration is accelerated, dyspnœic. The *vocal fremitus is diminished*, due only to the excessive tension of the thoracic wall and the considerable retraction of the bronchi from the thoracic wall.

Percussion shows, providing there is fluid in the pleural cavity, besides air, which later in the course of the disease will be present in the majority of cases, *dulness* to the upper border of the fluid. This dulness will at once be replaced by a high note upon change of posture, owing to the great motility of the exudate. Above the horizontal surface of the fluid the percussion sound is clear. The quality of this sound is usually of the normal pulmonary resonance, *only clear tympanitic*, rarely metallic upon relatively small tension of the thoracic wall.

Tympanitic and Metallic Percussion Sounds.—In case the usual manner of percussion does not show a metallic note the latter may be obtained if percussion is done with the handle of the hammer or with the nail striking the pleximeter in quick taps, at the same time applying the ear to the thoracic wall. The *cracked-pot sound* may be determined in rare cases when a thoracic fistula or larger perforation exists in the lung. A change in the note of the sound upon opening or closing the mouth can be determined more frequently—namely, in cases in which there exists a larger communication between the pleural cavity and the main bronchus (by means of a cavity). *Respiratory change of the note* may also occur, that is, the sound becomes higher in pitch upon deep inspiration. The explanation of this fact, however, has not been found. The change of the note depends in most cases upon the posture of the patient, so that the sound, especially the *metallic sound, becomes lower upon the patient sitting up, higher when in the recumbent posture* (Biermer). The reverse should properly be expected according to the change of the longest diameter of the pleural cavity by the displacement of the exudate when sitting or lying. In fact this condition (the high note when sitting, the low note when lying) occurs sometimes, especially as it seems when the exudate is considerable, while the usual condition is frequently explained in such a way, although not plausibly, that the diaphragm sinks downward when the patient is in a sitting posture owing to the greater weight of the exudate, which causes the lengthening of the largest diameter and a lower metallic note.

Auscultation gives the most important results in the diagnosis. The respiratory sounds are very weak and show nearly always a *metallic* accessory quality, owing to the resonance in the pleural space, also the bronchial respiratory sound and bronchophony. The râles which are formed in the bronchi also have a metallic quality. If some bubbles burst in the bronchial secretion, these give a metallic sound "the falling-drop sound," which is eventually also caused by the falling of drops of fluid from the upper portions of the pleural cavity. Sometimes the heart sounds are metallic in character, but this is seldom the case. I have only observed this metallic sound in a marked manner in sacculated, left-sided pneumothorax once. Of greater importance in the diagnosis than any of the auscultation signs so

far mentioned is, in my experience, the *succussion sound*, the metallic splashing upon the shaking of the patient. *It is a certain indication of the presence of pneumothorax.* If this succussion has also been found in large pulmonary cavities, this occurrence is extremely rare. Personally I have never observed it, in spite of frequent attempts, not even in the presence of the largest cavities. But as the succussion sounds cannot always be produced in pneumothorax, and the physical symptoms of cavities and pneumothorax frequently are and must be the same, the differentiation of both these conditions is not infrequently very difficult. We will therefore consider the *differential diagnosis* of these conditions.

Differential Diagnosis.—If it is a question of *an extensive free pneumothorax*, confounding it with other conditions can easily be avoided. At most those cases should be taken into consideration in which the high position of the left lung (possibly caused by shrinking) displaces the diaphragm and with it the stomach, high up in the thoracic cavity. In this case it is possible that the extensive tympanitic sound, the absence of respiratory murmur, and the eventual occurrence of metallic after-sounds in the lower half of the thorax, and finally even the succussion sound arising in the stomach, may lead to a wrong diagnosis of pneumothorax. However, in such cases the enlargement of the left half of the thorax is not present, and other sounds manifest themselves besides the metallic sounds. The formation of these other sounds coincides with the movements of the stomach and they can be easily recognised as gastric sounds, by the fact that their origin and course are independent of respiration. Lavage of the stomach naturally clears up the situation at once. About the same conditions prevail in *diaphragmatic hernia*, which is a rare condition, in which portions of the stomach and colon enter into the thoracic cavity, by perforation of the diaphragm. The metallic respiratory sounds here also depend mostly upon peristalsis. Filling and emptying of the stomach by means of the stomach-tube will produce a change in the respiratory murmurs and percussory condition, which are dependent upon the stomach, and the tube which passes the œsophagus will find an obstacle at the cardia owing to the latter being displaced.

Pyopneumothorax Hypophrenicus.—In accumulations of pus and air upon the right side between the diaphragm and liver, owing to perforation of an air-containing abdominal organ, a so-called *hypophrenic pneumothorax* may give rise to mistakes. Similar air-containing abscesses may also occur in the left-sided excavation of the diaphragm, especially in consequence of perforating gastric ulcers. The decisive factors for the diagnosis in such cases, according to von Leyden, who recently called attention to these hypophrenal abscesses, are the ætiology (preceding ulcer of the stomach and of the duodenum, typhlitic processes, perforation of the appendix, abscesses of the liver and spleen, paranephritis, peritonitis, tuberculous cavities, and finally, the effect of trauma upon the diaphragmatic region). Furthermore, absence of cough and sputum, indistinct signs of increased pressure in the thoracic cavity, slight displacement of the heart in contradistinction to marked downward displacement of the liver, absence of bulging of the intercostal spaces, preservation of vesicular breathing downward to the margin of the abscess, and displacement of the vesicular murmur upon deepest inspiration far beyond this margin downward. No respiratory murmur, only amphoric breathing and succussion sounds can be observed in the lower portions of the thorax corresponding to the position of the abscess. Measuring of the air pressure at the cannula reveals increased pressure upon inspiration in comparison to the reverse condition upon the presence of air in the pleural cavity.

Differential Diagnosis between Pneumothorax and Large Cavities.—The above-named pathological conditions which may simulate pyopneumothorax are generally very rare. On the other hand, the differential diagnosis frequently wavers between *sacculated pneumothorax* and *large*

cavities. I wish to remark in advance that the sacculated pneumothorax is a rare condition, compared with the frequency of large cavities. It should therefore be considered a rule, if no positive reasons point against the presence of cavity, that it is always advisable to think of a cavity and not of a sacculated pneumothorax. The following symptoms are in *favour of pneumothorax*: expansion of the thorax, bulging of the intercostal spaces, displacement of the heart, liver, and spleen. All these symptoms are eventually not present, or but slightly marked in circumscribed pneumothorax, in which the exhaled air remains within a small space which is limited by adhesions. *On the other hand, the most important sign, the succussion sound, will also be present in sacculated pneumothorax, while it almost never occurs in cavities.* The other symptoms of pneumothorax, metallic breath sounds and râles, the metallic note yielded on plexímeter percussion, are the same in both conditions, also the change of note upon sitting and lying. If the cracked-pot sound can be determined, it points decidedly more to cavity, because its occurrence in pneumothorax presupposes peculiar conditions regarding the situation of the perforation. This holds good still more upon the change of note on opening and closing the mouth. *Determining above all in a diagnosis of cavity formation are the following signs: If the vocal fremitus is intensified over cavities, or at least not diminished, as is frequently the case; if the intercostal spaces are sunken; if profuse ringing râles, which appear near the ear are present, and if vigorous cough and expectoration of sputum produce rapid changes in the auscultatory symptoms.* It is of no diagnostic value that the cavities are usually located in the apex of the lungs and circumscribed pneumothorax at other places, because the latter may occasionally also appear in the uppermost portions of the thorax; and, on the other hand, phthisical cavities, and, more so, cavities resulting from pulmonary abscess, gangrene or bronchiectasis, are also found in the more dependent portions of the lung.

Diagnosis of Special Varieties of Pneumothorax.—But if the diagnosis of pneumothorax is established, the second question to decide will be, whether *a closed, open, or valvular pneumothorax* is present, i. e., whether upon aspiration the air cannot enter or leave the pleura, or whether this occurs without obstruction, or whether air may enter upon inspiration but is prevented from leaving upon expiration (by special anatomical conditions of the perforation or by closure of the affected bronchi). The decision as to which of these forms of pneumothorax is present in part determines the therapy and therefore is of practical importance.

Open Pneumothorax.—The following signs are seen in *open pneumothorax*: Change of note upon opening and closing the mouth, "fistulous murmurs," "metallic bubble-bursting," "water-whistle murmur," i. e., a metallic gurgling which is produced when air bubbles ascend upon inspiration through the fistula which is situated below the level of the fluid into the pleural air space and burst with a metallic note. An open fistula is also indicated by the periodical occurrence of attacks of coughing, which depend upon the posture of the body, accompanied by expectoration of large quantities of sputum (usually thin and purulent fluid), a symptom, how-

ever, which may be absent in spite of the open communication of the bronchial air with the pleural cavity.

It has often been claimed that the condition of the diaphragm and mediastinum, which are displaced in closed pneumothorax and not in the open form, is a distinctive symptom between open and closed pneumothorax; but according to recent clinical anatomical experiences and to Weil's clinical investigations, this is not the case, because a dislocation of the heart and diaphragm exists in both conditions. *The degree of displacement only* is of any diagnostic value, inasmuch as very marked widening of the thorax and excessive bulging of the diaphragm downward, point to closed pneumothorax, or to the valvular variety.

A positive decision in the cases in question is obtained by the direct measuring of the air pressure existing in the pleural cavity. If a trocar, the side-arm of which is connected with a manometer, be introduced into the pleural cavity, the average pressure in open pneumothorax is equal to the outside atmospheric pressure, while it is higher in closed pneumothorax. The chemical examination of the gas in the pleural cavity is of less use in the diagnosis, and this gas is chiefly characterized by its deficiency in oxygen (according to Ewald, carbonic acid less than 5 per cent in the pleural cavity shows open, above 10 per cent closed pneumothorax). If there are two fistulas, one in the pulmonary pleura, the other in the thoracic wall, this fact can be easily determined if the thoracic fistula is temporarily closed with the hand after a deep expiration of the patient, who is then made to inspire deeply. If then the hand is taken away from the exterior fistula during the following expiration a current of air will escape.

Closed Pneumothorax.—A *closed pneumothorax* is to be diagnosticated, aside from the above-named conduct of the gas in the pleural cavity, if the displacement symptoms are excessive; if the thoracic wall is considerably bulged and the circumference of the thorax seems considerably increased on the affected side (up to 6 cm.); furthermore, if the respiratory sound is entirely absent, which is not conceivable in open pneumothorax, in which the air freely enters and leaves through the fistula. Of course it is possible, and this is the usual occurrence, that the respiration and the râles which arise in the lung are also audible in closed pneumothorax and have a metallic addition. While the height of the note of the metallic percussion sound changes upon sitting and lying of the patient, in the previously described manner, it remains unchanged upon opening and closing of the mouth, in every instance of closed pneumothorax. The absence of vocal fremitus corresponds to the high-grade tension of the thoracic wall in closed pneumothorax.

Valvular Pneumothorax.—The symptoms of *valvular pneumothorax* can be derived from those of the open and closed forms. It is at first a question of physical signs of an open pneumothorax. The air enters with each inspiration without obstruction, but is retained upon the following expiration. This very soon causes the occurrence of very high pressure in the pleural cavity, which is still further increased by the occurrence of a pleuritic exudate. Now there will be no more entrance of air to the pleural cavity upon inspiration and the symptoms of an open pneumothorax are replaced by those of a closed pneumothorax. The temporarily occur-

ring, plentiful (mouthful) expectorations of thin, purulent sputum are absent in valvular pneumothorax, even as long as it remains open, in contradistinction to the condition in the open form.

The *closure* is sometimes brought about, especially in the beginning of the process, by the gas pressure, and is simply "mechanical." At other times it is caused by coalescence of the perforation, an "organic," lasting one. This distinction is of importance with reference to the effect of therapeutic punctures. If air or fluid is evacuated by puncture in the *organically closed valvular pneumothorax* the pressure in the pleural cavity will be permanently reduced because the pneumothorax remains closed the same afterward as before. The closure, which is caused by the greatly increased pressure in the pleural cavity, ceases in consequence of the puncture in the mechanically closed valvular pneumothorax, thus reproducing an open valvular pneumothorax. Now air again enters upon inspiration, and, if the perforation be situated below the level of the fluid, it will enter through the latter into the pleural cavity. This produces *an inspiratory metallic fistular sound* which, in contradistinction to the fistulous sounds in permanently open pneumothorax, is only inspiratory and of short duration.

In order to form a positive opinion in regard to mechanical or organic closure of valvular pneumothorax, it is necessary to measure the gas pressure in the pleural cavity according to the principles laid down by Weil. We will then find *that by evacuation of air by means of puncture, the gas pressure in the pleural cavity remains equal to the atmospheric pressure or becomes negative and remains so in the organically closed pneumothorax. In mechanically closed pneumothorax, a positive pressure of the former extent soon becomes re-established after an initial decrease of the pressure.* It may finally be mentioned that it is necessary, in order to complete the diagnosis, to obtain exuded fluid by puncture so as to become positive regarding its nature. It is sometimes serous, at others purulent after it has been present for some time. It is possible that occasionally it may contain tubercle bacilli, from which finding we may conclude that tuberculous processes in the lung are the source of pneumothorax.

Ætiology as a Diagnostic Feature.—This leads us to speak of the diagnosis in its *causal* relation in different cases. It is obvious that the ætiology of the case is at once evident in pneumothorax which is brought about by external force. *Pulmonary tuberculosis* gives rise to the occurrence of pneumothorax in by far the majority, in at least nine tenths of the cases, and in this case it is usually small cavities which perforate into the pleural cavity during a rapid course of the tuberculous process before adhesions of the pleural surface take place. A pneumothorax is much rarer brought on by other processes in the lung which are accompanied by ulceration: bronchiectasis, pulmonary gangrene, pulmonary abscess, etc.; furthermore, internal trauma, especially the swallowing of pointed foreign bodies, or the bursting of emphysematous alveoli after vigorous exertions—crying, coughing, etc. In the same manner it is an unusual occurrence for it to be produced by ulcerative processes in the stomach and intestine. The perforation of the air-containing organs of the abdomen usually leads to hypophrenic pyopneumothorax. It is possible in isolated cases that the presence of the gas-producing micro-organisms in a pleuritic exudate is the cause of the pneumothorax, which has been recently proved by Levi, by cultivation of a gas-producing bacillus from a serous exudate.

The diagnosis of hæmothorax as well as that of hydrothorax has been considered in the discussion of the differential diagnosis between pleuritis and these conditions. It therefore only remains to take up the diagnosis of neoplasms of the pleura.

NEOPLASMS OF THE PLEURA

The diagnosis of carcinoma or sarcoma of the pleura is difficult under all circumstances. It succeeds sometimes and attains a certain degree of surety if various diagnostic factors occur unitedly in one and the same case. The clinical symptoms are usually ambiguous; the picture is sometimes that of a simple pleurisy, in others, that of a mediastinal tumour combined with pleural exudation. ***The fluid obtained by puncture is in most cases hæmorrhagic, dark to black-red.*** **(This was the case in all instances which were observed by me.)** ***It contains remarkably abundant numbers of fatty degenerated cells and large endothelium with vacuoles,*** **which are apparently due to degenerative processes in the cancer cells. If we succeed in finding desquamative tumour particles, the microscopical examinations of which reveals their carcinomatous structure, this will render the diagnosis of pleural carcinoma certain. If the carcinomatous masses are located at the costal pleura the exploratory puncture may prove negative, the needle becoming embedded in the tumour mass. To succeed, a large needle should be employed which will enter as far as the exudate, and it will then usually produce a purely bloody fluid. The thorax is usually enlarged on the affected side, in other cases retracted laterally, owing to the atrophy of the pleural surfaces produced by the malignant growth.**

Most of the above-named symptoms are not at all pathognomonic of pleural cancer. They are found in mediastinal tumours as well as chronic, especially tuberculous pleuritis. The symptoms collectively must determine the diagnosis. It is supported by the rapidly advancing cachexia of the patient, the demonstration of carcinoma in other regions of the body which are easier of access for examination, and by the occurrence of metastatic glandular tumours. ***It becomes certain, if we succeed in finding carcinomatous masses in the pleural exudate, or if the carcinoma extends from the costal bones inward upon the costal pleura as I have seen occur, or vice versa proliferating from the latter outward, forming a visible and palpable tumour of the thoracic wall.***

Echinococcus of the Pleura.—Finally, I will mention the occurrence of *echinococcus* in the *pleural cavity*. The signs at first are those of a growing pleural exudate, thoracic pain with dyspnœa, displacement of the heart, liver, etc. The distention of the thorax is not uniform, the course of the disease afebrile, similar to the development of a neoplasm of the pleura. If fluctuation occurs in an intercostal space, an uncomplicated solid neoplasm is to be excluded, and exploratory puncture should be made. If this reveals a water-white fluid *which contains no albumin*, this will exclude hydrothorax and a pleuritic exudate and the diagnosis of echinococcus becomes certain; but any doubt will disappear if we succeed in demonstrating *hooklets* and *portions of the cystic wall* microscopically in the aspirated fluid. It is possible that in cases in which suppuration exists in the pleural cavity besides the echinoccocus, that the parasite remains undiagnosticated in spite of the exploratory puncture unless this is repeated, and then reveals the last-named particles which are diagnostic.

DIAGNOSIS OF AFFECTIONS OF THE ABDOMINAL ORGANS

Preliminary Diagnostic Remarks.—While percussion and auscultation are prominent in the examination of affections of the thorax, of the heart, and of the lungs, these two methods of examination are less valuable in the diagnosis of *diseases of the abdomen. Palpation in these cases is of the greatest importance,* and its accuracy depends in not a small number of cases upon the finesse of the technique as a last resort. Few general rules can be given in this respect. The main point is practice and experience. The first rule is to put the abdomen of the patient into a state of the greatest relaxation. The patient is made to assume the dorsal decubitus, the head should be pressed firmly against a cushion, and he is asked to respire deeply with the mouth open. Medium deep respiration accomplishes more in some instances, because some individuals draw in and make the abdominal walls tense upon forced respiration. The examination is almost always facilitated if the patient is made to flex the thighs on the abdomen. In some cases it is advisable to place the sacrum high, at other times the examination in the lateral posture is necessary. *The physician should never palpate while standing, but in order to avoid any disturbance of his own body weight, he should sit on the edge of the bed. The hands should be placed flatly on the abdomen and the finger tips should not be bent. Strong impression of the fingers should be avoided. Palpation should commence lightly and should be gradually increased to stronger pressure—generally the latter is not at all necessary.* If this manner of palpation does not give results, *a second examination should be preceded by evacuation of the bowels by a purge. In all cases which are difficult of diagnosis chloroform is recommended.*

As the most important secretions of the body take place in the abdominal organs it is obvious that in affections of the latter, alteration of the secretions in a physical and chemical respect occur. *The exact examination of the gastric juice, of the bile, the urine, etc., and furthermore, the analysis of functional disturbances of the abdominal organs in general, forms a most important part in the diagnosis of abdominal affections.*

The researches of the last decade have been devoted with much energy to this department of pathology, so that to-day there is no question but that we have made progress in the diagnosis of the various abdominal affections and that we are able to diagnosticate more easily and with greater certainty.

DIAGNOSIS OF AFFECTIONS OF THE LIVER

INTRODUCTION

Anatomical Preliminary Remarks.—Only a few points which are especially important in the pathology shall be emphasized in the *anatomical* structure of the liver. The liver is supplied with a connective-tissue (Glisson's) capsule, from which continuations enter the interior of the liver with the vessels. The connective tissue separates the lobules of the liver although imperfectly (interlobular connective tissue), and extends also as fine fibres into the interior of the lobules in a radial, lattice-like arrangement (intralobular). The interlobular connective tissue contains the finer branches of the portal veins, hepatic veins (sublobular veins), and of the hepatic artery, which branches out encircling and nourishing the interlobular ramifications of the portal vein and of the hepatic veins as well as of the bile ducts. The veins which issue from the arterial capillaries enter into the interlobular portal-vein branches (interlobular veins) or the beginning of the capillaries of the portal vein. These latter extend into the hepatic lobules and enter into the central veins, the roots of the hepatic veins, which are situated in the centre of each lobule. The interlobular connective tissue contains, between the individual lobules, besides the above-named vessels and bile ducts, nerves and lymph vessels, which encircle the portal ramifications and enter the interior of the respective lobules extending around the capillaries (perivascular lymph channels) and which pass out of the lobule with the central vein.

Liver Cells (Glands); Biliary Capillaries.—The *liver cells* are inclosed between the portal-vein capillaries in the interior of the lobules. Each two of them are separated by a biliary capillary, while the blood capillaries extend over their borders so that bile and blood capillaries never are approximated but are always separated by a liver cell or by portions of one. It seems that the intercellular bile-duct capillaries are in connection with the finest lateral branches, and these with vacuoles, which are situated in the liver cell. The arrangement of the gland cells (liver cells) and of the canal system of the liver corresponds with certain modifications, it is true, to the construction of a tubulous gland. It is very peculiar here, that the glandular lumen —i. e., the bile-duct capillaries—are only bordered by two cells, while the lumen in other tubulous glands is inclosed by several (six and more) cells in the transverse section.

Portal Vein and Anastomoses.—It is of particular importance for the comprehension of the consequences of an impaired portal circulation in certain diseases of the liver to observe the origin of the portal vein as well as the anastomosis existing between the portal vein and the vena cava. It is well known that the portal vein receives the venous blood from the chylopoietic organs of the abdomen—i. e., stomach, intestine, pancreas, and spleen (*not* from the kidneys, urinary bladder, and sexual organs)—and carries it to the liver, in which the capillary dissolution of the portal vein and the transference of its blood into the hepatic veins and the inferior vena cava takes place. The branches which go to make up the portal vein are the *superior gastric*, the *vena lienalis* (with its branches vena gastro-epiploica sinistra, pancreatica, and often the vena mesenterica inferiora), *vena mesenterica superior* (with its branches vena intestinales ileo-colica, colica dextra et media, gastro-epi-

ploica dextra), and the *pancreatico-duodenalis*. As soon as the deflux of the portal blood is impaired by pathological processes in the liver, engorgement occurs in the contributing branches of the portal vein, therefore in the stomach, intestines, spleen, and in the peritonæum (ascites). The blood gradually also advances into the communicating ducts which exist between the portal vein and the inferior vena cava.

Such anastomoses are: 1, *the communication between the superior gastric with the diaphragmatic and inferior œsophageal veins*, which return the blood to the azygos vein and then to the vena cava; 2, *the hæmorrhoidal plexus, which consists of the median and inferior hæmorrhoidal veins*, which return their blood to the hypogastric and inferior vena cava and the superior hæmorrhoidal vein, which returns its blood into the inferior mesenteric vein and therefore into the portal vein; 3, the communication between the portal vein and the vena cava is brought about by *venous plexuses at the outer surface of the peritonæum*, the branches of which return their blood in part into portal branches and in part into the inferior cava through the lumbar, vesical, and hæmorrhoidal veins; 4, *the communication between the accessory portal branch with the vena cava*, branches off from the trunk of the portal vein extending into the suspensory ligament adjacent to the ligamentum teres and passes externally in the neighbourhood of the umbilicus into two venous twigs which communicate with the epigastric vein and the internal mammary vein.

In those cases in which the passage is obstructed for the deflux of the portal-vein blood into the liver, the blood flows into those communications, thus providing a sufficient outflow of the portal-vein blood to the heart with evasion of the liver. The anastomosed venous branches then greatly distend, according to the intensity of the obstacles and the overengorgement of the blood, which is either externally visible as varices ("hæmorrhoids," "caput Medusæ"), and is diagnostically important, or gives rise to dangerous internal hæmorrhages (compare the chapter on Cirrhosis of the Liver, p. 180) upon the rupturing of the distended venous branches.

The *function of the liver* is very complicated and many-sided. Not only does it produce a specific secretion (*bile*), but it plays a very important part, which has only in recent years been sufficiently appreciated, in the *metabolism*, inasmuch as the carbohydrate economy and the transformation of albuminous substances is primarily governed by the liver, aside from the furtherance of fat resorption by the bile.

I. The *carbohydrates* introduced with the food are resorbed in the digestive tract as dextrose. If the resorption is to some extent considerable, we should expect that a great portion of the sugar leaves the body through the kidneys unabsorbed, because it has been shown that, if more than 0.2 per cent of sugar circulates in the blood at one time, the surplus is not assimilated by the organism but excreted as a foreign body in the urine. But as normally no sugar is secreted by the kidneys, we must assume that there is some kind of an arrangement in the economy which allows a regulation of the sugar contents of the blood to be about 0.2 per cent. Such a regulatory function, we know, is performed by the liver. The grape sugar, which is resorbed by the digestive tract, is forwarded through the portal vein to the liver and is here changed, by the reunion of a larger number of sugar molecules with the elimination of water, into glycogen—10 grape sugar $10C_6H_{12}O_6 - 10H_2O =$ glycogen $C_{60}H_{100}O_{50} = 10C_6H_{10}O_5$—and stored. Upon this reserve fund, animal starch, there is drawn according to requirement, as soon as the amount of sugar in the blood decreases, owing to its being consumed (in such a manner that the glycogen in the liver is changed back into sugar by a diastatic ferment contained in the blood and enters the circulation), but always only the amount required in the economy of the body for the time being, respectively the amount by which the sugar in the blood has decreased below 0.2 per cent owing to use. It may be taken for granted that the liver cells are the source for the production of glycogen, as about in twelve hours after sugar or starchy nourishment has been introduced into the economy they contain peculiar shining, coarse flakes, which are proved to be glycogen as they stain brown upon the addition of a solution of iodine and iodide of potassium; the liver cells are equally to be regarded as the precise regulators of the normal contents of sugar in the blood. Other sources of glycogen are, besides the carbohydrates, the albuminous bodies (especially their N-less atom complexes which are liberated by decomposition), and probably also the fat which very likely, before its employment as

fuel, is always first transformed (also in the liver) into glycogen. Severer disturbances of the function of the liver, therefore, produce glycosuria, either because more sugar is formed from the amount stored in the liver and exported, owing to the disturbances, or—the usual case, especially in permanent glycosuria—less sugar is transformed into glycogen.

II. The function of the liver is of great importance. Besides the assimilation of carbohydrates, it is also concerned in the nitrogen metabolism.. It can be assumed, by reason of chemico-physiological facts, that in the disintegration of the albumin bodies there occur in the tissues various amido acids (glycocoll, leucin, tyrosin, asparaginic acid) as split products, and these in the further course of decomposition furnish ammonia. It is especially the *lactic-acid ammonia* which may probably be regarded as the main product of albumin decomposition which continually flows into the liver in small quantities. Here it is transformed into carbonate of ammonia, and the latter, as was proved by the investigations of von Schröder, transformed into urea by the activity of the liver cells with the withdrawal of water (probably with the formation of carbamate of ammonia).

CH_3	CH_3	becomes by oxidation	$CO<^{ONH_4}_{ONH_4}$	becomes by withdrawal of water $-H_2O$	$CO<^{NH_2}_{ONH_4}$	becomes by further withdrawal of water $-H_2O$	$CO<^{NH_2}_{NH_2}$
CHOH	CHOH						
COOH	$COONH_4$						
(Sarcolactic acid.)	(Lactate of Ammonia.)		(Carbonate of Ammonia.)		(Carbamate of Ammonia.)		(Carbamid, Urea.)

As yet we do not understand the manner in which this formation of urea in the liver cells takes place; it may be that it is brought about under the influence of a ferment, as appears from recent investigations.

If these assumptions are correct, which, according to results of experiments obtained so far, are at least probable, it is to be expected that the formation of urea suffers in far-reaching disturbances of the function of the liver—i. e., the urea is excreted in lesser quantities, the other nitrogenous substances, in particular the primary substances of urea, in increased amounts. As a matter of fact, numerous examinations of the urine of patients afflicted with hepatic disease, in whom a marked reduction of the function of the liver could be supposed (thus in cirrhosis of the liver, acute yellow atrophy of the liver, and acute phosphorous poisoning), have shown that the urea in the urine had more or less decreased in such cases, while the *amount of ammonia in the urine* had increased, and the more so, the more pronounced the destruction of the liver cells was found in the individual case. However, this increase in the amount of ammonia could also be brought about in a different manner than by a reduced activity of the liver in those cases of severe affection of the organ —namely, by an increased acid formation in the organism. We know that a consequence of this fact is the binding of ammonia at disposal before its transformation into urea; and thus an increased elimination of ammonia in the urine would be natural as soon as an abnormally large amount of acid products is formed in the body. In fact, Münzer succeeded, by the administration of sodium bicarbonate, in reducing the ammonia in the urine to more than one half in a case of acute phosphorus poisoning. But nevertheless, the withdrawal of ammonia by acids does not, in my opinion, explain all facts in this difficult, not yet quite clear chapter of metabolism. Neither is, according to recent investigations, the occurrence of *leucin and tyrosin in the urine*, in acute yellow atrophy of the liver, to be explained in such a manner that these substances, introduced into the liver as primary stages of urea, are not transformed here any further into ammonia and urea. The splitting off of ammonia from amido acids should not at all be considered as a normal function of the liver, because in birds with extirpated liver, leucin and tyrosin do not occur in the urine, but, if such creatures are fed on these substances, they are excreted as ammonia. For further particulars, see Acute Yellow Atrophy of the Liver, p. 176.

Résumé.—Summarizing the results of investigation gained thus far, it may be assumed as certain that the liver, although not the only, yet is a very important place of urea formation, and that, under the action of the liver cells, the main product of albumin decomposition which reaches the liver, ammonium lactate, respectively

ammonium carbonate, is transformed into urea. If affections of the liver prevail which lead to the supposition of an impairment of the urea-forming function of the organ, the excretion of urea in the urine may appear decreased, but only when the shortage of destroyed portions is not replaced by compensatory activity of the remaining glandular tissue of the liver which is still able to functionate, and this appears to be largely possible.

The excretion of uric acid in the urine appears to be altered *in* quite a number of *affections of the liver*; in general it is increased. This is difficult to explain under all circumstances. While mammals excrete more *urea* upon ingestion of the most varying N-containing substances, especially on ingestion of organic ammonium salts, of amido acids, and especially also of uric acid, the substances mentioned, therefore, are partly transformed, as we have seen, under the influence of the activity of the liver, into urea. Birds will, under all circumstances, furnish uric acid as end product of the N-containing substances administered (even upon administration of urea or ammonium carbonate). We may therefore consider it demonstrated to-day, that the *liver* is the place of formation of uric acid in the alvine organism. For, if the liver is extirpated in birds, the largest quantity (50 to 60 per cent) of nitrate of urea does—as is shown by Minkowski's excellent investigations—not appear any more in the form of uric acid in the urine, but as ammonium lactate, which, therefore, is split off outside of the liver, and which may be considered to be the material for the uric-acid synthesis which is possible in the bird only if the activity of the liver has been sustained. The formation of uric acid in *mammals* is different. As has been previously stated, uric acid introduced per os, is transformed into urea in mammals, and the question is justified why not only urea, but also uric acid besides, is excreted in the organism of the mammal. This cannot be explained satisfactorily up to the present time. This much is an ascertained fact, that uric acid in mammals is not formed, as was believed formerly, as a less oxidized by-product in the transformation of the albumin of nutriment, but is formed in all tissues from the nuclear constituents of disintegrating cells (nucleins), especially of decaying leucocytes. Feedings with large quantities of pure nuclein increase the excretion of uric acid considerably, as does the administration of nutriment rich in nuclein (liver, thymus). The nucleins furnish upon transformation so-called xanthin bases (especially hypoxanthin and xanthin), which upon sufficient oxidation change into uric acid, which is quite comprehensible according to the composition of these substances (hypoxanthin $= C_5H_4N_4O$, xanthin $= C_5H_4N_4O_2$, uric acid $= C_5H_4N_4O_3$). It was now assumed that, if the uric acid which is thus formed, is introduced into the kidney without passing the liver, it will appear as such (uric acid) in the urine, while uric acid, which reached the liver from the spleen and the lymphatic system of the digestive tract in general, was transformed into urea in the liver. But the correctness of this hypothesis is contradicted by the fact already mentioned, that the administration in the food of substances rich in nuclein (for instance, calf's thymus), does cause a considerable increase of the uric acid excreted in the urine. Besides, a transformation of urea into uric acid through the activity of the liver is at the outset unlikely, according to what we have stated regarding the negative action of uric acid to leucin and tyrosin. If, nevertheless, in various affections of the liver (hepatic cirrhosis, acute yellow atrophy of the liver, etc.), an increase of uric acid and xanthin excretion, although by no means a considerable one, has been determined, it is probable that it can be explained by the disintegration of liver cells which is associated with these diseases—i. e., by the entrance of materials rich in nuclein into the decomposition of substances.

III. *Bile Secretion and its Disturbances.*—The specific secretion of the liver cells is *bile*, which is of importance for the *absorption of fat* in the intestine; for, upon the omission of bile at least one half of the fat in the intestine is no longer absorbed. After the neutral fat has been split by steapsin—i. e., the fat-splitting enzyme of the pancreatic juice—into glycerin and free fatty acids, and the latter, transformed into soaps by the alkalies of the intestinal juices and, entering as soluble soap molecules among the unchanged molecules of the neutral fats, have caused the separation of the fat into smallest particles, "emulsifying," now the resorption of the fats into the lymph channels is promptly consummated through the action of the bile. How this is done is by no means clear. It is probable that the bile, apart from the favour

ing of fat emulsion by its contents of sodium, exerts a specific influence upon the intestinal epithelia, and, possibly, also upon the musculature of the intestinal villi, owing to which fact these portions of the intestinal wall are stimulated in a plentiful manner into a resorption of the fat. It seems that the secretion of bile, similar to the secretion of urine, also serves to eliminate products of metabolism from the body. The quantity of bile which is daily secreted amounts to no less than one kilo, so that the omission of bile deflux into the intestine may possibly contribute to the dryness of the fæces in jaundice. The most important constituents of bile are, as is well known, *bile acids* (held by sodium), *cholesterin* and *bile pigment* (*bilirubin* and biliverdin). Biliverdin is obtained by oxidation from bilirubin, upon more powerful oxidation, a blue, then a violet, and, finally, a reddish-yellow pigment (choletelin), transformations which form the basis of the well-known Gmelin reaction (overlaying of nitrous-acid-containing nitric acid with the bile-pigment-containing fluid). Both the bile acids as well as the bile pigments are formed in the liver by the liver cells. For the constriction of the hepatic duct or of the ductus choledochus causes an accumulation of cholates and bile pigment in the blood, whereas, after extirpation of the liver (in birds), bile constituents cannot be demonstrated either in the blood or in the urine. The genesis of the bile acids has not as yet been determined, but so much more certain is the origin of the bile pigment from the blood pigment. If, namely, hæmoglobin is injected into the blood, the formation of bile pigment in the bile rises enormously. The same is the case if the hæmoglobin, for some reason or other, separates from the stroma of the red blood corpuscles (thus after the injection of cholates, after extensive burns of the skin, after intoxication with hydrogen arsenide, toluylendiamin, with morchella, etc.), inasmuch as then the liberated Hb, entering the liver in the circulation, is transformed into (albumin and) hæmatin. The latter, splitting off iron and absorbing water, passing into bilirubin:

(Hæmatin) $C_{32}H_{32}N_4O_4Fe + 2H_2O - Fe = C_{32}H_{36}N_4O_6$ (Bilirubin)

In the intestine, then, the bilirubin is reduced, by the always present bacteria, to ***hydrobilirubin*** (*urobilin*), which is partly excreted with the fæces, partly resorbed. A small part of the restored urobilin is again secreted in the bile, another part passes into the urine, and here can be easily demonstrated (on addition of zinc chloride solution and ammonia after previous shaking of the urobilin with chloroform) by the green fluorescence of the fluid.

Urobilin Jaundice; Urobilinuria.—If the urine contains an increased amount of urobilin without bilirubin being demonstrable, and the skin appears more or less intensely yellow, we speak of "*urobilin icterus.*" This form of icterus is not very rare, according to my experience. I have seen cases in which the skin showed a very marked icteric colour for months and even for years, without the appearance of even traces of bilirubin in the urine. The obvious conclusion that the yellow colour of the skin was caused by a deposition of urobilin in the same, is not correct, inasmuch as, according to my investigations, and the observations of others, the substance which turned the skin yellow was exclusively bilirubin in such cases.

The *explanation* of the occurrence of the combination: jaundice of the skin by bilirubin and urobilin excretion in the urine (without bilirubin), of "urobilin icterus," is deficient as yet, as we shall see. Easier of explanation is the excretion of abundant quantities of urobilin in the urine without the simultaneous jaundice of the skin. This simple urobilinuria is found in the most varying pathological conditions, especially in the course of infectious diseases (in some more marked than in others, especially in pneumonias), in certain intoxications, on resorption of large blood extravasations, and in the stage of decline of a retention icterus. In the latter case, after removal of the bile-duct occlusion, so large a quantity of bile reaches the intestine at one time that a considerable transformation of bile pigment into urobilin in the intestine and resorption of large quantities of urobilin takes place, and their natural consequence is marked urobilinuria. The destruction of numerous erythrocytes plays a part as an ætiological factor both in the excretion of much urobilin in the urine in infectious diseases and some in intoxications and in the resorption of large blood extravasations. But, as D. Gerhardt observed recently that, in cases of

icterus with complete occlusion of the bile ducts and with extravasations, large quantities of urobilin can be demonstrated in the urine, it can scarcely be doubted that in these cases the intestine does not take any part in the formation of urobilin, but that this latter substance is produced directly from the hæmoglobin. This is the more probable because in such cases there appears in the urine, besides urobilin, another pigment, hæmatoporphyrin, which forms, besides urobilin, during the artificial reduction of hæmoglobin, while it cannot be produced from bilirubin. Regarding those cases in which, besides an exquisite urobilinuria (without simultaneous excretion of bilirubin in the urine), a jaundice of the skin by bilirubin prevails ("urobilin icterus," or, rather, "icterus with urobilinuria"), it may be assumed that in these cases an affection of the liver and of the bile ducts is followed by a resorption of moderate quantities of bile, which, although able to turn the skin yellow upon their deposition in the tissues, are not sufficient to appear in the urine, because this requires a more marked saturation of the blood with bilirubin than is necessary to jaundice the skin. These moderate quantities of bilirubin gradually colour the skin a more marked yellow, although not a deep and dark yellow, as is the case in the common retention icterus, and must be transformed into urobilin if this substance is to be excreted in large quantities in the urine. But *when* this happens, it is absolutely impossible to decide; all hypotheses which have been proposed in this respect are unsatisfactory, in my opinion, and it is probably better at present to forego a positive opinion on this question.

Icterus.—Disturbances in the flow of bile and resorption of the same in the blood characterize the condition, which is designated as *icterus*. The production of bile takes place in the liver, the function of which organ, as we have observed, is quite varied. The bile that is produced flows into the biliary capillaries, whereas the other products of activity of the liver cells (sugar and urea) pass to the blood capillaries which border on the edges of the liver cells. We do not know why this process is regularly being accomplished in such a manner under normal circumstances. A change in this twofold secretion is followed by icterus, if the bile flows in the wrong direction—i. e., into the blood capillaries. This "parapedesis of the bile" (Minkowski) may take place with or without the interference of mechanical factors; however, the latter are, in by far the majority of cases, determining for the origin of icterus. So soon as the pressure within the bile ducts rises or falls in the blood-vessels, an alteration in the flow of bile must take place, in such a manner that it diffuses in the direction of least resistance—i. e., into the blood-vessels.[1] From this point of view two forms of icterus may be distinguished: 1. *Retention icterus*, the origin of which is due to an obstruction to the flow of bile in the bile ducts, a *retention* of the secretion. 2. *Aspiration icterus*, the origin of which is due to a marked fall of the blood pressure in the liver vessels.

An example of the latter variety of icterus production—i. e., by sudden fall of the blood pressure in the liver vessels—is icterus neonatorum. It is brought about in such a manner that, immediately after birth, the blood capillaries of the liver are suddenly filled less, owing to the loss of the blood of the umbilical veins, and, besides, because the pressure in the abdominal aorta and with it in the hepatic artery abruptly falls with the first inspiration (B. Schultze). The category of *retention icterus*, on the other hand, embraces almost all cases of icterus, even those cases in which the large bile ducts are obstructed by stones or by mucous plugs in catarrhal jaundice, or which are compressed from outside by tumours in the liver; furthermore, icterus upon obstruction of the fine bile canaliculi by disintegrating epithelia or particles of mucus in the various affections of the liver. It is possible that a catarrh of the finest branches of the bile-duct system may be the cause of that form of icterus

[1] We may assume, according to the most recent experimental experience, that the absorption of bile into the blood does not take place directly into the blood capillaries of the liver, but always into the lymph vessels, especially into the perivascular lymph sheaths, and that the thus resorbed and carried bile does not enter the blood current directly but first through the thoracic duct. However, this alteration in our present views regarding the resorption of bile is as yet of very little practical value in pathology.

which occurs in the course of intoxications with dissolution of the red blood corpuscles (so-called "hæmatogenous" jaundice). In this case it may happen that the toxine causes a catarrh of the finest bile-canaliculi, and, owing to the erythrocytolysis, a more marked production of bile pigment and a more thickened condition of the blood results. Both factors would act in the same manner, i. e., produce an obstruction to the flow of bile and thus cause icterus.

3. These two forms of icterus, which are probably caused solely by mechanical conditions, are possibly opposed by a third form, which, however, is but very rarely to be considered—namely, *icterus caused by disturbance of bile secretion.* This form should be assumed in those cases in which no anatomical lesion of the liver can be found—i. e., in which, in the entire area of the bile ducts, any obstacle to the flow of bile is absent and the icterus is not preceded by a fall of blood pressure, so that absolutely nothing remains to explain its occurrence but to recur to a functional disturbance of the liver cells proper. The origin of icterus in such cases should, therefore, be looked for: (*a*) In an overflow of excessively secreted bile also to the blood-vessels (*hypersecretion icterus*). Thus especially in cases of hæmoglobinæmia without anatomical changes in the liver, or in other forms of icterus; (*b*) in a flow of the bile in a wrong direction (*parasecretion icterus*), which is brought about solely in consequence of abnormal function of the liver cells even without excessive secretion of bile.

The presence of bile constituents in the blood and the absence of bile in the intestine causes a number of clinical symptoms: jaundice of the skin, more marked fat contents of the fæces, etc. We shall refer to them more explicitly when discussing the diagnosis of jaundice.

In the diagnosis of affections of the liver it is best to separate from diseases of the entire organ those maladies which refer only to the two channel systems of the liver—the bile ducts and blood-vessels; these latter will be discussed in an appendicular form. The affections of the liver proper are accompanied with either diminution, or, the more frequent occurrence, enlargement of the organ, and it is practical in general to start from the change in the size of the liver present as a basis in the diagnosis.

Two affections of the liver, in which *atrophy of the organ* typically predominates in the pathological picture, are principally to be considered in a clinico-diagnostic respect: an acute disease, *acute yellow atrophy of the liver*, and a chronic one, *cirrhosis of the liver.*

ACUTE YELLOW ATROPHY OF THE LIVER

This most interesting affection is quite rare. The diagnosis is, as a rule, not easy except when the symptoms are fully developed and the course of the disease can be observed in its various phases.

Onset of Acute Yellow Atrophy.—*The commencement of acute yellow atrophy* of the liver is not marked. The symptoms of a gastric or intestinal catarrh with supervening icterus usually usher in this dangerous disease. Fever may be present or absent. This prodromal stage of the affection gives the impression of an innocent catarrhal jaundice, nor can it be distinguished from it. After it has persisted for several days, in some cases several weeks (in the last case I observed three weeks and a half), the pathological picture assumes, more or less suddenly, an unmistakably grave character.

Stage of Full Development.—*Delirium* sets in, *convulsions, abdominal pain, somnolence,* and finally *coma.*

At the same time a *rapidly increasing reduction of the volume of the liver occurs,* so that the liver may become reduced to one half or even one quarter its natural size within a few days. The atrophy is generally

most pronounced in the left lobe of the liver. The hepatic dulness accordingly diminishes, sometimes until it disappears entirely, not only owing to the decrease in volume of the liver, but also because the latter sinking posteriorly is covered by the intestines. I was able in the last case I observed to produce a *depression in the abdominal wall*, which disappeared very gradually, on *palpation of the liver* below the xiphoid process by pressing for some time. This compression was evidently caused by the impression which was produced by pressure upon the surface of the relaxed liver and which also involved the adjacent abdominal wall. Possibly this symptom may be of diagnostic use in future cases.

The *spleen*, in contradistinction to the reduction in size of the liver, increases in volume in the majority of cases. *Hæmorrhages* may occur in various portions of the body (hæmaturia, metrorrhagia, hæmatemesis, cutaneous ecchymoses, etc.), the same as in jaundice from other causes so also in this form of grave icterus. These hæmorrhages hasten the fatal termination, as was the case in an example I am going to report farther on. The *stools* are usually without bile, or contain very little bile. *Fever* is sometimes observed at the onset of the disease, *but is absent almost without exception in the later and graver stage of the disease.* Mostly, in about 50 per cent of the cases, a rise in temperature up to 105° F. and above occurs shortly before death (on the last day). The pulse accordingly is slow at the beginning, and towards the end of life a frequency of 120 and more per minute is noted.

Urinary Changes.—*The observation of eventual changes in the urine is important from a diagnostic standpoint.* The urine has been subjected in a great number of cases to an exact chemical examination, and in most cases (at first by Frerichs and Städeler) *leucin* and *tyrosin* were found in it, besides also aromatic oxyacids, especially oxymandelic acid. Aromatic substances (phenylacetic acid, phenylpropiolic acid, etc.) have been found, as is well known, as products of the marked *albumin putrefaction*, and their formation may be considered generally as a consequence of great disintegration of the albumin of the body. Tyrosin ($C_6H_4<^{OH}_{CH_2.CHNH_2.COOH}$) = the amido derivative of hydroparacoumaric acid, belongs to the class of aromatic substances. Feeding experiments with tyrosin have shown that at least sometimes larger quantities of aromatic oxyacids appear in the urine, especially oxyhydroparacoumaric acid ($C_6H_4<^{OH}_{CH_2.CHOH.COOH}$), which represents the homologue of oxymandelic acid ($C_6H_4<^{OH}_{CHOH.COOH}$), which is found in the urine in acute yellow atrophy of the liver. It is probable now that in the affection with which we have to deal, the products of disintegration which arise in total degeneration of the liver cells, furnish the material for the formation of amido acids and aromatic oxyacids. According to the degree of deficiency of oxidation, tyrosin would be formed, or in less reduced oxidation aromatic oxyacids, and eventually be excreted in the urine. Besides the aromatic substances, albumoses have been also determined, probably originating from the same source. At any rate, the urine in most cases contains leucin and tyrosin, easily demonstrable in the sediment, tyrosin needles, even in the spontaneously excreted sediment of the non-inspissated urine. However, these substances may be absent in some cases. They were certainly so in the last cases I observed. Positive results of the urine analysis are therefore of diagnostic importance. The same holds good in the *decrease of urea*, which, however, is not constant, but is highly interesting. Considerable amounts of *ammonia* and larger quantities of *lactic acid* have been demonstrated in the urine. We have already considered the sig-

nificance of these urinary findings (see p. 171). *Albuminuria* is sometimes present, at other times absent. It is of no pathognomonic importance because it may occur in consequence of the icterus, generally the same is the case with the formation of casts. *Sugar* has sometimes been found in the urine, and von Jaksch has been able to produce alimentary glycosuria in three patients suffering from phosphorus intoxication by the administration of 100 grammes of grape sugar.

The *blood* does not show any marked alterations. It seems that the red blood corpuscles upon examination of the blood incline to take the form of thorn apples. The leucocytes appeared enlarged in the last of my cases. An increase of the same has also been determined.

Any of these symptoms may occasionally be absent, but nevertheless the characteristic change in the liver may be found post mortem. This applies even to jaundice and to the diminution in size of the liver. Both manifestations may eventually not develop because the course of the affection is too short. A profuse gastric hæmorrhage caused such a rapidly fatal termination in one of my cases *that material diminution of the organ could not be observed in spite of the acute fatty degeneration.*

Case of Acute Yellow Atrophy terminating Fatally before Decrease in the Size of the Liver could be Determined.—The clinical picture in this case was that of a grave icterus with pain on pressure upon the epigastrium, coma, and hæmorrhagic diathesis. The autopsy (Zenker) showed *a liver of normal size with the appearance of a high degree of fatty degeneration*, spleen small, *catarrh of the bile ducts, fatty degeneration of the heart and kidney, hæmorrhagic diathesis* (ecchymoses of the pleura, the liver substance, and the endocardium). The *stomach contained an enormous quantity of bloody blackish-red fluid and masses adhering to the mucous membrane*, after the removal of which the membrane appeared pale gray. The large and small intestines contained moderate amounts of grayish-black masses. The result of the microscopical examination showed deviations from the usual picture of fatty liver, so that the most probable cause of the alteration of the liver had to be ascribed anatomically to incipient acute yellow atrophy of the liver, the development of which was suddenly interrupted by the acute gastric hæmorrhage leading to a rapidly fatal termination.

A reduction of the hepatic dulness was not demonstrable in a case of acute yellow atrophy of the liver recently reported by Gerhardt, in spite of the actual reduction of the liver, because, as seen at the autopsy, the small liver was prevented from sinking towards the posterior abdominal wall by connective-tissue-like adhesions with the anterior abdominal wall. It is even possible that *the liver may appear enlarged at the onset of the disease.* This was the fact in a case recently observed by me in which the liver, which had been swollen until then, did not commence to atrophy until three days before death. It is obvious that in *phosphorus poisoning* the liver is constantly enlarged during the first period and during the entire course of the disease.

Jaundice is certainly absent only in the very rarest cases, which rapidly lead to death. Otherwise it is the most constant symptom of yellow atrophy of the liver, and equally as constant is the absence of fever in the second stage of the disease.

Diagnosis of the Various Stages.—*The affection cannot be diagnosticated in its first stage.* It is *suspicious* if icterus occurs in a pregnant

woman during the second half of the pregnancy, because it is during this time especially that the development of acute atrophy of the liver has been relatively often observed.

Differential Diagnosis of Jaundice in the Infectious Diseases.—I would also like to designate as suspicious *abdominal pains* complicating icterus at the onset of the disease. Such pains have existed in the above-described case from the very beginning until the final termination, a period of three weeks. *The diagnosis is comparatively very simple in the second stage,* but mistaking the disease for other affections is possible. *Severe nervous symptoms* may, as is well known, be due to any grave icterus. They are especially frequent and severe if the icterus occurs in the course of infective diseases, as in pneumonia, septicopyæmia, etc. But as these affections are accompanied by high fever, and acute atrophy of the liver, on the other hand, does not show any rise of temperature just at the time of the grave cerebral symptoms, the differential diagnosis is usually not difficult for these reasons. The diagnosis of acute yellow atrophy of the liver becomes absolutely certain if a gradually occurring reduction of the volume of the liver becomes demonstrable.

Jaundice in Hysteria.—It may be possible that in cases in which icterus attacks females who incline to *hysteria*, the intoxication caused by the resorption of bile or alteration of metabolism cause so strong a reaction of the irritable nervous system that a picture is presented of severe cerebral irritation with delirium and convulsions. I have seen this years ago in a case in which the pathological picture strikingly resembled cholæmia; but upon more exact examination several minor features were found which pointed to the hysteric character of the nervous symptoms. Also the effect of an antihysterical therapy, as well as the course of the disease, especially the sudden disappearance of the nervous disturbances, will point to the correct diagnosis.

Tympanites of the Transverse Colon simulating Atrophy of the Liver.—The diagnosis is always doubtful until the reduction of the liver can be positively demonstrated, but the doubts gradually disappear when the percussion shows *the borders of the liver to be gradually decreasing in extent.* It should not be forgotten, however, *that the transverse colon may often upon great inflation push the liver posteriorly to such an extent that the borders of the liver are reduced to a minimum. In rare cases they may even disappear entirely at the anterior thoracic wall according to my experience.* However, in such cases tympany is more general and the percussion borders of the liver change according to the degree of inflation—i. e., they reappear eventually with the evacuation of fæces and flatus. The examination of the urine requires particular attention in such cases, for the positive finding of leucin and tyrosin clears the diagnosis.

Cases terminating in Recovery.—Finally, the *rapid fatal termination* contributes materially towards the diagnosis. The severe stage of the disease lasts from one day to one week, not counting rare exceptions. *If recovery occurs* after a diagnosis of acute yellow atrophy of the liver, it is *more than likely that the diagnosis was wrong.* However, according to my experience, there are some (although very rare) cases in which the disease does not terminate fatally. I have twice observed icterus with all its symptoms developed and in which undoubted diminution of the liver had occurred, when, contrary to all expectations, the patients recovered. In one of the cases leucin and tyrosin could be demonstrated for weeks in the urine; besides, there existed a transient cardiac dilatation (fatty degeneration) and leucocytosis.

nificance of these urinary findings (see p. 171). *Albuminuria* is sometimes present, at other times absent. It is of no pathognomonic importance because it may occur in consequence of the icterus, generally the same is the case with the formation of casts. *Sugar* has sometimes been found in the urine, and von Jaksch has been able to produce alimentary glycosuria in three patients suffering from phosphorus intoxication by the administration of 100 grammes of grape sugar.

The *blood* does not show any marked alterations. It seems that the red blood corpuscles upon examination of the blood incline to take the form of thorn apples. The leucocytes appeared enlarged in the last of my cases. An increase of the same has also been determined.

Any of these symptoms may occasionally be absent, but nevertheless the characteristic change in the liver may be found post mortem. This applies even to jaundice and to the diminution in size of the liver. Both manifestations may eventually not develop because the course of the affection is too short. A profuse gastric hæmorrhage caused such a rapidly fatal termination in one of my cases *that material diminution of the organ could not be observed in spite of the acute fatty degeneration.*

Case of Acute Yellow Atrophy terminating Fatally before Decrease in the Size of the Liver could be Determined.—The clinical picture in this case was that of a grave icterus with pain on pressure upon the epigastrium, coma, and hæmorrhagic diathesis. The autopsy (Zenker) showed *a liver of normal size with the appearance of a high degree of fatty degeneration*, spleen small, *catarrh of the bile ducts, fatty degeneration of the heart and kidney, hæmorrhagic diathesis* (ecchymoses of the pleura, the liver substance, and the endocardium). The *stomach contained an enormous quantity of bloody blackish-red fluid and masses adhering to the mucous membrane*, after the removal of which the membrane appeared pale gray. The large and small intestines contained moderate amounts of grayish-black masses. The result of the microscopical examination showed deviations from the usual picture of fatty liver, so that the most probable cause of the alteration of the liver had to be ascribed anatomically to incipient acute yellow atrophy of the liver, the development of which was suddenly interrupted by the acute gastric hæmorrhage leading to a rapidly fatal termination.

A reduction of the hepatic dulness was not demonstrable in a case of acute yellow atrophy of the liver recently reported by Gerhardt, in spite of the actual reduction of the liver, because, as seen at the autopsy, the small liver was prevented from sinking towards the posterior abdominal wall by connective-tissue-like adhesions with the anterior abdominal wall. It is even possible that *the liver may appear enlarged at the onset of the disease.* This was the fact in a case recently observed by me in which the liver, which had been swollen until then, did not commence to atrophy until three days before death. It is obvious that in *phosphorus poisoning* the liver is constantly enlarged during the first period and during the entire course of the disease.

Jaundice is certainly absent only in the very rarest cases, which rapidly lead to death. Otherwise it is the most constant symptom of yellow atrophy of the liver, and equally as constant is the absence of fever in the second stage of the disease.

Diagnosis of the Various Stages.—*The affection cannot be diagnosticated in its first stage.* It is *suspicious* if icterus occurs in a pregnant

woman during the second half of the pregnancy, because it is during this time especially that the development of acute atrophy of the liver has been relatively often observed.

Differential Diagnosis of Jaundice in the Infectious Diseases.—I would also like to designate as suspicious *abdominal pains* complicating icterus at the onset of the disease. Such pains have existed in the above-described case from the very beginning until the final termination, a period of three weeks. *The diagnosis is comparatively very simple in the second stage,* but mistaking the disease for other affections is possible. *Severe nervous symptoms* may, as is well known, be due to any grave icterus. They are especially frequent and severe if the icterus occurs in the course of infective diseases, as in pneumonia, septicopyæmia, etc. But as these affections are accompanied by high fever, and acute atrophy of the liver, on the other hand, does not show any rise of temperature just at the time of the grave cerebral symptoms, the differential diagnosis is usually not difficult for these reasons. The diagnosis of acute yellow atrophy of the liver becomes absolutely certain if a gradually occurring reduction of the volume of the liver becomes demonstrable.

Jaundice in Hysteria.—It may be possible that in cases in which icterus attacks females who incline to *hysteria*, the intoxication caused by the resorption of bile or alteration of metabolism cause so strong a reaction of the irritable nervous system that a picture is presented of severe cerebral irritation with delirium and convulsions. I have seen this years ago in a case in which the pathological picture strikingly resembled cholæmia; but upon more exact examination several minor features were found which pointed to the hysteric character of the nervous symptoms. Also the effect of an antihysterical therapy, as well as the course of the disease, especially the sudden disappearance of the nervous disturbances, will point to the correct diagnosis.

Tympanites of the Transverse Colon simulating Atrophy of the Liver.—The diagnosis is always doubtful until the reduction of the liver can be positively demonstrated, but the doubts gradually disappear when the percussion shows *the borders of the liver to be gradually decreasing in extent.* It should not be forgotten, however, *that the transverse colon may often upon great inflation push the liver posteriorly to such an extent that the borders of the liver are reduced to a minimum. In rare cases they may even disappear entirely at the anterior thoracic wall according to my experience.* However, in such cases tympany is more general and the percussion borders of the liver change according to the degree of inflation—i. e., they reappear eventually with the evacuation of fæces and flatus. The examination of the urine requires particular attention in such cases, for the positive finding of leucin and tyrosin clears the diagnosis.

Cases terminating in Recovery.—Finally, the *rapid fatal termination* contributes materially towards the diagnosis. The severe stage of the disease lasts from one day to one week, not counting rare exceptions. *If recovery occurs* after a diagnosis of acute yellow atrophy of the liver, it is *more than likely that the diagnosis was wrong.* However, according to my experience, there are some (although very rare) cases in which the disease does not terminate fatally. I have twice observed icterus with all its symptoms developed and in which undoubted diminution of the liver had occurred, when, contrary to all expectations, the patients recovered. In one of the cases leucin and tyrosin could be demonstrated for weeks in the urine; besides, there existed a transient cardiac dilatation (fatty degeneration) and leucocytosis.

Differential Diagnosis between Phosphorus Poisoning and Acute Yellow Atrophy of the Liver.—According to my view it is simply an ætiological *question whether acute phosphorus poisoning*, or *acute yellow atrophy* of the liver is present. The same as the noxa which is the cause of the last-named disease, and which up to now has not been discovered,[1] causes fatty degeneration and reduction in size of the liver, so may also intoxication by phosphorus terminate in lasting fatty degeneration of the hepatic parenchyma and in atrophy of the organ, instead of the usual fatty degeneration which accompanies enlargement of the liver. However, the latter is rarely the case in the course of acute phosphorus poisoning. But then both pathological pictures, idiopathic yellow atrophy of the liver and that which is caused by phosphorus intoxication, are identical in all details, and the artificial characteristics are clinically insufficient.

The second of the typical forms of reduction in the size of the liver, the chronic form, is *cirrhosis of the liver*. Before considering the diagnosis of the latter, I wish to mention an atrophy of the liver which presents more anatomical than clinical interest—namely, *simple chronic atrophy of the liver*.

Simple Chronic Atrophy of the Liver.—This affection is one of the manifestations of marasmus which leads to atrophy of the organs in general, and especially to atrophy of the liver. The *diagnosis* of the latter is possible if the dulness of the liver gradually decreases uniformly in senile, marantic or cachectic individuals and the stools become poor in bile corresponding to the reduction of the hepatic parenchyma without being due to occlusion of the bile ducts. *Simple marantic atrophy differs from cirrhosis in so far as ascites is absent*, or at least does not prevail in comparison to other symptoms of dropsy, nor are swelling of the spleen and the other symptoms of cirrhosis present. The consistence of the marantic atrophic liver is slightly harder than the normal, owing to the preponderance of the connective tissue after the parenchyma of the liver proper has more and more disappeared.

HEPATIC CIRRHOSIS.—ATROPHIC INTERSTITIAL HEPATITIS.—LAENNEC'S ATROPHY OF THE LIVER.

The *diagnosis of cirrhosis of the liver* (interstitial hepatitis) generally offers no difficulties when the liver is palpable. The latter is possible when the ascites which dominates the pathological picture is but slightly developed, or if the fluid can be pushed from the liver for some moments by palpation, or if it can be aspirated. In many cases the diagnosis is only possible by an examination following immediately upon paracentesis of the abdomen.

The *liver* is shrunken, its dulness diminished, especially over the left lobe. If the liver is palpable under the above-named circumstances its consistence appears hard. Nodules can be felt on the surface and border, but only when they have grown to considerable size, owing to the development of large cicatricial connective-tissue proliferations.

[1] All reports in reference to pathogenic micro-organisms as a specific cause of acute yellow atrophy of the liver have as yet been unconfirmed. Also the assumption of an auto-intoxication as a result of decomposition from the intestinal tract is but a hypothesis; it appears probable that syphilis in its early stages is able to produce acute atrophy of the liver.

It cannot at present be decided whether the liver is larger in the first stage of the disease and gradually becomes smaller. It is certain, however, that the most experienced physicians have not observed cases proving without doubt that an enlargement of the liver existed at the onset with a chronic course of cirrhosis followed by gradual atrophy of the liver. I, although my material in this department of pathology was limited, have not seen an unobjectionable example of such a course of the disease. If other reliable observers, on the other hand, declare the occurrence of an enlargement of the liver in the first stage of Laennec's cirrhosis as undoubtable, there remains nothing for the present but to assume that it is rare at any rate, and this factor should not (or at most cautiously) be taken into consideration in the diagnosis.

If the liver is uniformly granulated, the granules being the size of a lentil to that of a pea (the usual case), they are *not* palpable, in my experience. It is true that sometimes palpation gives a sensation as if the surface of the liver contained small nodules. However, I believe this is more the result of self-suggestion than of fine technique. Mistakes occur quite usually if granular fatty accumulations become palpable in the abdominal wall during the disappearance of the panniculus adiposus of the abdominal wall. The differentiation of such superficial granulations from the deeper liver nodules is said to be possible by observing the displacement of the latter upon respiration—a differentio-diagnostic expedient of more theoretical value than it has been proved clinically.

Results of Portal Obstruction.—Owing to compression and obliteration of a large number of branches of the portal vein between the lobules of the liver by the new-formed atrophied connective tissue, an *engorgement occurs in the portal vein,* thus causing a number of manifestations which are characteristic and of diagnostic importance, especially portal-vein engorgement, and indirectly therefore characteristic of hepatic cirrhosis. They are as follows:

Enlargement of the spleen is absent only in a quarter of the cases. It is characterized very markedly as an effect of portal engorgement by the rapid change in the size of the spleen. Thus upon sudden emptying of the portal-vein system by hæmorrhages from branches of the portal vein (for instance by profuse gastric hæmorrhages) we may thus be enabled to determine an acute transitory diminution of the enlarged spleen. But the engorgement in the portal-vein system is by no means the sole cause of enlargement of the spleen in atrophic cirrhosis of the liver, because the spleen may eventually appear enlarged in the initial stage of the disease at a time at which no material engorgement has developed at all in the portal-vein system. As a matter of fact Th. Oestreich succeeded recently in demonstrating that the splenic tumour in the first stages of cirrhosis depends upon a cellular hyperplasia of the parenchyma, which is to be regarded as a consequence of irritative processes affecting the liver and spleen, and which in the later stages may lead to a proliferation of the connective tissue in the spleen or to actual atrophy of the pulp.

The engorgement in the gastric and intestinal veins is the cause of *hyperæmia* of the *gastric* and *intestinal mucous membranes,* of the development of chronic gastric and intestinal catarrhs, of chronic constipation and of tympany (the last two symptoms are probably due to serous infiltrations of the intestinal muscles), of gastric and intestinal hæmorrha
Upon continuation of the engorgement in the inferior mesenteric

upon the superior hæmorrhoidal veins, and upon the hæmorrhoidal plexus *in toto, hæmorrhoids develop* which give rise to well-known discomforts and to temporary hæmorrhages from the anus.

The most important clinical symptom of portal-vein engorgement is *ascites*, which, developing slowly, often leads to an enormous distention of the abdomen. It is characteristic that ascites in cirrhosis of the liver is almost always the first sign of dropsy. Not until later, owing to the increasing intra-abdominal pressure, which grows with the accumulation of fluid in the abdomen, does there occur impairment to the venous reflux into the iliac veins and into the inferior vena cava, and with it *œdema of the lower half of the body.*

It is possible that in *some cases anasarca of the lower extremities may also occur with moderate ascites* when the reflux of the venous blood from the lower extremities is impeded by collateral overfilling of the epigastric and hypogastric veins from the portal-vein system.

Collateral Venous Circulation.—But, on the other hand, the *opening of such collaterals* between the *portal-vein system* and the *vena cava* often prevents engorgement symptoms for some time. Of especial importance for the diagnosis, because it causes visible distention of the veins externally, is the already-mentioned collateral communication between the superior hæmorrhoidal veins (a tributary of the portal vein) with the medium and inferior hæmorrhoidal veins, and thereby with the hypogastric vein and inferior vena cava. The surcharge of this collateral circulation manifests itself externally in the form of hæmorrhoidal varices at the anus. The surcharge of another collateral communication causes still more marked symptoms, "*of the accessory portal-vein branch*," the communication between the *epigastric* and the *internal mammary vein in the region of the umbilicus* (compare pp. 171, 174). Overfilling of the communication from the portal vein, as is natural in great obstruction to the portal-vein current in the liver, causes an overfilling of the epigastric and internal mammary veins, producing varicose distentions of the dermal veins around the umbilicus (*Caput Medusæ*). However, a great extension of this venous condition is rare, and a much more frequent form of venous circle in the abdominal skin must not be confounded with it. In every greater development of the abdominal pressure, especially in the higher grades of ascites, there occurs an impediment of the blood current in the inferior vena cava. This causes an engorgement of the blood coming from the lower extremities into the inferior epigastric and internal mammary veins, and in consequence a distention of the superficial dilatation of the veins of the abdominal wall. These venous enlargements are, in contradistinction to the condition in Caput Medusæ, disseminated very irregularly over the entire surface of the abdomen, and especially are they developed in the lateral portions of the abdomen. Of the further collateral communications between the portal vein and vena-cava system, the development of which is of importance eventually for the diagnosis of certain complications which occur in the course of hepatic cirrhosis, I may mention the communication between the superior epigastric vein with the lower œsophageal veins. The latter empty their blood into the azygos and hemiazygos veins. An obstruction of the portal-vein system therefore causes an overfilling of the œsophageal veins and eventually the formation of varices, the occasional rupturing of which may not infrequently be the cause of a fatal *hæmatemesis*. Besides, as has been recently emphasized by Italian investigators, the reflux of the blood from the intercostal and pleural veins of the right side may be impaired upon overfilling of the œsophageal veins and indirectly of the azygos veins, thus causing *hydrothorax of the right side*. In other cases again, the fluid which accumulates in the right pleural cavity is of an inflammatory nature, inasmuch as an irritation and inflammation of the liver serosa may occur in the course of the cirrhosis and may extend through the diaphragm to the right pleura. A *continuous hum, venous murmur*, occurring in the region of the liver, may be traced to the engorgement of the venous trunk of the portal vein and

of the branches entering into the same. This venous murmur may, if the ascites and the dislocation of the liver are marked, be brought about by the narrowing of the vena cava at the point where it communicates with the liver, thus causing the murmur.

Urine.—The other symptoms of cirrhosis of the liver are of very inferior importance. The *urine* is secreted very scantily owing to the poor and insufficient filling of the arteries, contains a large amount of sediment, may contain albumin owing to the engorgement in the renal veins caused by the increased intra-abdominal pressure, or as the result of complicating inflammatory processes in the kidneys (granular kidney is most frequently associated with cirrhosis of the liver). Blood may also appear in the urine in consequence of the engorgement of the veins of the bladder. *The excretion of urea* is slightly diminished, that of ammonia rather increased; however, the quantity of urea and ammonia which is found in the urine is not sufficient to prove that a disturbance of urea synthesis exists in cirrhosis.

It seems that the occurrence of *sugar* in the urine is to a certain extent characteristic. In this respect the food of the patients seems to be of importance, for glycosuria occurs upon ingestion of sugar-containing foods; but glycosuria is absent in many cases of cirrhosis (and other affections of the liver), so that we may assume that sugar occurs in the urine only in those cases in which the disturbance of the function of the liver cells has far advanced and in which the organs of the glycogen formation outside of the liver (especially the muscles) are not sufficiently able to compensate for the destroyed function of the liver cells.

Jaundice.—Whether the urine contains bile pigment depends upon whether the cirrhosis is accompanied by icterus or not. Icterus is by no means a constant symptom of cirrhosis, it being found in but about one sixth of the cases, and is usually slightly developed, so that if severe icterus exists with *constant* decoloration of the fæces (transitory clay colour alternating with a normal colour is not infrequently observed in consequence of deficient bile formation), the presence of the cirrhosis is improbable.

Ætiological Diagnosis.—Before entering upon the differential diagnosis, I wish to mention the *ætiology* of cirrhosis of the liver, because the consideration of the same may sometimes be of use in the diagnosis. It is beyond doubt that the *abuse of alcohol* is the most frequent cause of the disease. It must be left undecided for the present, whether alcohol, as such, irritates the connective tissue of the liver primarily, causing inflammation, or whether the liver cells are directly injured and the connective tissue in consequence proliferates and later atrophies, or whether both at the same time are the effect of the alcohol, also whether alcohol is only indirectly the cause of the hepatic alteration, inasmuch as it may result in the abnormal products of fermentation in the intestinal canal which are absorbed and carried to the liver. All other noxæ, like malaria, gout, lead-poisoning, etc., which are said to be causes of cirrhosis of the liver, are scarcely worth considering if we do not refer to syphilis (see p. 187), so that in doubtful cases the proof of the absence of alcohol is of great importance for the presence of cirrhosis of the liver.

Differential Diagnosis.—The diagnosis of cirrhosis of the liver must not be made from symptoms alone, but owing to the similarity of the symptoms which are common to all, or at least to a great many affections of the liver, the question is to be decided whether no other affection of the liver is present.

If we do not refer to the so-called first stage of cirrhosis of the liver in the differential diagnosis for the above-named reasons, mistaking various other chronic affections of the liver which lead to atrophy may be possible in cases in which the ascites allows palpation of the liver to be made, and in which the organ will be found diminished in size, especially in the left lobe. One form of *syphilis* of the liver is to be considered which is accompanied with the development of connective tissue, and therefore shows similar morphological changes, with the same clinical symptoms subsequently, as does common cirrhosis (see p. 187). The tendency to *great* atrophy of the connective-tissue proliferation caused by the syphilis is characteristic to a certain extent, so that extensive retractions occur upon the surface and the borders of the liver. This causes the liver to assume a lobulated form, so that the effects of lacing and the irregular surface can be plainly felt, which is seldom the case in common cirrhosis. *Pains* are also more frequent in syphilitic hepatitis, in my experience, than in the other form. It goes without saying, that other manifestations of tertiary syphilis are necessary for the diagnosis of syphilis of the liver.

Atrophic Nutmeg Liver.—The *atrophic nutmeg liver* is another affection which gives rise to atrophy of the liver and which may lead to mistakes in diagnosis. The diminution of the liver, icterus, and ascites are present, as in cirrhosis; but the ascites does not precede the anasarca, it usually follows. Nor is it possible always to demonstrate beyond doubt the conditions (heart and lung defects, etc.) and symptoms of general engorgement of which ascites only forms one part, while on the other hand chronic malaria and other infectious diseases, lead-poisoning, and, above all, abuse of alcohol, which have preceded the affection, speak decidedly in favour of the existence of atrophic cirrhosis.

Simple marantic atrophy of the liver shows a quite uniform decrease of the liver, which takes place in all its dimensions, thinning of the borders of the liver, absence of unevenness of the surface, absence of icterus and of the symptoms of portal-vein engorgement. However, simple atrophy is of more anatomical than of clinical interest.

If the accumulation of fluid in the abdominal cavity is so considerable *in the course of cirrhosis*, that palpation of the liver cannot be thought of and percussion is also rendered difficult, the next question to be decided is, not what variety of hepatic affection is present, but whether we have to deal with one at all. Paracentesis is indicated in such cases, to enable a diagnosis. If the fluid is yellowish and clear it may be ascites or a large ovarian cyst. The specific gravity of the fluid, especially if below 10.15, decides in favour of ascites, besides other characteristic symptoms which are not to be discussed here. After the diagnosis of ascites has been established, the question arises whether it is an isolated dropsical manifestation or, at least, the fundamental symptom of dropsy, or only a partial symptom of general dropsy. In the two former cases the diagnosis inclines to disease of the liver. *Pylephlebitis* and *pylethrombosis* may then also be the cause of the ascites, besides any other affection of the liver. Very rapid development of ascites and the other consequences of portal-vein engorgement (swelling of the spleen, hæmatemesis and distention of the abdominal

veins in the course of the disease) and rapid recurrence of those symptoms after paracentesis, favour occlusion of the portal-vein trunk. But to diagnosticate the same requires, above all, the finding of the cause, a compression of the vessel in pylethrombosis, or of an ulcerating process in the region of the portal vein, or of gall-stones in suppurative pylephlebitis. But it must not be forgotten that pylethrombosis does occur in cirrhosis of the liver, and suppurative pylephlebitis may occur as a consequence of *chronic peritonitis*. The latter is to be thought of, if the fluid which is withdrawn from the peritoneal cavity is not clear but more or less cloudy and shows varying quantities of leucocytes on microscopical examination. Chronic peritonitis is usually accompanied with pains and fever. The peritoneal fluid in the tuberculous and carcinomatous forms is *hæmorrhagic*, as a rule, thickening of the peritonæum or actual tumours can plainly be felt after withdrawal of the fluid, and carcinoma or tuberculosis may also be demonstrated in other organs. The absence of engorgement symptoms in the portal-vein system is of especial importance in that form of chronic diffuse peritonitis which is not complicated by pylephlebitis. It should not be left unmentioned that not infrequently (a quarter of the cases) tuberculosis, and especially tuberculous peritonitis were found coexisting with cirrhosis of the liver, a fact which may possibly be explained in such a manner that alcoholism, which causes cirrhosis of the liver, also predisposes to tuberculosis.

From the above it will be seen, that the difficulties in the diagnosis of cirrhosis of the liver are not a few, and I must emphasize again in conclusion, that this diagnosis should be well considered in every case and in every direction, before it can be established with certainty, because it is much more difficult according to my experience, in the majority of cases, than is usually supposed, and it is not a rare occurrence that the autopsy reveals a wrong diagnosis.

CONNECTIVE-TISSUE HYPERPLASIA OF THE LIVER—HYPERTROPHIC CIRRHOSIS—ELEPHANTIASIS OF THE LIVER

This affection, which until recently had not been studied anatomically, is accompanied by an enlargement of the liver caused by hyperplasia of the connective tissue, and has until lately been identified with the "first stage" of common cirrhosis of the liver. The principal difference between both pathological processes may be, that the development of connective tissue in common cirrhosis is an inflammatory one with subsequent cicatricial atrophy, and a non-atrophic *hyperplastic* one in the hypertrophic form and in a similar manner as is the case in elephantiasis. Furthermore, in atrophic cirrhosis, the liver cells are affected in structure and function and from the beginning the finer portal-vein branches become compressed and obstructed; while in hypertrophic cirrhosis the liver cells show a normal condition, the lumen of the liver vessels is distended, and the communication between the portal vein and the liver veins is absolutely unobstructed. This explains the diagnostically important fact that in

hypertrophic cirrhosis, in contradistinction to the atrophic form, the engorgement symptoms in the region of the portal vein, characteristic of the latter, fail to appear (at least in the majority of the cases, especially in the earlier stages of the disease)—i. e., the symptoms of gastric and intestinal hyperæmia, the hæmorrhoids, and especially the ascites, are absent. The latter symptom occurs only as an expression of general cachexia. On the other hand, enlargement of the spleen (sclerotic) and usually intense icterus, are associated with the affection. Icterus is so regularly associated with it that a number of investigators, as is well known, consider this affection of the liver as originating in an inflammation of the bile ducts. Fæces free from bile alternate with bile-stained fæces in the course of the icterus which forms the principal symptom of the disease, but the cause of which is as yet unexplained. The appetite is not lessened and the digestion not impaired. The urine, in contradistinction to its condition in atrophic cirrhosis, is abundant and always contains bilirubin. The excretion of urea is normal. The *blood* of the patients shows a decrease in the number of red corpuscles to one half of the normal number and a relative increase of the leucocytes, according to the investigations of Rosenstein. This author found, in connection herewith, a hæmorrhagic diathesis and anæmic murmurs over the heart. The enlargement of the liver is usually very considerable. The consistence of the organ is moderately hard, its border sharp or slightly rounded, the surface smooth, at most showing but a few isolated flat prominences. The course of the disease is chronic, its duration from five to ten years.

Differential Diagnosis—Carcinoma of the Liver.—In the differential diagnosis there are to be considered: *carcinoma of the liver*, which is also characterized by an enlargement of the liver, hardening of the organ and jaundice. On the other hand, it is distinguished from hypertrophic cirrhosis by the usually distinct roughness of the liver surface and of the border (even in infiltrated diffuse development of carcinoma), and the *absence of enlargement of the spleen*. According to my experience, ascites is absent but rarely in carcinoma of the liver, the duration of the affection is shorter, and the cachexia progresses much more rapidly. Besides, carcinoma of the liver is usually an affection of advanced life and in the majority of cases is secondary, so that we are usually able to demonstrate a primary carcinomatous affection of another organ; of the stomach, of the rectum, etc. It is also possible that an *amyloid affection of the liver* is to be considered in the differential diagnosis, because in this disease also the liver is enlarged, hard, having a smooth surface, the spleen is usually enlarged and, as a rule, ascites is absent. On the other hand, *in amyloid liver there is almost never icterus*. Besides, the albuminuria, which usually occurs simultaneously with amyloid liver, and the well-known ætiological basis of the amyloid affection, furnish firm points of support in the diagnosis. The differentiation of elephantiasis of the liver from simple icteric liver and *multilocular echinococcus* is much more difficult. The liver is large and hard to the touch in the latter disease. Icterus and splenic enlargement are almost always present as in hypertrophic cirrhosis. The surface of the liver, however, shows hard globular tumours which may become soft in the further

course of the disease, and the liver is sensitive to the touch. It is easy to mistake this disease for *enlargement of the liver arising in the course of icterus* produced by the occlusion of the bile ducts when the enlargement of the liver becomes more marked. In case of "icteric enlargement" of the liver, a rare sequence of icterus, the consistence of the liver is, although harder than normal, considerably softer than in hypertrophic cirrhosis. Furthermore, the liver, if it enlarges as a result of jaundice, enlarges much more rapidly than it does in hypertrophic cirrhosis. The course of the latter disease is generally a much more protracted one. Finally, even in permanent occlusion of the bile ducts (excretory) the icteric liver becomes smaller after a few months, owing to the gradually occurring atrophy of the parenchyma of the liver, unless a genuine biliary hypertrophic cirrhosis has developed after some time. The enlargement of the liver in *leucæmia* will scarcely give rise to mistakes. The enlargement of the spleen predominates here and requires an examination of the blood which will at once clear up the diagnosis.

Secondary Atrophic Liver.—Although there can be no doubt, according to the above statements, that atrophic and hypertrophic cirrhosis are two different forms of disease, it cannot be disputed that cases have been observed in which both types of cirrhosis were present at the same time (French "Type Mixte"), mixed forms in which the liver and spleen are enlarged and hard, but in which icterus is absent or only slight, and ascites also occurs gradually, as in Laennec's atrophic liver. Even the granular condition is present in such cases, *but the liver remains large until the death of the patient*, although an atrophic process may be surmised, as can be proved by the symptoms of portal-vein engorgement (*"secondary" atrophic liver*, Rosenstein).

SYPHILITIC AFFECTIONS OF THE LIVER—SYPHILIS OF THE LIVER

Syphilitic Cirrhosis.—It is well known that the liver is one of those internal organs which are most prominently affected by syphilis. There are cases of diffuse interstitial syphilitic hepatitis, which presents itself sometimes as a hypertrophic and at other times as an atrophic cirrhosis. The first form is found principally in children who are hereditarily syphilitic. The atrophic form is seen in adults. The luetic nature of the affection cannot be recognised in such cases from the clinical symptoms alone, because they do not differ from those commonly seen in cirrhosis, and the diagnosis of syphilitic cirrhosis can here be made only provisionally from the general condition of the body and the manifest saturation of the same with the syphilitic virus. But the syphilitic liver usually presents anatomical changes, which show the deviations from the usual conduct of the liver in cirrhosis and which allow of an almost certain diagnosis.

Specific Changes of the Liver in Syphilis.—Deep depressions occur on the surface and at the border of the liver from proliferation of the connective tissue in portions of the liver, most frequently in the neighbourhood of the suspensory ligament, which contracting cause atrophy of the inclosed liver tissue. The organ is either enlarged or reduced in size, with large projecting nodules, which are either due to unaffected portions of

the liver retaining their original size while the affected portion is contracted, or—the usual case—to deposits of gummata. These nodules vary in size from that of a millet-seed to that of an apple; they are relatively soft to the touch, especially when the adjacent liver tissue is affected with amyloid degeneration and increase in volume and consistence, which is not infrequently the case in the course of syphilis.

Syphilitic Perihepatitis.—The serous covering of the liver may also be involved in the syphilitic process localized in the liver in the form of a *perihepatitis*, which sometimes manifests itself by the presence of an audible and palpable friction sound in the hepatic region. It is very rare that this syphilitic hepatitis may be an independent disease. The provisional diagnosis, syphilitic perihepatitis, is then permissible only upon an ætiological basis—i. e., if undoubtedly pronounced tertiary syphilis is present. In most cases perihepatitis is rather one of the syphilitic phenomena of the liver tissue, and its diagnosis can be positively established in most cases if the just-described anatomical changes of the liver are considered. The diagnosis is completed by the subsequent symptoms. A varying degree of *ascites* and the remaining symptom-complex of portal-vein engorgement may be expected according as the process of connective-tissue proliferation becomes localized along the large branches of the portal vein (syphilitic peripylephlebitis), or if only small branches of the portal vein are compressed or obstructed. *Icterus* is present in about one third of the cases, especially when the atrophying connective tissue constricts large bile ducts, obstructs the flow of the bile, and thus produces icterus of a high grade with decoloration of the fæces, etc. The *spleen* is usually enlarged, due to engorgement of the portal vein or to interstitial induration of the organ, similar to the changes in the liver. Gummata formation or amyloid affection of the spleen may also be responsible for the enlargement of the organ. It may be mentioned finally that *pains* in the hepatic region, which are increased by external pressure, are the rule in this affection of the liver, which may sometimes be of value in the differential diagnosis.

Differential Diagnosis.—Nodular syphilitic liver is most easily confounded with *cancer of the liver*. The following symptoms point to syphilis: The nodes are slightly harder, and above all the deep depressions at the border of the liver, the *enlargement of the spleen*, and the albuminuria which is the result of secondary affection of the kidneys (parenchymatous nephritis, or amyloid disease). Youth especially, a favourable course of the affection of the liver which is very rare, and the far-reaching luetic infection of the organism, point to the diagnosis of hepatic syphilis. The last-named factors allow also of the differentiation of rarely occurring diffuse syphilitic infiltration of the liver from non-syphilitic cirrhosis, to a certain extent, at least provisionally. Among other affections of the liver which may give rise to a faulty diagnosis of syphilis of the liver, multilocular echinococcus is to be especially mentioned. I advise, that a search be made for cicatricial depressions on the border of the liver above all, the demonstration of which, with that of undoubted syphilitic changes in other parts of the body, furnish the most important support for the diagnosis of syphilitic hepatitis.

SUPPURATIVE HEPATITIS—ABSCESS OF THE LIVER

Abscess of the liver may be diagnosed with certainty in some cases, with probability in others, and in some the diagnosis is impossible, and a positive diagnosis cannot be made at all.

Size of the Liver.—The liver is *mostly enlarged*; it is characteristic to some extent, that the enlargement of the organ does not extend downward principally, as in other enlargements of the liver, but usually upward into the thoracic cavity, and then manifests itself upon percussion of the lower border of the lung as a dull area, which is found at the anterior surface of the thorax, its highest point in the axillary line, and gradually declining towards the vertebral column. The motility of the lung and liver border, on respiration, is diminished or absent, owing probably to the great upward displacement of the diaphragm and to the perihepatitic adhesions (Pel). However, the volume of the liver is not always increased, not even if the organ is filled with numerous abscesses of considerable size.

Surface of the Liver.—Flat nodes or prominences, which slightly taper towards the centre of the tumour, can be felt on the *surface of the liver*, if, exceptionally, the situation of the abscesses is on the left lobe of the liver or towards the border of the enlarged right lobe. If the abscesses are in the lowest portion of the liver this may cause the border of the organ to become irregular.

Consistence of the Liver.—The *consistence* of the tumour is hard at the beginning. It may be so hard that at first the diagnosis of carcinoma is to be considered. The tumour becomes soft to the touch in the later stages, and even fluctuation may be recognised if the abscess reaches the surface. The abnormal softness of certain areas of the palpable liver surface, in comparison to other portions of the same, in fact, an indistinct fluctuation may eventually be recognised upon palpation, in my experience even when a thin layer of liver parenchyma about a finger-breadth in thickness is situated over the abscess.

Pain.—The palpation of the liver surface is *painful*, the intensity of the pain (of the spontaneous, as well as that caused by palpation and percussion) is quite varying. As a rule, the entire liver is slightly sensitive to the touch, but some areas are especially painful, which correspond either to the region of the abscess or to a circumscribed peritonitis, in which case friction sounds are palpable and audible. The classical shoulder pain is present in the majority of the cases, but should never alone determine the diagnosis, nor should the presence of pains in general determine the pathological picture. *The spleen is never enlarged* in abscess of the liver, except only in those cases in which the abscess occurs in consequence of pylephlebitis or pyæmia.

Accordingly as to whether the abscess perforates externally or internally into one of the abdominal organs, there occurs a discharge of pus, through the skin which gradually becomes necrotic, also with the vomit, with the fæces, with the urine, or with the sputum, leading to empyema or pericarditis, or to peritonitis with their well-known symptoms and signs which need no explanation.

The physical changes of the liver form the most important basis for the diagnosis in abscess of the organ as well as in other affections of the liver. But there are other considerations in the diagnosis of liver abscess, which result from the presence of other symptoms, that are sometimes characteristic and facilitate the recognition of this condition.

Fever.—The most important symptom to be considered is *fever, which, as is well known, is absent in nearly all other affections of the liver.*

If the above-described results of the physical examination unquestionably prove an affection of the liver, *the determination of simultaneous fever considerably restricts* the diagnostic considerations regarding the affection present. Aside from a rise of temperature in carcinoma of the liver, which is rare, and the otherwise well-characterized symptom-complex of cholelithiasis, only acute yellow atrophy of the liver is to be considered, which is usually accompanied with fever in the first and last stages of its development, but which, at the same time, presents such a clinical picture, differing from liver abscess, by the progressive decrease of liver dulness, that mistaking one disease for the other is hardly possible. The diagnosis is more certain if the fever, as is usually the case in abscess of the liver, is intermittent or remittent and accompanied with chills. But it must not be forgotten, that the fever may be absent in rare cases. It is necessary, before a positive diagnosis is made, to exclude always *other febrile diseases* which are accompanied with *enlargement of the liver and icterus,* above all *croupous pneumonia.*

Jaundice.—*Icterus,* it is true, is absent in by far the majority of the cases; if it is present and intense, it points to occlusion of the large bile ducts of the hepatic or of the choledochus duct, either because they are compressed by a large abscess on the concave surface of the liver, or a stone produces the obstruction and gives rise to the development of abscess of the liver.

To illustrate this I will quote a case coming under my observation, which is of importance, showing the source of certain abscesses of the liver.

A labourer, sixty-six years old, *was suddenly attacked by pains in the epigastrium.* At the same time dyspeptic symptoms appeared, alternating chills, fever and *icterus,* which gradually increased in the course of the eighteen days of the disease, and which were accompanied with excretion of bile pigment in the urine and decoloration (although not complete) of the fæces. The liver was enlarged, palpable below the costal arch, its border could be felt one finger-breadth above the umbilicus; the surface of the organ was smooth and firmer than normal except near the right border of the costal arch, where it was softer. But there was no evidence of distinct fluctuation. The result of the physical examination only showed an obstacle obstructing the flow of bile in the large bile ducts. It is most probable that the enlargement of the liver was the result of the icterus, considering the smooth condition of the surface of the enlarged organ and the acute character of the disease. The unequal, at some places soft consistence of the organ pointed to suppurative hepatitis, but according to this state, it would not have been possible to make more than a provisional diagnosis, unless fever, *which* was *very intermittent* and accompanied by daily chills on and after the ninth day, had not led to the diagnosis of liver abscess. The most probable cause of this abscess of the liver was considered to be gall-stones owing to the sudden onset of the disease, to the obstruction of the bile ducts, and to the pains, which were localized in certain areas of the liver.

Death occurred on the nineteenth day of the disease. The *autopsy* showed circumscribed purulent perihepatitis to the right, the liver was enlarged with uneven prominences having a flat surface, corresponding to abscesses below. There were about ten abscesses found beneath the surface, over which the liver was found to give the sensation of fluctuation to softness. The ductus choledochus contained sev-

eral small and large (to the size of a cherry stone) concrements, which permitted only of a limited passage of bile into the duodenum upon pressure upon the gall bladder. Section of the liver shows that the abscesses correspond to the branches of the portal vein. The trunk of the portal vein contained thrombi, and in its further course puriform masses, which were connected with the abscesses. *The first (purulent) thrombus was found in the trunk of the portal vein corresponding exactly to a gallstone lying in the hepatic duct.* The pressure of the stone caused, in my opinion, the formation of clots in the portal vein and facilitated the immigration of pus cocci from the hepatic duct, from the intestine through the vascular wall (which had become more or less necrotic owing to the pressure of the adjacent stone). It is most probable that the puriform dissolution of the thrombus of the portal vein occurred in this manner, and from here the dissemination of the pus into the branches of the vessel, resulting in the formation of multiple abscesses in the liver.

Symptoms, like agrypnia [insomnia], delirium, psychical depression, dyspnœa, hiccough (singultus), dyspepsia, emaciation, etc., which are sometimes observed in the course of abscess of the liver, aside from the above-named manifestations, are either so inconstant, or of so ambiguous a nature that it is not permissible to apply them to the diagnosis of an abscess of the liver.

Differential Diagnosis.—However, even with the most careful observation of the morbid picture, it is possible to confound abscess of the liver with other affections, thus with *intermittent fever* (*quotidian*), if the abscesses are situated deep within the body of the liver, if the upward displacement of the right border of the lung is absent, the surface of the organ appears smooth and uniformly consistent, and, at the same time, the fever is typically intermittent. The absent enlargement of the spleen, the inefficacy of quinine, and the prevalence of those symptoms which point directly to an affection of the liver, are in this case the best protection against wrong diagnoses. If the abscess, as usual, is situated at the upper convex surface of the liver, a *right-sided pleuritic exudate* may be simulated. But a differentiation from the latter is usually possible by observation of the course of the upper border of the dulness, inasmuch as the dulness which is caused by abscess of the liver is found, in contrast to pleuritic exudates, at several *circumscribed areas* of the thorax, especially at its anterior surface and in the axillary region, declining towards the spinal column, and a dislocation of the heart to the left can almost never be determined. However, it occurs quite often that (by spreading of the inflammation to the pleura) abscess of the liver is complicated by purulent or serous right-sided pleurisy. A further source of mistakes may be, as is conceivable, a *hypophrenic abscess.* The presence of this condition is favoured by a marked downward displacement of the liver, a smooth, entirely soft tumour in the epigastric region, the dulness of which diffuses to the right and upward, similar to that of a right-sided, moderate, pleuritic exudate (see p. 160), while the soft tumour formed by an abscess of the liver is eventually demarcated from above by firm hepatic tissue. If, as usual, air is found besides pus in a hypophrenic abscess, the differentiation of the latter from liver abscess no longer presents any difficulty. *Abscesses of the abdominal wall in the region of the liver,* caused especially by tuberculosis of the ribs, are situated more superficially than

abscesses of the liver, and are not associated with enlargement of the liver, etc.; a needle thrust into the abscess cavity does not follow the respiratory movements, as is the case in abscess of the liver. A *purulent echinococcus sac* cannot be distinguished from abscess of the liver, unless the history or the demonstration of echinococcus hooklets in the pus evacuated by exploratory puncture furnish positive proofs. A confounding with *cholelithiasis without pus formation* is sometimes possible, because the latter disease is occasionally accompanied with chills or attacks of fever, and, on the other hand, gall-stones may, according to the case that I have just described, be the cause of secondary abscesses of the liver. In such instances, it is usually only to be decided whether gall-stone colics, the diagnosis of which is generally easy, have supervened secondarily by the formation of an abscess of the liver, a decision which can be rendered from the chronic course and the usually fatal termination of the disease, from the gradual formation of a dulness over the normal course of the border between lung and liver, or even a fluctuation on the surface of the liver, etc., after the case has been observed for some time.

Finally, as a rule which should never be neglected, I wish to emphasize the point, to make a positive diagnosis of abscess of the liver only when a source for the formation of pus has been demonstrated or at least can be surmised with a great degree of probability. Special attention should be paid to inflammations in the region of the portal vein—gastric ulcer, typhlitis, dysentery (with the frequent finding of amœbæ in the pus of the abscess), purulent hæmorrhoids, etc., pulmonary gangrene and endocarditis with metastatic propagation of the pus into the liver, through the blood current of the hepatic artery, suppurating wounds of the periphery of the body, wounds of the bones, but, above all, injuries of the skull bones, traumatism of the liver itself, cholelithiasis, etc. If the rule be observed, to make a positive diagnosis of suppurative hepatitis only after a *cause* can be found for the origin of suppuration in the liver, then, it is true, abscess of the liver will be more rarely diagnosticated, but wrong diagnoses will be generally avoided. To conclude, I do not wish to conceal that it is absolutely impossible in a certain number of cases to find the source of an abscess of the liver, and that small abscesses in the liver may take a latent course and elude the diagnosis.

HYPERÆMIA OF THE LIVER

Results of the Physical Examination.—The liver has increased in volume and is uniformly hard to the touch; the surface is smooth, the border blunt, to be felt deeper than under normal circumstances, according to the degree of enlargement either above or below the umbilicus, towards the anterior superior spinous process of the ilium. The demonstration by palpation of liver enlargement is supplemented by the result of percussion. *Icterus* may be present or absent, according to whether the engorgement in the intralobular capillaries is carried to the interlobular capillary net; this may cause compression of the finest interlobular bile ducts or even catarrhal swelling of their walls, preventing the flow of bile and giving occa-

sion for the production of icterus. *Ascites* is but rarely found, if not only cases of well-developed congested liver are considered in the diagnosis, but also the milder grades of this affection.

The more marked the venous engorgement, the sooner is the development of ascites to be expected, either as a partial manifestation of the general venous stasis, or due to secondary engorgement in the portal-vein system originating in the veins of the liver. The *enlargement of the spleen*, which is sometimes observed, is also connected with engorgement of the portal-vein system. But it is usually not present, because the portal stasis, which forms only indirectly, is too insignificant to cause an enlargement of the spleen.

Change in the Size of the Organ.—*Characteristic of the engorgement process in the liver is the change in the size of the organ* according to the degree of stasis present—i. e., the increase of the liver circumference upon increase of the hyperæmia, the decrease upon improvement in the circulatory conditions, which may occur either spontaneously or after therapeutic measures intended to stimulate the activity of the heart have been employed. The decrease in the size of the liver in such cases is accompanied by a diminution of anasarca, hydrothorax, ascites, the disappearance of albumin from the urine, increase of diuresis, etc.—in short, with a lessening of the stasis symptoms in general.

Atrophic Nutmeg Liver.—If the size of the liver diminishes, although the last-named engorgement symptoms do not cease, but, on the contrary, develop more and more, an *atrophic nutmeg liver* may be diagnosticated. If ascites remains of the same degree, whereas the other stasis symptoms decrease, a combination of cirrhosis with congested liver, secondary inflammatory processes which might develop in a hyperæmic liver, should be considered.

Ætiological Diagnosis.—With the last-named diagnostic considerations we have approached a factor which plays a determining *rôle* in the diagnosis of hyperæmia of the liver—viz., the ætiology. *Hyperæmia of the liver must never be diagnosticated unless a cause for its origin can be positively demonstrated.* Attention is to be paid, therefore, whether circulatory obstructions are present which give rise to stasis in the region of the inferior vena cava. Here are to be considered affections of the heart and lungs, marantic conditions with deficient activity of the heart, very rarely compression of the inferior vena cava above the entrance of the hepatic veins by aneurysms, mediastinal tumours, etc.

Hyperæmia of the Liver Due to Increased Blood Supply.—Hyperæmias of the liver which are caused by an *increased influx of blood to the liver* are extremely rare in comparison to those which are brought about by stasis, at least as far as they become subject to diagnosis. Hyperæmias of the liver which are observed in persons who habitually partake of excessive amounts of food and alcoholic liquors may in part belong in this category; it will be possible, in the majority of cases, to demonstrate here also conditions favouring *stasis*. Furthermore, those "vicarious" hyperæmias of the liver which are sometimes observed upon the non-appearance of the menses and which, undoubtedly, are brought about by the action of vaso-motor nerves, should also be counted among the hyperæmias due to increased blood supply, as should also the hyperæmias of the liver in diabetes which, however, can almost never be demonstrated clinically. Those liver congestions which are observed in the course of infectious diseases should, in my opinion, be considered as the beginning of toxic inflammatory processes in the liver.

FATTY LIVER

Results of the Physical Examination.—Enlargement of the liver caused by deposits of fat in the same, presents, as demonstrated by palpation and by percussion, almost always moderate grades, so that the border of the organ, even in very marked fatty degeneration, does not extend below the umbilicus. However, if the patient is placed correctly, and palpation is performed with but slight pressure, it is possible, in most cases, in my experience, to feel the border of the liver and its movements on respiration even in fat people. The border is usually thickened and rounded; *the surface of the liver is quite smooth, the consistence remarkably soft.*

All secondary manifestations—slight portal stasis (ascites and swelling of the spleen, however, never take place), slight discoloration of the fæces, diarrhœas, and icterus—are in doubtful connection with fatty liver and are of no value diagnostically.

Ætiological Diagnosis.—To avoid errors it is advisable always to ask whether the ætiological factors are such in the given case as to ascribe the formation of a fatty liver to them—i. e., whether *universal adiposity* is present or *progressive cachexia*, which cause fatty degeneration of the cells, as clinical experience teaches beyond any doubt.

Thus we see fatty liver occur in the course of *carcinomatous affections*, *anæmic conditions*, and especially in association with *pulmonary tuberculosis*. The cause of this remarkable phenomenon was until recently considered to be a necrosis of the cells, which was brought about by deficiency of oxygen, the N-containing atom-complexes of the necrosing albumin being burned, those without nitrogen remaining and becoming transformed into fat. The same process was considered to be the cause of the development of fatty liver in *phosphorus-poisoning*. But recent investigations have shown that this conception of the formation of "fatty degeneration" is incorrect or at least very unlikely. For it has been proven that the consumption of oxygen in anæmics has by no means decreased (see Anæmia), and also that the transformation of albumin (if we do not take into consideration exceptional cases in severe anæmias) is indubitably normal. Furthermore, Rosenfeld has demonstrated recently that it is impossible to produce fatty liver at all in animals with very little fat, by phosphorus-poisoning, and that that variety of fatty liver which occurs upon poisoning of non-emaciated animals, is brought about by immigration of fat from the well-filled fat deposits of the body into the liver cells, which are becoming subject to albumin degeneration. It has been assumed, therefore, that, in such cases, the degenerated cells of the muscles and glands develop an "avidity" for fat, "go to appropriate the fat, for equalization, to secure their stock on hand from the fat deposits," etc. These views are based upon the fact that fat formation from albuminates, which formerly was generally considered as established, and which, theoretically, actually appears quite plausible, has become very questionable in recent times, and furthermore upon the fact that Rosenfeld observed that the blood of his test animals became markedly fat-containing in the course of phosphorus intoxications. The development of fatty liver in the tuberculous is in some cases greatly favoured, besides by the above-mentioned factors, by abundant administration of cod-liver oil.

Differential Diagnosis.—If attention is paid only to the uniform enlargement and the smoothness of the surface and border of the liver, it is possible to confound fatty liver with *amyloid liver*. But this is easily avoided by considering the consistence of the palpable large liver, which is much harder in amyloid degeneration. At the same time, splenic tumour (which is always absent in fatty liver) and, at least in the majority of cases, the excretion of a clear urine which is poor in sediment and rich in albumin, can almost always be demonstrated in amyloid degeneration. In

inebriates, *hyperæmia and beginning cirrhosis* are to be considered diagnostically, besides fatty liver; a greater consistence of the liver and the early appearance of symptoms of portal stasis point to the presence of the same.

The fatty degeneration of the liver cells which is due to local changes of the liver in tumours of the same, congested liver, etc., is only of pathologico-anatomical, and not of clinical-diagnostic interest.

Cloudy Swelling of the Liver.—As a further expression of the altered metabolism there should be mentioned the "cloudy swelling" of the liver parenchyma. It may possibly be considered as the expression of toxogenous albumin degeneration, caused by the action of the toxine on the cells, during which, according to the degree it attains, secondary fatty degeneration may take place or not. It occurs in the most various *infectious diseases* and *intoxications* and may sometimes be diagnosticated from the uniform swelling, the smooth surface, and the *slightly softer consistence* of the liver. In enteric fever, especially, I have often succeeded in making this diagnosis *intra vitam*. The diagnosis is supported by the appearance of albumin and casts—i. e., if it may be assumed, under such circumstances, that, besides the liver cells, renal epithelia were also irritated by the infective toxine and that they were injured in their function.

Another product of severe disturbance of metabolism, which finds its expression especially in the liver, is the

AMYLOID DEGENERATION OF THE LIVER—AMYLOID LIVER

Principal Symptoms.—The liver in this affection, if the degenerative process exists for some time, is larger, often exceedingly large. The *surface* of the organ is quite *smooth*, the border *rounded*, the consistence unusually *hard*; the tumour develops *painlessly*. *Icterus is absent, as are the symptoms of portal engorgement, except ascites*, which is usually present in the higher grades of amyloid liver, but should not be considered as an effect of stasis of the portal circulation in the liver, but which is partly due to hydræmia being associated with affections leading to amyloid degeneration. Besides amyloid liver there are usually found *amyloid spleen*, palpable as a hard, large tumour, and *amyloid kidney*, which leads to the excretion of a clear urine rich in albumin; very rarely is the liver the only organ that is affected by amyloid degeneration.

Deviations from the Usual Conditions.—Almost all these symptoms which are characteristic of amyloid degeneration, show occasionally deviations from the above-described usual conditions, which must be known in order to avoid making a wrong diagnosis, in a given case.

Sometimes the volume of the liver is *not increased* if the amyloid degeneration has not existed for a very long time; the *border* may remain very *sharp* even in enormous degeneration, as I had the opportunity of observing recently. If the growth is excessively to the left, it may be possible eventually to feel the *incisura interlobularis* as a *deeply gaping furrow which opens widely towards the free* border, a condition which occurs also in other excessive enlargements of the liver, for instance, cancer of the liver, and which may lead to wrong diagnoses if the examination is not made with the greatest of care. The point of the right border of the left lobe of the liver, which is very easily grasped from the gaping interlobular incisura when palpating, creates the impression, in such cases, of being the apex of an enormously enlarged spleen, especially as the tumour is distinctly displaced downward upon inspiration.

The otherwise smooth *surface* may eventually become *uneven* owing to synchronous cirrhotic, especially syphilitic processes in the liver. It is particularly possible that

gummatous nodes permeate the amyloid organ, creating a cancer-like condition on palpation. It is not even always necessary for the consistence of the organ to be hard if a simultaneous fatty infiltration of the liver predominates. In the train of syphilis of the liver, which so often finds expression as amyloid degeneration, there develops sometimes, besides, diffuse syphilitic perihepatitis, and with it *pains* may accompany the development of amyloid liver, so that in these cases the rule does not hold good that the latter affection develops painlessly. *Icterus* is not observed in the course of amyloid liver; there are exceptions to this rule, however—namely, when enlarged portal lymph glands happen to compress the large bile ducts. Then it is possible that compression of the *portal vein* and engorgements in its region may take place, which will develop fully if amyloid liver supervenes upon luetic cirrhosis. It has already been stated, that amyloid *tumour of the spleen* is not a constant accompaniment; but, even if the spleen is affected with amyloid degeneration, it may be possible that *it cannot be demonstrated*, inasmuch as the organ is surrounded by the powerfully developed liver, pressed, and thus prevented from enlarging. Although the combination, amyloid liver, spleen, and kidney is quite frequent, it is well known that one or the other link of this chain may be absent; it is possible also that, in spite of daily examinations of the urine, upon deposition of amyloid substance in the kidneys, *albuminuria may not be found*, as I was recently again taught by a striking example.

Although the diagnosis of amyloid liver, as can be deduced from the above statements, is not always very simple, yet it certainly belongs to those most easy to recognise in the chapter on Affections of the Liver. It is so sharply differentiated from other tumours of the liver by its smoothness and resistance, by the absence of icterus and of portal stasis, etc., that it is unnecessary to enter upon the differential diagnosis. Furthermore, the diagnosis is considerably facilitated and rendered positive if we do not forget that amyloid liver is never an independent disease, but always appears as the expression of a severe disturbance of metabolism. If, therefore, the demonstration is impossible in the given case of long-lasting suppurations, of caries and necrosis of the bones, furthermore, of pulmonary tuberculosis with cavity formation, or, finally, of long-lasting syphilitic infection, the diagnosis of amyloid liver should be avoided, no matter how clearly the entire conduct of the enlarged liver points towards it, and even if simultaneous enlargement of the spleen and albuminuria appear to render the diagnosis certain. Usually still other pathological conditions—intermittent fever, carcinomatous cachexia, leucæmia, etc.—are considered to be the cause of amyloid disease. However, their relation to the development of amyloid disease is very doubtful, according to my experience. At any rate, these ætiological factors are so rarely of importance that it is more advisable not to consider them at all in the diagnosis of amyloid liver or at least only when the complex of the various symptoms of amyloid degeneration of the organism is complete.

CANCER OF THE LIVER

The symptoms of cancer of the liver are distinctly marked in the majority of cases, so that it can be readily diagnosticated. In a smaller number of cases the development of carcinoma of the liver is absolutely latent; the presence of cancer in the liver is then determined only at the autopsy, and could, at most, be only surmised during the life of the person afflicted.

In this case it is a question either of an obscure seat of the carcinomatous nodes in the dome of the liver situated in the excavation of the diaphragm, or, of early excessive development of ascites which renders palpation of the liver impossible, or, finally, of carcinomatous nodes which are just developing, especially secondary carcinoma of the liver.

Results of Palpation and Percussion.—The *volume* of the liver is usually enlarged very early and may sometimes assume enormous dimensions, particularly in carcinoma. The increase in volume sometimes affects the entire organ, sometimes preferably the right, then again the left lobe. If the latter is affected by the formation of the carcinoma, the liver may extend far into the left hypochondrium, so that we may be inclined at first to think of a neoplasm of the stomach or of a tumour of the spleen. But careful palpation will soon prove that the tumour mass which protrudes from the left hypochondrium belongs to the liver.

The border of the liver in this case is felt (gently pressing upon the abdominal walls from below up) *continuously* to pass from the umbilicus to the left into the left hypochondrium. The most characteristic phenomenon of liver tumour is the border, which even in marked infiltration is still always comparatively sharp to the touch; upon deep inspiration of the patient it moves downward. However, the inspiratory locomotion is by no means pathognomonic of tumours of the liver (respectively spleen); it is also found, as I have frequently determined, in gastric tumours if they are not exclusively situated at the lower border of the stomach, but extend up into the hypochondrium. An adhesion of the gastric tumour to the adjacent region is in such cases *not*, as is usually supposed, *conditio sine quâ non* for its locomotion upon inspiration; but of course inspiratory locomotion will follow much more certainly and extensively, if the tumour of the stomach is coalescent with liver or spleen.

Percussion reveals a more or less considerable increase of the liver dulness, which extends principally downward towards the abdominal region, in the direction of least resistance, whereas the borders of liver percussion do not advance towards the lung until the increase in volume has assumed greater dimensions, and that posteriorly more than anteriorly. The upper border of dulness becomes movable upon deep inspiration owing to the fact that the expanding lung enters more or less far into the pleural sinus. The dulness extends upward by several intercostal spaces upon very extensive enlargement of the liver, anteriorly to the fifth rib and above, posteriorly to the scapular angle.

The contour of the liver tumour may in some cases be *seen* to move upward and downward upon respiration under the thin abdominal walls; but the movements of the tumour surface are rarely *felt*, because circumscribed inflammatory changes occur in the peritoneal covering of the liver giving rise to friction, which can be demonstrated by palpation during the respiratory movements.

The consistence of the liver is hard, often hard as stone, aside from rare exceptions of especially soft medullary carcinomata of the liver. The surface and the border show larger or smaller *protuberances* over the right lobe of the liver, which extends below the costal arch as well as over the left lobe in the epigastrium. But the inequality of the surface of the liver is not essential to the diagnosis of hepatic carcinoma. Only quite recently I have observed an enormous carcinoma of the liver which I obtained for section, in which the secondary cancer of the liver formed

only very slight protuberances on the surface of the organ, a condition which even forms the rule in primary infiltrated carcinoma.

The subsequent manifestations which occur in carcinoma, besides the above-named symptoms, especially *icterus, ascites, the conduct of the spleen*, etc., are only of secondary diagnostic significance in comparison to the above changes in the liver, which can be positively determined by palpation and percussion.

Icterus is found in about 50 per cent of the cases. Usually it is only of a moderate degree, because it is generally due to a compression of small bile ducts, not of the great channels of outlet. If an existing icterus increases in such a remarkable manner that the skin turns a deep yellow and the fæces become entirely discoloured, an obturation of the ductus hepaticus or ductus choledochus by growing carcinomatous nodes at the entrance of the liver, or by enlargement of carcinomatously degenerated portal lymph glands, should be thought of, especially if a high-graded development of ascites by pressure of the tumour in question upon the portal vein goes hand in hand with it. If the latter is not the case—i. e., if a high degree of icterus is present with no or with but slightly developed ascites—it is more probable that gallstones, which are not infrequently found in carcinoma of the liver, or a catarrh of the ductus hepaticus or ductus choledochus, can be observed as the cause of the total occlusion of the flow of bile.

Ascites is usually only of medium intensity, if we do not consider the above-mentioned exceptional case, that the tumour compresses the portal vein. However, ascites can be demonstrated in the majority of cases; it usually supervenes during the later stages of the affection, especially when the energy of the heart slackens and the carcinomatous marasmus becomes predominant. Exploratory puncture will reveal either bright, cloudy, or bloody fluid in the peritoneal cavity. The latter condition, blood in the ascitic fluid, points decidedly to the presence of carcinoma of the liver, in case of a doubt whether the ascites is due to carcinoma or, on the other hand, to cirrhosis or amyloid of the liver. It is possible, even with a marked development of the ascites, to feel the enlarged liver through the fluid if the latter, by shock palpation, is for a few moments pushed away from the liver.

Enlargement of the spleen is absent in carcinoma of the liver in contrast to other affections of the liver, *a fact which is always to be considered in the diagnosis.* Rare exceptions occur, if, for instance, a direct compression of the portal-vein trunk occurs, and with it a stasis in the region of the portal vein.

Pain may be of more diagnostic significance in this affection of the liver than in any other, because it is almost always excruciating; but it may be entirely absent, and this symptom should, therefore, be considered only of secondary diagnostic significance. The character of the pains, their lancinating tendencies, and their radiation to the right shoulder are absolutely unimportant factors diagnostically.

All other symptoms which are observed in cancer of the liver will, although they do not determine the diagnosis, tend to supplement it in one or the other respect; thus the presence of a cancer cachexia, of dyspepsia, the condition of the urine, etc. It is of some importance that the latter sometimes contains pigment, especially melanin, the presence of which in the urine points to pigment cancer in the body; although exceptionally it has been also found in non-pigmented cancer of the liver. Of importance is also the enlargement of peripheral lymph glands in the course of the disease; however, valuable as this symptom may be for the diagnosis, we have rarely a chance to determine the enlargement of inguinal and jugular lymph glands.

Differential Diagnosis.—It is usually not difficult, if the above-mentioned symptoms are duly considered, to decide whether cancer of the liver is present or not. The best mode of procedure in diagnosis is as follows:

After having determined that the liver is the affected abdominal organ, we must decide whether it is affected in the manner characteristic of carcinoma. Easily excluded are, by observation of the consistence, size, and condition of the surface and border of the liver, simple echinococcus, fatty

liver, and abscess of the liver which, owing to their soft consistence, cannot be mistaken for carcinoma of the liver. Neither is it usually difficult to differentiate this latter affection from amyloid liver, inasmuch as the latter, although large and hard, appears quite smooth (especially the border); besides, in amyloid degeneration of the liver the spleen is enlarged, no icterus is present, and the ætiologic basis of the affection is sharply defined. The differential diagnosis between amyloid liver and cancer of the liver becomes difficult only if the former is permeated with large gummatous nodes which then protrude from the liver surface as protuberances, causing the organ to grow to a considerable size. In this case the liver may (as may also occur in carcinoma) become much broader, encircle and, as it were, overlap and compress the spleen, so that the latter is prevented from enlarging, and thus an important differential-diagnostic characteristic is lost. The decision in such a case is brought by the long duration of the disease, by the demonstration of other unquestionable symptoms of lues, of albuminuria (which is but rarely found in carcinoma and only in pronounced weakness of the heart or long-lasting, marked icterus), and, of local symptoms, above all, the cicatricial fissures at the border of the liver. = *Lues.*

Icterus Liver.—The differential diagnosis becomes more difficult *if the liver enlarges in the course of a retention icterus* and becomes coarse; the absolutely smooth condition of the liver surface and of the border, the absence of ascites in spite of most pronounced jaundice, and the but moderate increase in the volume of the organ point, in case of doubt, against cancer of the liver. Emaciation, even if rapid, is not necessarily demonstrative of carcinoma, as it also occurs in the course of retention jaundice. The intensity of the jaundice does not play a part in the differential diagnosis, as it is only possible to confound those rare cases of cancer of the liver which are localized at the entrance of the portal vein into the liver, and which are characterized by a high degree of jaundice, with icterus liver. The smooth surface of the liver and the absence of ascites are also important for the diagnosis of *hypertrophic cirrhosis* in contrast to carcinoma, with which "elephantiasis of the liver" otherwise has in common the enlargement and hardness of the organ, also the jaundice, but from which it is also distinguished by enlargement of the spleen which is absent in carcinoma.

Multilocular Echinococcus.—It is especially difficult to differentiate cancer of the liver from multilocular echinococcus and the other neoplasms which are found in the liver. It is obvious that enlargement of the liver with usually hard protuberances at the surface, as are found in multilocular echinococcus, will give rise to errors. But the long duration of the affection, which may last several years, the very gradual appearance of cachexia and, above all, the almost always supervening *enlargement of the spleen* (absent in only about one tenth of the cases) point to multilocular echinococcus, in case of doubt, whereas jaundice and ascites occur in both affections of the liver, the former very frequently (absent in only one fifth of the cases) in multilocular echinococcus. If a central softening occurs in this latter affection, we will be able to aspirate, by means

exploratory puncture, a smeary mass which contains detritus, cholesterin crystals, and hæmatoidin crystals. *Sarcoma, adenoma,* and the more benign neoplasms of the liver will, of course, also present exactly the same objective symptoms in the liver as carcinoma. A diagnosis of these conditions at the bedside is impossible; only the formation of sarcoma in the liver may be thought of, if the organ is permeated by large (a rare case) nodes, and if tumours in other parts of the body, which can be positively recognised as sarcomata, render the metastatic character of the liver tumours probable, especially if no ascites is present which appears, without exception, to be absent in sarcoma of the liver. Under such circumstances it may eventually be surmised that, exceptionally, another affection than the formation of a carcinoma is present in the liver.

Primary and Secondary Carcinoma of the Liver.—After carcinoma of the liver has been diagnosticated in the manner indicated above, it remains to decide whether it is primary or secondary. This part of the diagnosis is by no means irrelevant, because, with the demonstration of a primary carcinoma in another organ, the diagnosis of carcinoma of the liver becomes exceedingly probable. The stomach is most frequently the organ which in this respect presents the primary carcinomatous affection; furthermore, the mammary gland, the rectum, the uterus and adnexa, the bones, etc. Therefore, it should never be omitted, upon demonstration of carcinoma of the liver, to examine the entire body for a primary carcinoma, especially because secondary cancer of the liver is exceedingly more frequent than primary cancer. A digital examination of the rectum and vagina should be made in every instance, the gastric region should be carefully palpated, and the stomach should be examined with a tube in regard to its acidity. Only if no decrease in the acidity can be demonstrated, and if the examination of the other organs which are most commonly subjected to primary carcinoma has proved negative, may the existence of a *primary carcinoma of the liver* be assumed. A rather smooth surface of the liver generally forms the rule in this condition, whereas multiple, larger, palpable prominences characterize, usually at least, the secondary nature of cancer of the liver. Remarkable in primary carcinoma are the very rapid course of the disease and the sometimes insignificant development of the tumour and of the clinical sequelæ in comparison to the conduct of secondary carcinoma of the liver, in which sometimes enormous swellings of the liver develop which then dominate the pathological picture.

Case of Carcinoma of the Rectum with Enormous Secondary Carcinosis of the Liver.—An example may illustrate what has been stated above: A vine-grower, aged fifty years (admitted to the hospital November 14, 1887, died November 23, 1887), was attacked, seven weeks before admission to the hospital, with constipation, loss of appetite, and pains in the right hypochondrium. The abdomen became swollen, sleep was disturbed, nausea and vomiting were absent.

The examination showed the lower thoracic region much distended, the hepatic region prominent; the liver could be palpated as an enormous, uniformly hard tumour, the border of which was slightly painful to the touch. No protuberance could be felt on the surface; the border was sharp, and could be plainly followed one finger-breadth below the umbilicus. The entire tumour presented itself as consisting of two separate parts; they were divided at their lower border by a deep fissure in the left

parasternal line, upon the right side of which the border of the liver—which could be easily followed from the right—rose abruptly. The tumour on the left side extended from the left parasternal line (rising upward to the costal arch) to the left anterior axillary line. Gradually cachexia set in, œdema of the ankles, jaundice, and vomiting. The *vomitus* contained no blood; *no hydrochloric acid.*

It was undoubtedly an enlargement of the liver, but the origin of the left tumour was questionable—i. e., whether it was a continuation of the liver tumour or a tumour of the stomach connected with the former.

In favour of carcinoma ventriculi were the extension of the tumour in the left hypochondrium to the left axillary line, to which point the enlarged liver could scarcely be presumed to reach; furthermore, the division of the left-sided tumour from that on the right side by a deep fissure, the dyspepsia, and the absence of hydrochloric acid in the vomitus. *Against tumour of the stomach* was the sharpness of the lower border of the left-sided tumour, which became continually more prominent towards the end of life, and its quite uniformly smooth surface and extension to below the left costal arch. The decision was finally brought about by a digital examination of the rectum, for which there really was no urgent reason. It revealed, immediately over the external sphincter, several small, hard nodules, which were attached to the rectal wall and which were arranged like a string of pearls. Death due to marasmus occurred on November 23d.

The *diagnosis* was: *Carcinoma recti, enormous secondary carcinoma of the liver* without palpable formation of nodes, with unusually marked enlargement of the organ in breadth.

The *autopsy* confirmed this diagnosis and explained the peculiar division of the tumour into two separate parts. The enormously enlarged liver occupied the entire breadth of the abdominal cavity and was permeated with the smallest nodes, none being larger than a cherry stone. The left lobe of the liver occupied the left hypochondrium, *the suspensory ligament appeared greatly displaced to the left* (owing to the enormous increase in volume of the organ in all its dimensions), *therefore, the fissure between the two halves of the tumour, which was felt at the bedside, corresponded to the interlobular fissure which was greatly displaced to the left* (from its natural position in right sternal line to the left parasternal line). The anterior wall of the rectum, immediately over the anus, presented a rather hard carcinoma (1.5 cm. thick), which ascended in several nodular cords.

Special Form of Cancer of the Liver.—Necessary as it is to pay attention in the clinical diagnosis as to whether a primary or a secondary carcinoma is present, it is of no importance to the clinician to consider differentio-diagnostically the *special form* of carcinoma in the given case.

We will be able in most cases, by reason of the above-mentioned diagnostic rules, positively to diagnosticate a carcinoma of the liver. However, cases occur in which this is not possible until certain pathological conditions of the abdominal organs, which render the diagnosis of carcinoma of the liver difficult, have been considered or excluded.

Cancer of the Stomach—Cancer of the Pylorus.—Cancer of the pylorus, in my experience, gives mostly rise to doubts in diagnosis, especially if it has caused a downward displacement of the stomach and led to adhesions with the border of the liver. The most important means of differentiation is palpation, and especially that of the border of the liver. We should endeavour, above all, to determine its contours to the left and right of the palpable tumour, respectively below the latter; if we do not succeed in doing this, it points to carcinoma of the stomach. Downward locomotion of the tumour on inspiration is, as has been previously stated, of no account in the differential diagnosis, because the downward-moving

liver may communicate the inspiratory locomotion to a tumour which is adjacent to the liver, and locomotion upon inspiration is frequently characteristic of carcinoma of the stomach as such. Sometimes we may succeed in palpating the border of the liver laterally from the tumour and to follow it along the upper border of the latter as a sharp margin beyond the other side of the tumour, so that it is possible to separate the margin of the liver and the tumour by palpation. I have succeeded in such cases in making a positive diagnosis of carcinoma of the stomach even when an extensive tumefaction in the anterior wall of the stomach was contiguous to the margin of the liver and extended beyond the median line in the epigastrium. Percussion is of no avail in these cases, nor the chemical analysis of the stomach contents, because it happens quite often that carcinoma of the liver is due to a carcinoma of the stomach which remains latent to palpation. The filling of the stomach with water or air is often of no definite value either in a differential diagnostic respect, for only if the tumour of the stomach is not at all coalescent with the liver, and therefore can be moved away from the liver downward or posteriorly in these experiments, will it be possible easily to recognise that the tumour belongs to the stomach.

Carcinoma of the Gall-Bladder.—This tumour, which is difficult also in other respects to differentiate from cancer of the pylorus according to its location, can be recognised as an adnexum of the liver by means of filling and emptying the stomach, simultaneously while controlling percussion, or by means of distention of the stomach with gas, and by the impossibility of preventing the upward locomotion of the tumour on expiration (see Carcinoma of the Stomach), provided no coalescence with the surrounding organs interferes with the certainty of the examination. The fact that a carcinoma of the gall-bladder is rarely due to primary carcinoma of the stomach is also of value in the diagnosis in so far as the stomach contents show normal acidity in primary carcinoma of the gall-bladder. It is true this latter condition is also found in cancer of the duodenum; but in the latter affection it will not take long for secondary dilatation of the stomach to appear, whereas in carcinoma of the gall-bladder this is the case only if the tumour assumes very large dimensions and exerts a constant pressure upon the duodenum. Finally, the superficiality and the shape of the tumour are also to be considered in the diagnosis of carcinoma of the gall-bladder in case of doubt (see p. 218).

Renal Tumours.—As the right kidney and the transverse colon with the right flexure are adjacent to the liver, it is quite feasible that tumours of these organs may also simulate tumour of the liver. Diagnostic errors are especially frequent in large *renal tumours*, and they have happened to every diagnostician, the same as to me, in spite of the greatest care in making the diagnosis. The differentiation is not difficult as long as the renal tumours remain small. Their bimanual examination—i. e., pushing the tumour forward, with one hand in the renal region, towards the other placed upon the anterior abdominal wall—usually allows of no doubt as to the place at which the tumour originated. But, if the tumour of the kidney has grown large, bimanual examination is of no value. Of greater value is the symptom, characteristic of large tumours of the kidney, that the ascend-

ing colon takes an oblique course over them, from above downward, and, if it is not filled with fæces, it has a tympanitic sound. It is understood that an evacuation of the intestine by purges and washing should precede the examination, and it will become necessary eventually to fill the intestine by injections of water or the introduction of gas while controlling percussion, to demonstrate the exact location of the transverse colon. Of importance is the conduct of the *upper border of the liver.* If a renal tumour forces the liver upward, the upper boundary of percussion of the organ will never extend as far up as in a carcinoma of the liver, provided it is of such an enormous extension that the differential diagnosis between tumour of the kidney and tumour of the liver is at all to be considered. It may occur in large growths of the liver that the tumour mass, which more or less permeates the entire organ, pushes the diaphragm to the highest possible point—i. e., to the fourth rib and above! The eventual displacement of the tumour upon inspiration is of less value, as it may not be present even in very large tumours of the liver. As it is often possible to define the boundary between tumours of the stomach and the liver by palpation, so is it often successfully done with tumours of the kidney also, inasmuch as the palpating hand may enter between the margin of the ribs and the tumour, which does not affect the liver, and can discern the upper margin of the tumour.[1] If this cannot be accomplished, the bulging of the ribs at the lower aperture of the thorax will, in such cases of renal tumour at least, not be as considerable and as uniform as in large carcinomata of the liver. This characteristic phenomenon is also of significance in the differential diagnosis of all other tumours not relating to the liver. Of course, the condition of the urine may also be determining in the diagnosis. But care should be exercised here, as the urine may be normal even in large renal tumours, and, on the other hand, hæmaturia and albuminuria may occasionally occur also as a complication, in tumours of the liver.

Tumours of the Intestine.—Tumours which are situated in the ascending colon or in the right flexure, can be differentiated from hepatic carcinoma more readily than renal tumours. If the tumour is caused by accumulation of fæces, the fact that they have a doughy feel or, at least, that their form may be slightly changed by kneading, admits of the positive conclusion that the tumefaction is caused by coprostasis, and still more so if it is diminished in size by purges or enemata, and the note, which was originally dull on percussion, gradually becomes tympanitic. The latter holds good generally also for intestinal tumours which are caused simultaneously by neoplasms and impaction of fæces.

It is of especial importance that, no matter whether it is a question of intestinal neoplasm or of coprostasis, an accumulation of gas always takes place above the location of the tumour; the abdomen appears distended and the other symptoms of intestinal obstruction become apparent—symptoms which are absent in carcinoma of the liver, with but few, exceedingly

[1] An example which proves conclusively that it is possible to discern by palpation renal tumours from the liver, even if this organ is enlarged, will be reported in the discussion of the Diagnosis of Renal Carcinoma (see under Fig. 11).

rare exceptions. Tumours which affect the transverse colon are generally very movable; usually they become displaced downward, owing to the greater motility of this portion of the intestine, and therefore are not of much account in the differential diagnosis.

Tumours of the Abdominal Wall.—Other tumours than those mentioned, tumours of the omentum, cannot, upon close inspection, very well be confounded with tumours of the liver.

Only *large tumours of the abdominal wall,* which are situated in the right iliac region, are to be considered in the differential diagnosis. However, they are for the most part easily distinguished. The following factors point to tumour of the abdominal wall in contrast to cancer of the liver: Insufficient motility of the lower margin of the tumour on inspiration. The tumour can be made to remain stationary on expiration, bulging of the abdominal walls without corresponding extension of the tumour into the abdomen; furthermore, the fact that these tumours can be easily grasped from the abdominal wall and remain palpable even on vigorous contraction of the abdominal muscles or show a feeling of fluctuation in the centre upon palpation. The skin is usually coalescent with the tumour and unmovable at the apex of the tumour.

It is obvious that those symptoms which are characteristic of carcinoma of the liver—ascites, icterus, cancer cachexia, etc.—are of value in the diagnosis; but their absence should not be considered of too great significance.

ECHINOCOCCUS OF THE LIVER

It is well known that echinococcus of the liver occurs in two varieties, multilocular and unilocular. Only the diagnosis of the latter will be considered in this chapter, as the diagnosis of multilocular cyst has already been described in Carcinoma of the Liver. We may state incidentally that, according to the most recent investigations of Mangold, in multilocular echinococcus it is a question of the importation of the ovum of another tænia than of the tænia of unilocular echinococcus.

Results of Physical Examination.—The symptoms of unilocular echinococcus of the liver are so striking that the diagnosis can almost always be made with certainty. The *enlargement of the liver,* which may develop *ad maximum* and which may actually cause the lower portions of the thorax to bulge out, is the first striking phenomenon, also the arching out of the hepatic region in the epigastrium which often, at first glance, creates the impression that it does not affect the entire surface of the liver uniformly.

Percussion reveals a considerable displacement of the borders of the liver downward *and upward*; the upper border of the dulness may then, according to the development of the echinococcus cysts at the convex surface of the liver, be irregular and arched in its course.

Special Results of Palpation.—Palpation yields the most important diagnostic points: The tumour is soft, elastic, fluctuates, and shows, at least in the majority of cases, a *hydatid thrill,* best determined if three fingers, slightly spread apart, are placed upon the skin and percussion is done by short taps upon the middle finger.

That the tumour concerns the liver is easily ascertained by the demonstration of the respiratory displacement of the lower contour of the tumour, etc. (see p. 200 etc). As soon as fluctuation becomes distinct—previous to this a diagnosis of unilocular

echinococcus is out of the question—it is only possible to confound echinococcus with abscess of the liver or extensions of the gall-bladder. Other fluctuating abdominal tumours—hydronephrosis, renal echinococcus, aneurysm, ectasis of the urinary bladder, cysts of the ovaries, etc.—should with careful examination hardly be considered diagnostically. Only right-sided hydronephrosis and right-sided renal echinococcus may give rise to mistakes, if careful attention is not given to the course of the lower border of the liver and to the respiratory motility of the tumour, and the condition of the lower aperture of the thorax is not sufficiently considered, as the latter appears remarkably bulged in large tumours of the liver in contrast to renal tumours. Extensions of the gall-bladder can be recognised as such, by the situation and shape of the tumour in question, above all by the fact that the upper margin of the elastic tumour ends exactly at the border of the liver. Neither is it difficult to differentiate echinococcus of the liver and *abscess of the liver*, inasmuch as, in the former, fever with chills, so characteristic of abscess, and collapse are absent; of course these differential symptoms fail if the echinococcus cyst suppurates. Then only the history (anamnesis) and, above all, exploratory puncture of the tumour may be the determining factors, inasmuch as we may succeed in demonstrating microscopically, besides pus cells, echinococcus hooklets in the fluid extracted from the suppurating echinococcus cavity.

Exploratory Puncture.—*Puncture of the tumour with a Pravaz syringe is really the best means of supporting the diagnosis.* In non-inflammatory echinococcus we obtain, on exploratory puncture, a clear fluid which (at least in my experience almost without exception) is *free from albumin* and contains echinococcus hooklets or characteristically stratified cyst-wall membranes. The fluid containing succinic acid and large quantities of sodium chloride may support the opinion regarding the origin of the material from an echinococcus cyst.

Differential Diagnosis between Echinococcus of the Liver and Pleural Exudate.—The diagnosis between these two affections may be doubtful in cases in which the echinococcus develops principally upward and the diaphragm extends towards the thoracic cavity. The absence of fever points to echinococcus in such a case, as does the unusual course of the upper dulness line which in echinococcus, contrary to the facts in pleural exudate, usually declines towards the spinal column. However, all these characteristics are of a doubtful nature, and here, too, only the result of exploratory puncture is determining, which, however, should not be undertaken carelessly, for we have seen this slight operation followed by peritonitis and spreading of the echinococcus to the peritoneal cavity. It is best to do the puncture immediately before the radical operation which is to be undertaken; sometimes it becomes unnecessary, inasmuch as echinococcus cysts or component parts of the same, after perforation of the cyst into the adjacent organs has taken place, appear in the sputum, vomitus, or fæces, or may even appear through the perforated skin, thus at once clearing up a, so far, doubtful case.

PERIHEPATITIS

Perihepatitic Friction Sound.—The inflammation of the peritoneal covering of the liver is mostly diagnosticated only as a secondary symptom of the various affections of the liver, or possibly also as a downward continuation of a pleurisy through the diaphragm, or as a partial symptom of a peritonitis. The symptom which is most important for the diagnosis, is the peritoneal friction sound in the region of the liver, which can be observed on auscultation and palpation upon deep inspiration of the patient. It is differentiated from pleuritic friction by observing the locality of its occurrence, which can be determined in the epigastrium or below the boundary of the right pleural sinus—i. e., in the mamillary line of the seventh intercostal space, in the axillary line from the ninth rib downward. The friction sound disappears, if in the later course of perihepatitis adhesion occurs between the surface of

the liver and the anterior abdominal wall. All the other symptoms are of minor significance diagnostically, as they do not occur regularly or are subject to various explanations; thus, the pain in cases of acute perihepatitis, the sequelæ of obstruction of the portal vein, or occlusion of the bile ducts in the development of inflammation at the entrance of the portal vein into the liver (porta hepatis), dyspeptic symptoms, glycosuria, etc.

CHANGES IN FORM AND POSITION OF THE NORMAL LIVER

Corset Liver is the only one of the changes in form of the structurally unaltered organ which is to be considered practically. Its presence is often a source of error for the inexperienced diagnostician. The tight-lace lobe—i. e., that portion of the liver which is situated beyond the lacing groove, especially of its right lobe—is prolonged downward, frequently to the ilium; it is very movable, because it is connected with the upper portion of the liver by means of the atrophic transverse portion, the lacing groove, and it may be harder owing to secondary connective-tissue development. Thus, especially if an intestinal coil enters the lacing groove, it may appear as though the tight-lace lobe were a movable mass not in connection with the liver. This may give rise to the question whether movable kidney, intestinal tumour, etc., may be present.

The decisive factor for the diagnosis is, in my opinion, not so much the demonstration of a motility of the lower border of the "corset" lobe upon respiration, and much less an eventual continuity of the dulness of the upper and lower portions of the liver, but rather the *following up, by palpation, of the lower border of the liver.* It is best to start at the epigastrium, palpating from here the usually sharp border, which slightly overlaps the finger, and demonstrating its continuity with the border of the lowest part of the "corset" lobe by careful, soft palpation. *Of importance, above all, is the angle which is to be palpated between the part of the liver situated to the left, and the "corset" lobe.* The diagnosis is positive, if the latter can be plainly felt. If the corset lobe has become thicker, owing to engorgement, it usually gives the impression of a renal tumour, or of a *floating kidney.* I am convinced, according to experience gained during the last years, that the diagnosis of movable right kidney in women often means a confounding with "tight-lace" liver, a wrong diagnosis which is so much more apt to occur, as such laced-off lobes of the liver, upon bimanual examination, are easily brought out of the renal region by the left hand, towards the anteriorly palpating right hand, and are very apt to impress even an examiner who is well practised in palpation as movable kidney. We are very much surprised, as I can confirm from my own experience, if laparotomy or autopsy in such cases reveal a "corset-" lobe liver as an anatomical substratum, instead of a movable kidney, which was diagnosticated, apparently, with greatest certainty.

Changes of Position.—Besides *left-sided position of the liver in situs viscerum inversus*, there occur various changes in position and situation of the originally normally placed organ. Often it is a question of simple version of the liver around its transverse axis, anteriorly or posteriorly, of "anteversions" or "retroversions." While anteversions of the liver develop principally in women owing to tight lacing, the organ is pushed upward by increase of the contents of the abdominal cavity owing to ascites, ovarian tumours; downward, by pleural exudates, emphysema, etc. These simple changes in position of the liver can easily be demonstrated as consequences of the above-named pathological conditions, and should be well differentiated from real *floating liver*, a rare occurrence, in which the organ has really become disengaged from its connection with the diaphragm, so that intestines, fluids, or tumours enter between the latter organ and the convex surface of the liver. But this forcing away of the liver from the diaphragm is only possible if its natural peritoneal suspensory ligaments, the coronary ligament with its lateral continuations (triangular ligaments) and the supensory ligament become much stretched and loosened or if a congenital continuation exists of the coronary ligament, an actual mesohepar. But even then, it is impossible for the liver to descend unrestrictedly into the abdominal

cavity, to "wander" *ad libitum*, because it is firmly attached to the inferior vena cava and the latter to the spinal column, and a loosening of this close connection is in any case only possible to a very limited extent. A descent of the liver is favoured in certain cases by a gain in weight of the organ due to engorgement processes, tumefaction, etc., and by relaxation in elasticity of the abdominal wall in pendulous abdomen (Hängebauch). These factors, referring to the genesis of floating liver, should be considered in the diagnosis, which does not encounter any great difficulties, the symptoms being quite striking. The shape of the tumour, its sharp, palpable lower border, its descent upon the patient standing, the possibility of grasping the convex surface of the liver, which otherwise is situated in the excavation of the diaphragm, from the right hypochondrium, make it at once appear probable that the palpable, movable organ is the liver. Between the lower border of the lung and the convex surface of the liver, there is found, according to the given case, fluid which can be displaced, if the organ is replaced in its normal position in the excavation of the diaphragm, or a tympanitic sound which disappears on reposition of the liver. Dragging pains and other abnormal sensations were present in every case. This anomaly is found almost exclusively in the female; the two cases I observed happened to be in men. In one of them the liver had descended below the umbilicus; the length of the suspensory ligament was 7.5 cm., that of the left triangular ligament 4 cm.

DISEASES OF THE BILE DUCTS

STENOSIS OR OCCLUSION OF THE BILIARY PASSAGES —JAUNDICE

The result of stenosis or complete occlusion of the bile ducts is more or less well-marked jaundice, which is due to stasis of bile, the flow of which from the bile ducts has become partly or entirely impossible owing to an obstruction.

Obturation of the Cystic Duct.—Those cases are excepted, in which the obstruction is in the cystic duct, when the bile which has formed in the liver can freely flow through the hepatic duct and the ductus choledochus, so that there is no reason for the occurrence of icterus; the gall-bladder will, at the same time, either atrophy or distend. In the latter case, serous transudation in the gall-bladder will cause the development of *dropsy of the gall-bladder,* or empyema of the gall-bladder will be brought about by inflammation and suppuration. It is true that here also, during a certain period of the affection, the bile which was originally retained in the gall-bladder is resorbed; however, this resorption takes place so slowly and the total amount resorbed is so small that no icterus takes place. Jaundice sometimes even fails to make its appearance in incomplete occlusion of the hepatic or choledochus duct, because sufficient bile passes, so that a more marked resorption of bile into the lymph vessels and the blood does not take place. But, in the last-named cases, the obstacle is usually sufficient to cause a stasis of the flow of bile and with it pronounced jaundice.

Symptoms and Effects of Jaundice.—*The diagnosis of jaundice,* therefore, forms in most cases the starting-point for the diagnosis of occlusion of the bile ducts. The diagnosis becomes positive as soon as the jaundice is fairly pronounced. The well-known signs of abundant resorption of bile are: *Yellow discoloration of the skin and mucous membranes* (wh

become more marked upon dislodgment of the blood by pressure) appearing a few days after the occlusion of the biliary passages, *discoloration of the fæces* to a whitish-gray, clay-coloured appearance; if the bile is entirely prevented from flowing, dryness and larger fat-content of the stools, intensely disagreeable smell or peculiarly sour odour of the dejecta and the flatus.

The *abundance of fat in the stools* in jaundice is caused by the omission of bile in the intestinal contents which, as has been previously explained, forms the most essential factor in the intestinal tract. The unresorbed fat which is passed with the excrement, is excreted either in an undecomposed condition, or as calcium, magnesium, or sodium-soap of the higher fatty acids in the shape of needle or bundle crystals. The fat in the icteric stools causes their exquisitely *white colour*; of course, the want of bile pigment contributes to the discoloration.

The cause of the *foul smell* of the excrement of the jaundiced individual is probably in the main due to the fact that the insufficiently absorbed fat encloses the albumin bodies in the intestine, prevents their resorption, and thus favours putrefaction. It may also be that, although a general antiseptic action of bile has as yet *not* been demonstrated, owing to the absence of bile, some varieties of bacteria develop more in the intestine, thus causing certain processes of decomposition to develop more markedly.

The condition of the *urine* is, above all, important for the diagnosis, and jaundice should never be diagnosticated without an exact examination of the urine having preceded the diagnosis. Provided a greater resorption exists, the *urine appears dark-brown with a golden yellow froth*, and the bile pigment in the urine is *chemically* demonstrable by means of the well-known Gmelin reaction.

Besides constituents of bile it is often possible to demonstrate *albumin* in the urine of jaundiced patients. The cause of this albuminuria is due to an anatomical change (swelling) and disturbance of function of the epithelia of the uriniferous tubules by the bile acids. If the irritation of the renal parenchyma is then only enfeebled, nothing but a few casts appears in the urine—i. e., in such cases the chemical demonstration of albumin will be negative in spite of the presence of casts. But as soon as the irritation of the renal tissue becomes more marked and lasts for some time, albuminuria will not be wanting, and then there will sometimes be found the pronounced picture of a *nephritis* with the excretion of blood, epithelial casts, etc., in the urine.

The action of the bile, especially of bile-acid salts, upon the *nervous system* is undeniable in every well-pronounced case of jaundice. The patients are apathetic, lose their mental energy, owing to the depressive action of the bile upon the brain, become tired and sleepy. Xanthopsia occurs sometimes, but very rarely; furthermore, *pruritus* and, sometimes, urticaria occur. Toxic paresis of the myocardium causes, in the majority of cases of jaundice, the (often considerable) *decrease of the blood pressure and of the frequency of the pulse*. But it seems that the latter also depends in part upon a central stimulation of the inhibition apparatus of the heart by the bile constituents, as Weintraud succeeded recently in a case of catarrhal jaundice to remove the icteric bradycardia regularly by the administration of atropine, which, as is well known, paralyzes the peripheral ends of the vagus. Furthermore, the deficient ingestion and utilization of food in jaundice leads to disturbances of metabolism, more

or less pronounced emaciation, hæmorrhagic diathesis, hæmorrhages from the various organs and, finally, to *cholæmic intoxications* with delirium, coma, convulsions, etc., which, at least partly, may be considered due to an increase of disturbances of metabolism.

Enlargement of the Liver in Jaundice.—Among the *local* consequences of bile stasis upon occlusion of the biliary passages affecting the liver proper, there is to be mentioned *enlargement of the liver.* It is quite marked in some cases without our being able to demonstrate another cause post mortem than the dammed-up bile. However, *this enlargement of the liver due to jaundice is not frequent,* in my experience, so that I can only advise, in case a very considerable enlargement of the organ is demonstrable, always to think primarily of other causes of swelling of the liver—carcinoma, elephantiasis of the liver, etc. Only after the latter have been excluded, is it permissible to make the diagnosis of enlargement of the liver due to bile stasis (jaundice liver). It may be, as I have observed, that, in such cases of liver enlargement due simply to jaundice, the volume of the liver may be quite large and its consistence coarse; but both size and hardness of the organ may sometimes, according to my experience, be entirely reduced by an energetic Carlsbad "cure." *If jaundice persists for some time,* the liver may, on the other hand, owing to the reduction of its cells, be gradually *reduced below its normal volume.*

The Gall-Bladder in Icterus.—It is of the greatest importance to determine *the conduct of the gall-bladder in the given case. This organ enlarges* in some instances and protrudes as a long narrow tumour in the right parasternal line, below the border of the liver, to be separated from the contours of the liver by percussion and palpation. But palpation will give reliable results in this case only if the distention of the gall-bladder is very considerable, so that the organ forms a firmly elastic tumour, coarse to the touch. However, it is regrettable that in most cases, in order to determine the size of the gall-bladder, we are restricted to percussion, which generally, especially if the bladder is but moderately filled, gives only uncertain results.

Diagnosis of the Location of the Occlusion of the Biliary Passages.—If it is possible to determine by percussion or palpation an ectasis of the gall-bladder, it points, with prevailing jaundice, to the ductus choledochus (below the entrance of the cystic duct) as a seat for the obstruction to the flow of bile, whereas, in case of occlusion of the hepatic duct and its branches, of course, no filling of the gall-bladder can occur. If the cystic duct is occluded, it is also possible, as has been stated, that ectasis of the gall-bladder occurs, either by transudation or by inflammatory exudation and suppuration in the gall-bladder; but icterus is not present in that case. It is therefore possible to diagnosticate the location of the occlusion of the biliary passages by observation of an eventual coincidence of jaundice and dilatation of the gall-bladder. Exceptions to the given rules occur, although rarely, when special conditions (for instance, valve-like, incomplete closure) modify the usual effects of obturation of the biliary passages in the given case.

Diagnosis of the Cause of the Occlusion.—The diagnosis of occlusion of the biliary ducts always remains incomplete, unless the various *cause*

of this obturation which come into consideration are discussed in a differentio-diagnostic respect, and the decision rendered in favour of one or the other.

But there are many causes of obturation of the bile passages, and it is therefore advisable, for convenience sake, to discuss them in three categories.

Obturation of the Lumen of the Bile Ducts; Catarrh of the Bile Ducts.—1. *Deposit of an obstruction in the lumen of the bile ducts.* In the first place, the frequent *catarrh of the bile ducts* is to be thought of, which by swelling of the bile-duct mucosa and formation of mucous plugs, leads to the obturation of the biliary canals; the resistance to the flow of bile from the bile ducts need be but very insignificant, because the pressure of the secretion of bile is but weak. A duodenal catarrh may also give rise to retention of bile, by a swelling of the mucous membrane in the region of Vater's diverticulum (ampulla of Vater). A catarrhal jaundice may be diagnosticated, if the icterus is in connection with an undoubted gastric or intestinal catarrh, commences without swelling of the liver, leads within a few days to a complete discoloration of the fæces, and terminates without pain in the liver within a few weeks. However, in some cases, catarrhal jaundice may be protracted for months, for instance if, as I have seen in a fatal case of catarrhal jaundice, a lymph gland, which is situated at the entrance of the cystic duct into the ductus choledochus, swells and forms a firm compressor of the lumen of the biliary canal.

Icterus due to Gall-Stones.—Gastro-intestinal jaundice is very apt to be confounded with jaundice due to cholelithiasis. If it is a question of youthful individuals, it is advisable rather to assume catarrhal icterus than *cholelithiasis*, as experience teaches us that the latter almost without exception does not occur until after thirty years of age. Other characteristics of cholelithiasis are, repeated colic with chills, eventually fever and concomitant vomiting, pain upon palpation of the liver, especially in the region of the gall-bladder, and by the liable character of the jaundice and of the attacks of pain. Not until it is possible to exclude these two most frequent causes of icterus due to obturation of the lumen of the bile ducts in the given case, more infrequent factors, foreign bodies which have entered from the intestine, and especially *parasites in the biliary canals* should be thought of. Relatively most often to be considered here is the *echinococcus*, either by perforation of the sac into the bile duct and obturation of the latter by echinococcus cysts, or—at any rate the much rarer case—because the echinococcus proliferates originally in the bile ducts. If the icterus is to be ascribed to this parasite, the other symptoms of the development of echinococci in the liver must be unquestionably present or echinococcus cysts must appear temporarily in the stools, vomitus, or sputum. If numerous ascarides lumbricoides occur in the dejecta, it is at least to be presumed that an ascaris which strayed into the bile ducts gave rise to jaundice, although there can no longer be a question of diagnosis, as little as in obturation by liver flukes (distoma hepaticum).

In a case of my observation, in which a distoma hepaticum obturated the hepatic duct, it was remarkable that, during the course of the affection (probably accord-

ing to the amount of bile that could pass the parasite—the autopsy, it is true, did not give any positive support for this supposition), the jaundice changed in intensity without the association of paroxysms of pain, in contradistinction to gall-stones. If in such a case the characteristic ova occur in the stools, it might be possible to make this bold diagnosis.

This case, owing to the rarity of such instances, may be reported here.

A Case of Distoma Hepaticum.—The patient, aged sixty-five years (admitted to the hospital July 28, 1880, died August 9, 1880), a lock-tender, two and a half weeks previous to his admission to my clinic, was taken ill with loss of appetite and pain in the gastric region, on July 26th with jaundice. The appearance of the stools was unchanged.

July 28th. Palpation of the slightly sensitive abdomen revealed, to the right of the umbilicus, about three finger-breadths from the median line, a small tumour, the skin over which was movable; the liver proper could not be palpated, the abdominal walls being very tense; according to percussion it was *enlarged*; the urine contained much bile.

July 29th. The above-mentioned small tumour, which now, with the completely relaxed abdominal wall, could be very distinctly felt as a firmly elastic tumour, was considered the filled *gall-bladder*; but the adjacent border of the liver could not be felt; *percussion of the liver does not reveal any enlargement.*

August 2d. Jaundice had decreased, the *dulness of the liver* was not increased, on the contrary, *rather diminished*; but the *volume of the gall-bladder was varying.*

August 6th. Liver dulness again increased, whereas the size of the gall-bladder again decreased. Death occurred on August 9th, due to a pneumonia of the left lower lobe.

The *clinical diagnosis* was: obturation of the ductus choledochus and cystic duct. Cause probably a tumour; gall-stones could not be diagnosticated.

The autopsy (Boström) showed: liver diminished in all diameters—flabby. Ductus choledochus not distended; thickening of the wall and stenosis of the duct immediately in front of the mouth of the cystic duct; the hepatic duct contained, about 5 mm. from its beginning, a liver fluke, after the removal of which dark bile welled out of the hepatic duct; the gall-bladder was much enlarged (14 cm. long, 8 cm. wide) and filled with a water-white fluid. The intestine contained bileless fæcal masses, no gall-stones. The region of the hepatic duct in which the distoma was located, contained a granulating ulcerating surface, starting from which towards the periphery a cicatricial stenosis of the ducts had taken place.

2. **External Compression of the Bile Ducts—Tumours in the Porta Hepatis.**—In another series of cases of jaundice it occurs that the bile ducts are *closed by pressure from without.* Thus unilocular echinococcus, above all, a *carcinomatous node of the liver* situated at the porta, or a *lymph gland* of the porta hepatis which is tubercular, amyloid, or carcinomatously *degenerated,* may compress the bile ducts owing to its growth. These conditions may be diagnosticated at least with a certain degree of probability if the clinical picture of the original disease is undoubtedly present, and if, in the course of the affection, a jaundice supervenes which develops slowly and gradually increases in intensity. If the icterus is the first phenomenon and the carcinomatous nodes which become gradually more and more palpable prominences are the secondary phenomena in the morbid picture, we may consider that the cancer formation, as sometimes happens, has commenced in the wall of the bile ducts. *Carcinomata of the duodenum* and of the *head of the pancreas* may give rise to compression of the bile ducts, and may eventually be diagnosticated as causes of jaundice, if a tumour is distinctly felt between the sternal and parasternal lines, and if,

besides, hæmatemesis, bloody stools, secondary gastrectasis, and cancer cachexia take place.

Other Abdominal Tumours.—It happens less frequently that the bile ducts are compressed by *tumours of the kidneys, of the omentum, by retroperitoneal, or ovarian, or uterine tumours* (or sometimes by expansion of the uterus during pregnancy), and only when these tumours assume very large dimensions. Their diagnosis does, as a rule, present no great difficulties and can, of course, not be discussed here. The accumulation of *fæces* in the hepatic flexure and in the beginning of the transverse colon may also be the cause of compression of the biliary passages. Demonstrable chronic obstipation or development of symptoms of intestinal stenosis, the uneven, but doughy feel to the touch, condition of the tumour, its diminution by purges and enemata secures the diagnosis in such cases—especially if these therapeutic measures not only cause the disappearance of the tumour but also that of the icterus. In rare cases, finally, it may be that an *aneurysm* is found to be the cause of the obturation of the biliary passages. In that case, it is a question of an enormous aneurysm of the abdominal aorta, respectively of the cœliac, hepatic, or superior mesenteric arteries. To diagnosticate the last-named aneurysm, it would be necessary, at all events, to palpate a distinctly pulsating tumour and to hear murmurs over the latter. These were, in the few hitherto observed cases, supplemented by abundant hæmatemeses and neuralgic pains caused by pressure of the aneurysm upon the nerve plexuses.

Common to all the last-named affections causing a retention icterus is that, owing to their compressing the biliary passages from without at the porta hepatis, almost without exception, *besides the biliary ducts the adjacent portal vein is affected by the pressure. Therefore, the symptoms of icterus are in such cases associated with those of portal-vein engorgement, ascites, etc.*, a very important fact diagnostically.

3. **Perihepatitis, Hepatic Syphilis, Duodenal Ulcers, Cholelithiasis.**—The last-named remarks will hold good mostly for those cases of *obturation of the bile ducts* which are still to be discussed, and *which are caused by processes of cicatricial atrophy*. Thus, it is possible that *perihepatitis* may become the cause of icterus and be diagnosticated, if a peritoneal friction sound becomes palpable and audible in the region of the liver; then it may be that, in the course of *syphilis of the liver*, if the atrophying connective tissue constricts the bile ducts, retention icterus develops, and, in the same manner, jaundice may occur in consequence of a cicatrized duodenal ulcer. This latter condition may be diagnosticated with a certain degree of probability if, in connection with a duodenal ulcer (the symptoms of which coincide in general with those of gastric ulcer), gastrectasis arises without the possibility of demonstrating a tumour at the outlet of the stomach or in the duodenum, and a high-graded, incurable jaundice which does not change in intensity occurs. If the latter develops in connection with *cholelithiasis* with passage of stones, and if, with the appearance of jaundice, the formerly frequent attacks of colic disappear permanently, a stenosis of the bile ducts may be thought of, caused by cicatrices of ulcers which arose in the respective bile duct due to the passage of stones.

If the obturation to the flow is not so marked that the lumen of the bile ducts is totally occluded; if, therefore, jaundice is only moderate and the fæces not completely discolored, the diagnosis, that jaundice is caused by a stenosis of the biliary passages,

is wanting in its best support. Now other affections, besides those named, which lead to incomplete jaundice, are to be considered—namely, the various affections of the liver already mentioned, which in their course give rise to jaundice: Hyperæmia of the liver, abscess, carcinoma, cirrhosis of the liver and, above all, connective-tissue hyperplasia of the liver and multilocular echinococcus (see table, p. 221).

In connection with the varieties of jaundice so far discussed we must consider another form of jaundice which has been frequently observed recently and which represents an independent disease (*morbus sui generis*), "*Weil's disease.*"

ACUTE FEBRILE INFECTIOUS JAUNDICE—WEIL'S DISEASE

Pathological Picture.—In 1886 A. Weil described in detail a disease which until then was unknown, the most essential symptoms of which presented a rather well-defined clinical picture, and the existence of which has, since then, been determined by a great number of observers. The disease commences acutely with fever without prodromes, suddenly with or without pronounced chills, mostly with headache, vertigo and restless sleep, *vomiting* and *diarrhœa, remarkable weakness*, which symptoms, on the second day, are followed by marked *muscular pains*, which are increased upon pressure. The temperature of the body rapidly rises to 103° F. to 105° F., and remains, with slight morning remissions, at this height from three to six days; the pulse is correspondingly frequent. With an increase of the nervous symptoms—i. e., with supervening delirium and somnolence—*jaundice* of varying intensity sets in on the third to the sixth day, sometimes even with the appearance of clay-coloured stools, painful *swelling of the liver*, and *enlargement of the spleen*, as well as *nephritis*. These symptoms are frequently associated with *hæmorrhages of the skin* and of the *mucous membranes*; epistaxis, bloody sputa, bloody vomit, blood in the urine and stools, ecchymoses; rarely exanthemata, herpes, roseola, etc. During the second week the fever falls by lysis and diminution of the swelling of liver and spleen, of albuminuria and cerebral symptoms, especially also of the pains in the muscles, which, however, remain sensitive and stiff for some length of time. After about a week of absence of fever and apparent convalescence, in about one half of the cases *a relapse* occurs of *the morbid symptoms in a milder form*, lasting about five to eight days, fever, increase of jaundice, albuminuria, etc. In cases that terminate fatally, the severe nervous symptoms and the hæmorrhages as well as the uræmic symptoms again become prominent. To the symptom-complex, during life, the following correspond at the autopsy: parenchymatous degeneration and cellular infiltration of the liver and the kidneys, enlargement of the spleen and hæmorrhages in various organs, among others also in the intestine with superficial erosions of the mucous membrane of the bowels.

Differential Diagnosis.—The claim of Weil's disease as an independent infectious malady is partly proved by the fact that it is essentially different in its symptoms from the clinical picture of other infectious diseases that take a similar course, and partly secured by the demonstration of a specific bacterial generator of the disease by H. Jäger. Weil's disease distinguishes itself under all circumstances from *relapsing* fever, with which it has in common the fever, muscular pains, cerebral symptoms, swelling of liver and spleen, inflammatory irritation of the kidneys and the relapse, by the absence of relapsing fever spirilli in the blood; this also holds good for the differentiation of Weil's disease from that variety of relapsing fever which is distinguished by the prevalence of jaundice—namely, "*bilious typhoid*," which otherwise cannot be distinguished in its symptoms from Weil's disease. *Cryptogenetic sepsis* may also eventually present a pathological picture which is similar to Weil's disease, inasmuch as in the former are also observed parenchymatous swellings of the abdominal organs, diarrhœas, hæmorrhages, and jaundice. A mistake, however, is scarcely possible after some observation. The exquisitely remittent or intermittent fever with frequently recurring chills, the absence of an unquestionable relapse, the occurrence of endocarditis, arthritic suppurations and pains in the bones

in the course of the disease, etc., are such typical symptoms in the morbid picture of septicopyæmia that the differential diagnosis between this affection and Weil's disease cannot long be in doubt. More difficult is the differentiation from *purulent cholangeitis*, the most striking symptom of which is also a febrile jaundice which develops on an infectious basis. The diagnosis in this case is principally supported by abscess formations in the liver which supervene in cholangeitis, by the intermittent character of the fever and the ætiology of the case, especially the previous occurrence of gall-stone colic. It is well known, finally, that jaundice occurs in rare cases as a complication of *enteric fever*; if in this case, besides enteric fever, enlargement of the spleen, diarrhœa, and severe nervous symptoms, there also develop swelling of the liver and nephritis of a severer grade, the diagnosis of Weil's disease may become questionable, especially as an eruption is sometimes observed in the latter disease also. But in reality the differential diagnosis between both affections is easy, because enteric fever does not commence suddenly and not often with a severe chill, and jaundice is not observed as an initial symptom in typhoid fever; furthermore, because the severe cases of enteric fever which are associated with enlargement of the liver, take a much slower course than in Weil's disease; changes in the respiratory organs occur in enteric fever as a rule, in Weil's disease only in exceptional cases, etc., and, lastly, the positive result of the Gruber-Widal reaction in enteric fever does away with the difficulty in the diagnosis.

Bacteriological Findings and Ætiology.—The diagnosis of Weil's disease will probably in future gain considerably in certainty by the *bacteriological examination* of the special case. For, according to the interesting discovery of H. Jäger, Weil's disease possesses a specific generator in the *bacillus proteus fluorescens* which he found in cases of infectious jaundice. This variety of bacilli, of exceedingly varying size and form, from the smallest coccus-like calibre to long threads, almost constantly of a green fluorescence, is, the same as all the proteus varieties, in direct connection with putrefaction. While the proteus, as a rule, causes disease but rarely, it appears that, under conditions favourable for its development (if the nutritive medium is rich in nitrogenous substances, by repeated passage through the animal body, etc.), it becomes distinctly pathogenetic—i. e., that it is able to enter the blood and the tissues of the body and to cause a severe septic infection. Clinical facts also point to this connection of Weil's disease with putrefactive processes, thus the observation of Fiedler, that the disease occurs remarkably often in butchers, and, above all, the result of H. Jäger's investigations. This author saw Weil's disease occur after bathing in a river that was contaminated by corpses of fowls which at autopsy showed signs of jaundice and enteritis and from the organs of which the same (mice-infecting or killing) proteus could be cultivated as from the organs of patients who had died of Weil's disease. The demonstration and culture of the pathogenetic proteus variety was also successfully accomplished with the sediment of the *urine* of living patients suffering from infectious jaundice, so that the examination of the urine of persons who suffer from questionable Weil's disease will, in the future, be of value in a differentio-diagnostic respect.

CHOLELITHIASIS

The autopsy reveals, in many cases, gall-stones which during the life of the patients never gave rise to symptoms. Even if a large number of gall-stones accumulates in the gall-bladder, they can be but rarely felt through the abdominal wall; if I succeeded recently in doing so, I consider it as an accidental incidence of favourably situated cases. As a rule, we have rather to depend upon a symptomatic picture which corresponds to the migration of the stones in the biliary passages.

Gall-Stone Colic.—The most prominent phenomenon in this picture is *gall-stone colic*, which, however, is by no means always typically developed, and which manifests itself by the occurrence of severe pains in the right hypochondrium; they usually commence about four or five hours after a meal and generally extend towards the epigastrium; however, they are concentrated more in the right half of the abdomen and occasionally radiate particularly to the right shoulder-blade and arm. At the same time chills occur, and intermittent fever (similar to the "reflex fever" after

catheterization of the urethra), *icterus*, swelling of the liver, vomiting, hiccough (singultus), diminished diuresis, a feeling of fainting or actual unconsciousness, and convulsions. The duration of the attack varies; it lasts for hours and days, and it may end abruptly.

Jaundice.—*The condition of the gall-bladder and the appearance of jaundice* require special discussion. Icterus is an important but by no means constant accompaniment of the attacks of colic, appears one half to one day after the lodging of the stone, and may last for some time after the paroxysm of pain has passed away—namely, when the stone stops in its course and causes a more lasting closure of the bile ducts. *Jaundice* is absent if the stone is located in the *cystic duct*. If the stone returns to the gall-bladder, as happens occasionally, its movement is of no consequence, after the paroxysm of pain has passed; if it remains in the cystic duct, the colic is followed by dropsy of the gall-bladder. If the stone is located in the *ductus choledochus, the gall-bladder becomes filled with bile and may*—at least in some of the cases of gall-stone colic—*be felt as an elastic tumour as early as during* the attacks, even several hours previous *to the onset of the colicky pains* (Gerhardt). Owing to the occlusion of the ductus choledochus by gall-stones (in contradistinction to obstruction of the choledochus from other causes) there develops, as experience proves, more frequently than the dilatation of the gall-bladder, which was to be expected, *atrophy of the gall-bladder*, inasmuch as an inflammation in the bile ducts, which is caused by the gall-stones, spreads to the gall-bladder and its surroundings and, by pressure and processes of wasting, causes an atrophy of the gall-bladder. A systolic blowing murmur is also sometimes heard at the onset of the attack. After a severe, long-lasting attack there also usually remains, as Gerhardt has found, in the region of the gall-bladder a circumscribed, peritoneal, audible, and sometimes palpable *friction sound* which rises and falls with the respiration and is due to circumscribed inflammation of the peritonæum at the gall-bladder and the adjacent portion of the peritoneal coat of the liver. The peritoneal pain, which is in connection with it and which differs in character from colicky pain, may persist for days after the colicky attack has passed away. In other cases the affection is not restricted to this circumscribed peritonitis, but a perforation of the bile ducts will occur, formation of fistula and expulsion of the stone by various routes, through the skin, the intestine, when symptoms of ileus may occur (see enterostenosis), through the urinary tract, into the peritonæum, etc. It may also occur that impacted gall-stones, if suppurative cocci enter from the intestine through the end portion of the choledochus which is not washed by the flowing bile, cause cholangeitis and pylephlebitis suppurativa or *abscess of the liver*. For an example of this possibility, see p. 190.

Differential Diagnosis.—If we try to impress this symptom-complex as sketched above in its main characteristics, upon our mind, it is usually easy to make the diagnosis of cholelithiasis. However, there are numerous deviations from this typical picture, and every experienced diagnostician knows how often the diagnosis of gall-stone colic is a doubtful one and requires careful differentio-diagnostic considerations:

Gastralgia.—In the first place, the question often arises whether gall-stone colic or *gastralgia* be present. *Gastralgia is favoured by the following symptoms*: The externally visible change in the gastric region, the distention on relaxation of the same, the termination of the attack with eructation, the eventual diminution of the pain by pressure upon the epigastrium, where, as in gall-stone colic, the pressure upon the liver (in the region of the gall-bladder) is painful. Of course the diagnosis becomes clear if the gall-bladder bulges out globularly, if jaundice sets in after the attack of colic, and gall-stones are passed in the natural manner with the fæces. However, the last-named symptoms (jaundice, etc.) are, as previously mentioned, not necessary sequelæ of an attack of gall-stone colic, and, at any rate, should not be referred to during the first days of the attack when the diagnosis is doubtful. The relatively most useful characteristic is, in my experience, the painfulness of the liver,

especially of its border upon pressure in the region of the gall-bladder; if this is not present, I leave the diagnosis in suspense until better diagnostic criteria become evident.

Gastric Ulcer.—Still more difficult is often the differential diagnosis between gall-stone colic and *ulcer of the stomach* or duodenal ulcer with cardialgia. I know of more than one case in which gastric ulcer was assumed for some time until a Carlsbad "cure" brought out gall-stones and proved the diagnosis of ulcer to be wrong. The fact that in these ulcerative processes, according to their anatomical seat, the pain is most frequently localized just in the region of the pylorus, near the gall-bladder, and that the palpation of those places causes pain, and excuses the error, especially because the impetus for the onset of the paroxysms of pain in gall-stone colic, as well as in gastric ulcer, is quite usually given by the ingestion of food. It is true, the injurious effect of the latter in gall-stone colic is not so constantly demonstrable as in ulcer, in which, besides, less the ingestion of food as such than the *quality* of the latter causes the occurrence of the attacks of pain. It is also of importance in the differential diagnosis that the pains in cholelithiasis often, after having lasted a short while, alternate with periods of entire well-being, while in ulcer of the stomach or duodenum the paroxysms of pain recur daily for weeks and months, and occur with a certain regularity upon change of position, ingestion of food difficult to digest, etc. It also points directly to the existence of an ulcer of the stomach if the vomitus contains abnormally large amounts of acid. If the acidity is normal, or below normal, no conclusions can be drawn from it, because this occurs in ulcer of the stomach as well, and probably sometimes also in duodenal ulcer. Swelling of the gall-bladder and jaundice, on the other hand, decidedly are in favour of the character of the disease being gall-stone colic, as jaundice is extremely rare even in duodenal ulcer.

Renal Colic, etc.—Colics due to stone in the kidneys rarely give rise to mistakes. The seat of the pains in the lumbar region and their radiation along the ureter towards the testicle and glans penis point to another cause of the origin of the paroxysm of pain than the liver, although the spreading of the pains to the upper half of the body and vomiting may be common to both attacks. The excretion of urine shows, although not always, frequently deviations from the normal. It is voided in small quantities, is dark, contains blood or mucus and, usually, sediment.

Other painful affections of the abdomen, such as peritonitis, typhlitis, intestinal colic, lead colic, etc., are not very apt, upon careful observation, to be confounded with gall-stone colic; but the diagnosis will, correctly, often be doubtful for some time. Thus it may sometimes occur that in general peritonitis the distention of the abdomen and its painfulness to pressure are absent, while, on the other hand, collapse, diminution of urine, and other symptoms, which are also found in gall-stone colic, may erroneously point to the existence of a peritonitis. Naunyn recommends that the mode of respiration be observed. In such cases, in diffuse peritonitis, owing to the arrest of the diaphragmatic movement caused by it, the respiration is purely costal, in contrast to the condition in cholelithiasis.

Nervous Liver Colic.—*Neuralgia of the liver*, which, it is true, is slightly problematical in character, cannot at all be distinguished from cholelithiasis if the latter runs its course without jaundice, without swelling of the gall-bladder, and without the diagnostically significant friction sound in the region of the gall-bladder. This will be the case if the stone which enters the cystic duct, after a short stay, returns into the gall-bladder; and this, it seems to me, according to what I have seen and in view of the fact that the gall-bladder undoubtedly is the main location for the formation of gall-stones, occurs frequently. On the other hand, if no friction sound occurs after several attacks of colic, the diagnosis of "nervous colic of the liver" is at least probable, especially if the colic occurs in nervous, anæmic individuals and alternates with other neuralgias. In such a case it may be assumed that contractures of the unstriped muscular fibres, which end in the walls of the bile ducts, produce the colic-like pain.

Malaria—Intermittent Hepatic Fever [Charcot].—But a mistaken diagnosis is liable to happen in an entirely different direction, a confounding of cholelithiasis with

malarial fever, if cholelithiasis runs its course simulating the clinical picture of an intermittent fever. Aside from the fever already mentioned which (even without any inflammation and suppuration of the biliary passages) may accompany the colicky attack itself, sometimes a morbid picture is observed which, in connection with an attack of gall-stone colic, takes its course with jaundice, swelling of the spleen, and severe attacks of fever which may last for weeks ("*intermittent hepatic fever*"). It is caused by a *purulent cholangeitis*, brought about by the action of virulent intestinal bacteria (especially bacillus coli communis) which, under normal conditions, are found regularly in the most inferior portion of the ductus choledochus and become dangerous only when a stasis of bile occurs, especially by gall-stones. Then the bacteria may, with deficient flow of the secretion, multiply, migrate deeper into the bile ducts, and cause suppuration in and around the biliary passages. The diagnosis of this intermittent hepatic fever assumes a firmer basis if the pronounced symptoms of liver abscess develop. The same as by gall-stones, it is possible that, occasionally, a suppurative cholangeitis may develop upon long-lasting bile stasis, especially in neoplasms. The differentiation from malaria is not difficult after some observation. In contrast to the condition in this affection, the swelling of the spleen in intermittent hepatic fever is moderate, jaundice predominates, and gradually one or the other symptoms of sepsis becomes prominent; whereas in malaria the intermittent fever runs a more typical course, quite regularly intermittent, the swelling of the spleen is larger and constant, the examination of the blood eventually shows the presence of plasmodia, and quinine acts specifically.

DISEASES OF THE GALL-BLADDER

The gall-bladder, as an integral part of the biliary ducts, always takes part in all affections of the latter; cholecystitis, etc., therefore, which occur in the train of cholangeitis, are simply symptoms of those affections. It becomes different, however, if the affection of the gall-bladder assumes a more independent character, or becomes so prominent in the pathological picture that it is easily accessible for the diagnosis. Only three affections of the gall-bladder are of clinical diagnostic significance in this respect:

Œdema of the gall-bladder, the impaction of stones in the same, so that it becomes palpable as a hard tumour, and *carcinoma of the gall-bladder*.

Œdema of the gall-bladder consists in a distention of the organ by serous fluid which, in consequence of transudation from the blood-vessels of the gall-bladder wall, takes the place of the gradually resorbed bile. It is presupposed in this case that the bile can no longer flow through the cystic duct into the gall-bladder, either because a carcinomatous node or a stone, etc., obstructs the communication with the biliary canals, or because a cholangeitis prevents, by a tough mucous plug, or by adhesive coalescence of the duct walls, the entrance of bile into the gall-bladder. Especially valuable for the diagnosis is the most frequent cause of œdema of the gall-bladder, cholelithiasis, in comparison to which all other ætiological factors become insignificant. The symptom most essential for the diagnosis is the demonstration of a smooth, firmly elastic tumour of the long shape of the gall-bladder in the parasternal line which protrudes over and beyond the sharp border of the liver. *The adhesion of the tumour to the liver is proved by the respiratory locomotion*. The tumour is also capable of lateral movements upon palpation and upon change of position of the patient. The motility may eventually be very considerable, as a recent case taught me, in which the tumour the size of an egg (which on laparotomy proved

to be the gall-bladder filled with pus and stones), could be displaced far to the left in the epigastrium. The circumference of the tumour can be defined by percussion; but only palpation gives *positive* results when the gall-bladder is filled completely, and if the fundus can be grasped between the fingers. However, in this case the symptom is missing that otherwise points most unquestionably to the fluid contents of the tumour, viz., the sensation of fluctuation.

Differential Diagnosis of Œdema of the Gall-Bladder.—After it has been determined that it is a question of a tumour of the gall-bladder, of tumour that is not solid, it becomes necessary to consider, respectively to exclude, other tumours in the neighbourhood of the gall-bladder which contain fluid, viz., abscess and echinococcus cyst. Form and location of the tumour, which *commences exactly at the border of the liver—i. e., does not extend to the surface of the liver*—here protect against a wrong diagnosis. Next are to be excluded ovarian cysts which extend to the region of the gall-bladder, and hydronephrosis. As it is possible that the tumour in dropsy of the gall-bladder sometimes assumes excessive dimensions (the size of a head and more) and, of course, is not accompanied by icterus, confounding it with such cysts is possible; but against this, apart from the form of the tumour, the determination of a direct connection of the same with the liver, especially inspiratory locomotion and the absence of expiratory fixation of the tumour, is important, whereas the demonstration of a connection with the kidney and the sexual organs cannot be accomplished; furthermore, the observation of a more *irregular, lateral growth* points absolutely against the presence of dropsy of the gall-bladder.

Impaction of the Gall-Bladder with Gall-Stones.—If the tumour, as happens in complete filling of the gall-bladder with fluid, is very resistant, so that there may be doubts as to its cystic character, the question suggests itself whether a carcinoma of the gall-bladder, or an organ distended by an enormous accumulation of stones, might not be present. Tumours of the gall-bladder brought about by the last-named contingency are relatively frequent, but generally easy to recognise by palpation, inasmuch as thereby a stony, sometimes distinctly *uneven* condition of the surface can be noticed. It is much rather possible to confound a resistant tumour caused by œdema with a carcinoma of the gall-bladder, which we shall briefly discuss in the next paragraph; it may be stated in advance that a combination of cholelithiasis and carcinoma of the gall-bladder is quite common.

Carcinoma of the Gall-Bladder is, upon the whole, a rare affection; it occurs either as primary cancer, or it originates, having spread by continuity, from a carcinoma of the liver or of an adjacent abdominal organ. The symptoms which render the diagnosis feasible are cancer cachexia and a hard, uneven, slowly growing tumour restricted to the region of the gall-bladder. Other symptoms, which were also observed in cancer of the gall-bladder, viz., vomiting, sometimes hæmatemesis, bloody dejecta, jaundice, ascites, are of no value in the diagnosis, and only of some clinical significance in so far as it is necessary to know that the above-named symptoms have frequently been observed in carcinoma of the gall-bladder, in order not to exclude the presence of cancer of the gall-bladder on account of their presence and erroneously to assume instead a cancer of the pylorus, duodenum, or transverse colon; jaundice was observed in the majority of cases—in about 70 per cent. *Primary* carcinoma of the gall-bladder almost always occurred *in combination with gall-stones*, and, according to the recent investigations of H. Zenker, it is very probable that carcinoma of the gall-bladder may originate in the formation of ulcers and cicatrices in the wall of the gall-bladder, stimulated by the gall-stones, as is similarly the case in cancer of the stomach. It is remarkable that women are attacked much more frequently than men (3 to 1), which may possibly be due to the predisposition of the female to cholelithiasis.

DISEASES OF THE BLOOD-VESSELS OF THE LIVER

PYLETHROMBOSIS—PYLEPHLEBITIS

Diagnostic Criteria Favouring Pylethrombosis.—The diagnosis of occlusion of the portal vein is based upon the subsequent symptoms of portal-vein stasis which were discussed in the diagnosis of cirrhosis: Ascites, swelling of the spleen, hæmorrhoids, gastric and intestinal hyperæmia which may be increased to hæmorrhage. If this symptom-complex is completely developed, we may conclude that the circulation of the portal-vein blood in the liver is obstructed; but, whether the *trunk*, as in pylethrombosis, is obstructed by a thrombus, or whether the *various branches*, as in cirrhosis, are constricted by a diffuse atrophic process, can never be determined at once. *Only if the symptoms of portal-vein stasis develop to their fullest intensity very rapidly*—i. e., *within a few days—it may be assumed that an occluding factor has become prominent which does not affect the various branches and capillaries of the vessel, but the trunk of the portal vein.*

If we consider that an indurative atrophy of the liver, with lobulation of the organ, seems to develop secondarily as a consequence of the occlusion of the portal vein, it follows that in this respect also *cirrhosis* of the liver must resemble the picture of pylethrombosis, and, on the other hand, cirrhosis of the liver has particularly been found to be the most frequent cause of pylethrombosis. The *differential diagnosis*, therefore, should consider principally the *rapid* development of the stasis symptoms and, furthermore, consider, above all, the *œtiology* of the individual case.

Ætiological Diagnosis.—We must investigate whether these symptoms of rapidly occurring portal vein stasis generally develop in marasmus and deficient circulatory conditions, or whether there is a reason for compression of the portal-vein trunk in a demonstrable cirrhosis of the liver, syphilis of the liver, cancer of the liver, gallstones, chronic peritonitis with atrophying cicatricial formations, or in tumours of adjacent organs (cancer of the stomach, tumours of the duodenum, pancreas, or abdominal glands, etc.) which, growing towards the porta hepatis, may cause a compression of the portal vein from without.

Suppurative pylephlebitis is accompanied by intermittent fever, associated with chills and the other signs of *septic infection*—swellings of the joints, pulmonary metastases, severe disturbances of the cerebral activity, and general loss of strength. *The symptoms that point to the special affection of the liver and portal vein are pains in the liver, jaundice*, which is present at least in the majority of cases, and *indications of portal-vein stasis* (ascites, diarrhœa, dilatation of the veins, of the abdominal wall, all in a moderate degree and inconstantly developed). *Enlargement of the spleen* is almost constantly present, because other causes than portal-vein stasis, especially septic infection as such, contribute towards its development. The liver is *not enlarged* unless abscesses develop in the organ. A *suppurative peritonitis* may supervene in the course of the affection.

Ætiological Diagnosis.—The diagnosis of suppurative pylephlebitis is always one of the most difficult to make. Only if the above-named symp-

toms are present in their entirety, the diagnosis may be made, and even then only if, at the same time, a special cause of suppurative pylephlebitis may, with certainty, or at least with great probability, be supposed—i. e., *an inflammatory area in the region of the portal-vein roots* or of the umbilical vein in the newborn. Ulcerative and suppurative processes in the region of the cæcum, due to perityphlitis, are primarily the causes which lead to suppurative pylephlebitis, less frequently hæmorrhoidal inflammations and ulcers of the stomach; furthermore, mesenteric and splenic abscesses. It is also possible that gall-stones may produce a puriform thrombus in the adjacent portion of the portal vein, as we have seen in a previously reported case.

Differential Diagnosis between Suppurative Pylephlebitis and Abscess of the Liver.—So far the ætiology of suppurative pylephlebitis and abscess of the liver coincide. The latter, as is well known, also develops most frequently through the introduction of pus into the liver by the portal-vein circulation, or follows a suppurative pylephlebitis as a subsequent manifestation. This latter affection, therefore, is congruent in some of the cases with abscess of the liver, and, when inflammations in the region of the portal vein are demonstrable, a consecutive formation of abscesses of the liver besides pylephlebitis, or pylephlebitis without the development of such, can only be separated when the abscesses have developed towards the surface of the liver and are palpable. *Abscess of the liver, arising in another manner than by suppuration of the portal vein (by means of the hepatic artery, etc.), is differentiated from suppurative pylephlebitis only by the fact that in the latter the symptoms of portal-vein stasis are developed to a certain extent.* On the other hand, in genuine cases of pylephlebitis there is missing, in contrast to abscess of the liver, swelling of the liver, especially enlargement of the liver upward into the right half of the thorax, and no indication of formation of a prominence and decrease in consistence at the surface of the liver.

ANEURYSMS OF THE HEPATIC ARTERY

This very rare affection manifested itself, in the cases so far observed, by a tumour. The latter should pulsate and show murmurs upon auscultation; however, these symptoms of aneurysm are only theoretically constructed to the demands of diagnosis. What, so far, has actually been observed, were *hæmatemeses or bloody stools*, almost always *jaundice* due to compression of the biliary ducts, and *neuralgic pains* in the region of the liver or epigastrium, caused by pressure of the tumours upon the nervous plexuses in the porta hepatis.

A positive *diagnosis* of aneurysm of the hepatic artery is, therefore, impossible, and only a provisional diagnosis should be attempted in very rare cases.

To facilitate the diagnosis of diseases of the liver, I append a table containing a compilation of the most important diagnostic factors to be considered. According to the details discussed, it is scarcely necessary to emphasize that what is stated in the table only corresponds to the *usual* condition of the symptoms of the various hepatic diseases, and that exceptions to this rule have not been considered.

Table for the Diagnosis of Diseases of the Liver

Size of the Liver.		Consistency of the Liver.			Border of the Liver.			Surface of the Liver.		Jaundice.			Ascites.		Pain.	Enlargement of the Spleen.	
Diminution.	Enlargement.	Soft to fluctuating.	Coarse, a little harder than normal.	Hard.	Smooth to sharp.	Thick, rounded.	Uneven lobular.	Smooth.	Uneven.	Absent.	Rare.	Frequent.	Absent.	Present.	Present.	Absent.	Present.
Simple atrophy.	Abscess of the liver.	Fatty liver.	Simple atrophy.	Cirrhosis.	Fatty liver.	Fatty liver.	Cirrhosis (only in rare cases at all palpable).	Hyperæmia.	Cirrhosis.	Amyloid.	(Only if bile ducts are directly affected by the pathological process in:)	Abscess.	Fatty liver.	Carcinoma.	Echinococcus multilocularis.	Carcinoma.	Echinococcus unilocularis (rarely by stasis in the portal-vein system).
Atrophic nutmeg liver.	Diabetic liver.	Abscess of the liver.	Icterus liver.	Syphilis of the liver.	Icterus.	Hyperæmia.	Abscess.	Fatty liver.	Abscess.	Adhesive pylephlebitis.	Echinococcus unilocularis.	Hyperæmia.	Elephantiasis hep.	Syphilis with cicatrices.	Acute yellow atrophy of the liver.	Fatty liver.	Hyperæmia of the liver.
Cirrhosis.	Icterus liver.	Echinococcus unilocularis.	Hyperæmia.	Connective tissue hyperplasia.	Hyperplasia (sometimes slightly rounded).	Amyloid.	Abscess.	Icterus liver.	Syphilis of the liver.	Fatty liver.	Syphilis of the liver.	Cirrhosis.	Icterus liver.	Echinococcus multilocularis.	Carcinoma.		Syphilis of the liver.
Syphilis of the liver (atrophic form rather rare).	Weil's disease.			Echinococcus multilocularis (becoming soft).	Echinococcus.			Elephantiasis.	Carcinoma.			Carcinoma.	Echinococcus unilocularis.	Cirrhosis.	Syphilis of the liver.		Cirrhosis.
Acute yellow atrophy of the liver.	Fatty liver.			Amyloid liver.	Simple atrophy.			Amyloid liver.	Echinococcus.			Elephantiasis hep.	Abscess.	Pylephleb. spec. adhæsiva.	Abscess.		Echinococcus multilocularis.
	Passive hyperæmia.			Carcinoma.				Leucæmia.				Icterus liver.		Amyloid (through the original process).			Hypertrophic cirrhosis.
	Syphilis of the liver.							Diabetic liver.						Hyperæmia (in later stages constant).			Amyloid liver.
	Leucæmia.							Acute yellow atrophy of the liver.									Furthermore, through general infection caused by acute yellow atrophy of the liver.
	Connective tissue hyperplasia of the liver.																Abscess of the liver.
	Amyloid liver.																Weil's disease.
	Carcinoma of the liver.																
	Echinococcus of the liver.																

Note.—The order of the diseases in these columns is generally so arranged that the respective symptom is the more characteristic of the special affection of the liver, the farther down in the column it is quoted.

DIAGNOSIS OF DISEASES OF THE PANCREAS

Introductory Remarks.—Important as is the *rôle* which the pancreas plays physiologically, exhaustively as it has been studied for decades, the effect of its secretions is still doubtful, and little have we succeeded as yet in determining the clinical symptoms which, *regularly* or *quite certainly*, correspond to the affections of the pancreas as the expression of a suspended or pathologically altered activity of the organ. However, the latest physiological investigations of von Mering and Minkowski regarding the effect of extirpation of the pancreas in animals have yielded extremely important facts. They prove, primarily, that diabetes will regularly occur after the total extirpation of the pancreas. Mellituria failed to appear if only the pancreatic ducts were constricted, and also if a portion of the pancreas which amounted to more than about one twelfth of the organ remained in the body (compare the chapter on Diabetes Mellitus. It is very interesting that Minkowski succeeded in one instance in producing, by partial extirpation of the pancreas, a diabetes which corresponded to the mild form in man (i. e., excretion of sugar in the urine upon administration of carbohydrates, disappearance of the sugar upon ingestion of meat). The utilization of the nutriments in the intestinal canal is also much reduced in the dog, according to Minkowski, after total extirpation of the pancreas, that of albuminous substances as well as that of starch and particularly of fat. Non-emulsified fat is excreted undigested with the fæces, artificially emulsified fat is utilized almost as little as the usual neutral fat, whereas natural emulsions of fat, such as milk, are absorbed at least one half. It is, therefore, not at all astonishing *that, in affections of the pancreas* (and that not only in carcinoma), *almost constantly a rapidly progressing emaciation of the patients was observed.*

In other respects the experimental results recently gained are partly decidedly contradictory to the former observations in regard to the omission of the function of the pancreas, and their clinical bearing cannot yet be fully appreciated. It may possibly be that at least a part of the former, mostly negative experimental and clinical experiences in this chapter, can be explained by the fact that, upon suspension of the activity of the pancreas, that of other glands of the digestive apparatus may interfere compensatorily with the process of digestion, so that the omission of the action of the pancreatic juice is more or less equalized.

Decrease of Indican Excretion in the Urine.—It is well known that one of the effects of the pancreatic juice is the transformation of albuminous substances into peptone and of the latter into leucin and tyrosin, from which, by putrefactive fermentation, skatol, phenol, and, above all, *indol*, are formed. As the latter develops in larger quantity from the pancreas-peptone than from the albuminous bodies of the flesh, probably the peptic action of the pancreatic juice, respectively of the trypsin, may be considered as preparing and favouring the formation of indol. But indol represents the preliminary stage of urinary indican, and so it might be expected that the excretion of urinary indican in pancreatic affections was *decreased*. Very recent physiological investigations (Pisenti), in which the ligature of the duct of Wirsung makes the contents of indican fall to a quarter of the normal, speak in favour of this assumption, but clinical observations do the same. Thus, recently, a case of ileus has been reported by Gerhardi in which the clinical manifestations pointed to an

occlusion of the small intestine, but in which the increase of indican, which otherwise occurs regularly in the latter affection, was absent. The autopsy revealed, as cause for this condition, an occlusion of the duodenum by the swollen, hæmorrhagically inflamed pancreas, the disorganization of which had suspended the formation of indol, and thus evidently had caused the condition that there was absolutely no indican in the urine. As the normal human urine always contains but very small quantities of indican, the absence or the insignificant excretion of indican in the urine in affections of the pancreas would be of diagnostic value only in such cases in which, as in the example just mentioned, according to the character of the affection, an increase of the urinary indican should be surely expected.

Action of Affections of the Pancreas upon the Solar Plexus.—*The close local relations of the pancreas to the cœliac plexus, respectively to the superior mesenteric plexus*, render it obvious that the compression of these abdominal plexuses of the sympathetic nerve by a pathologically swollen pancreas may cause *nervous symptoms*. In fact, attacks of *neuralgic pains* with manifestations of collapse (cœliac neuralgia) have been observed in the course of affections of the pancreas. A reflex *inhibition of the cardiac action* brought about by the pancreas and due to a sudden high-graded irritation of the abdominal sympathetic is also liable to occur.

Acute Hæmorrhages of the Pancreas.—This complication should be especially thought of in the interesting cases of sudden apoplectiform death (Zenker) in which the autopsy did not show any other change in the body which may explain the occurrence of the catastrophe than an extensive *hæmorrhagic* infiltration of the pancreas. The following are some of the symptoms characteristic of this condition: Severe pain in the epigastric region, nausea and vomiting, collapse, strong desire to defecate, rapid death—within half an hour or in a few hours to days; in other cases the (milder) attack is followed by a putrefaction of the pancreas and peritonitis, to which the patient succumbs. These hæmorrhages of the pancreas are caused by atheromatosis of the vessels in the aged, sclerosis of the vessels in the course of chronic nephritis and syphilis, fatty degeneration of the vascular walls, possibly also inflammations of the pancreas.

Jaundice and Ascites.—Of secondary value diagnostically are jaundice and icterus observed in the course of pancreatic affections. These pathological symptoms are brought about solely by the fact that the *enlarged head of the pancreas* may compress the ductus choledochus which descends alongside of it or perforates it, and thus causes a stasis of the bile, or that it presses upon the *trunk of the portal vein* which is situated in the groove-like furrow of its medial circumference, and thereby produces the symptoms of portal-vein stasis—swelling of the spleen, ascites, etc.

According to experience gained at the bedside we must admit that as yet there cannot be any question of symptoms which constantly occur in affections of the pancreas and which fully correspond to physiological research. The *result of palpation* is of slightly better value for the diagnosis of pancreatic affections, as will be seen in the discussion of the diagnosis of the various affections of the pancreas.

Most accessible to diagnosis are *tumour of the pancreas* (especially *cancer* and *cysts of the pancreas*) and *pancreatic stone colic*.

CARCINOMA OF THE PANCREAS

All those symptoms which in the course of time have been named as characteristic of carcinomatous affection of the pancreas have proved to be unreliable upon observation of a larger number of cases and of a more rigid criticism. Only *palpation* gives diagnostically serviceable results and sometimes permits of an at least very probable diagnosis of carcinoma of the pancreas, provided that tumours of other abdominal organs which are to be considered in a differential diagnostic respect, can be excluded with some degree of certainty.

Palpation of the Tumour.—It is well known that, in thin individuals with relaxed abdominal walls, it is easy in quite a number of cases to feel the spinal column from the lower part of the epigastrium and from the umbilical region. I can give the positive assurance that in such cases, beside the spinal column in the region between the right sternal and the parasternal lines, *sometimes the head of the normal pancreas can be distinctly felt through the pylorus and the transverse colon* (as proved by autopsy). This shows that tumours of the pancreas are not infrequently accessible to palpation; all the more as with development of malignant disease a progressive emaciation goes hand in hand.

Differential Diagnosis.—To decide whether the palpable tumour concerns the pancreas (most often the head of the pancreas is affected by the carcinoma) or those portions of organs which are situated laterally or in front of it—viz., pylorus, duodenum, transverse colon, or porta hepatis—it is necessary to make the diagnosis by exclusion.

Cancer of the Transverse Colon.—A *tumour of the transverse colon* is easiest to exclude. It is much more superficial, it can be displaced, it changes its location spontaneously, causes difficulties in peristalsis, and tympanites in the ascending colon. Bloody stools occur in both diseases.

Cancer of the Duodenum and of the Porta Hepatis.—The exclusion of *duodenal cancer* appears impossible, because the pressure of a pancreatic carcinoma upon the duodenum must produce the same symptoms as cancer of the duodenum: *icterus* with swelling of the gall-bladder, *pressure upon the portal vein* with its sequelæ, *neuralgic pains,* and especially also *stenosis of the lumen of the duodenum* with vomiting of non-fæcal masses and secondary dilatation of the stomach. *The demonstration of the pancreatic ferments in the vomitus and, in case of the occurrence of ileus, abundant indican in the urine* (see p. 222), at most would point *against* cancer of the head of the pancreas (which is generally affected by the neoplasm), while, on the other hand, a *longitudinal* form of the tumour, if it, following the course of the pancreas, proliferates from the right parasternal line to the left, would be *in favour* of carcinoma of the pancreas. *Exactly the same points* as hold good in the differential diagnosis between cancer of the duodenum and that of the pancreas, prevail for the differentiation of the latter from *tumours which are concentrated upon the porta hepatis.*

Cancer of the Pylorus.—This affection, which is most frequently to be considered in a differentio-diagnostic respect, can be better distinguished. In favour of this condition are, in contradistinction to carcinoma of the pancreas, the more superficial position, *the marked movability of the tumour* and the absence of the hydrochloric-acid reaction in the stomach contents. *Pancreatic carcinoma* is favoured, aside from the fatty stools, by an *adherent tumour, deeply palpable near the vertebra,* the complication with *jaundice* and with the symptoms indicating *compression of the portal vein,* swelling of the spleen, ascites, etc. Recently van Ackeren demonstrated, in a case of cancer of the pancreas, *maltose* in the urine of the patient, which was excreted in the urine probably because the degenerated pancreas was not able to perform the transformation of this intermediate stage between the carbohydrates and grape sugar into the latter

substance. It is possible, furthermore, that, by pressure of the carcinoma upon the abdominal aorta which descends immediately behind the pancreas, stenotic murmurs may arise and be heard in the linea alba from the epigastrium down. Other symptoms observed in carcinoma of the pancreas: digestive disturbances, epigastric pains, albuminuria, etc., are of no diagnostic value whatever. Diabetes is absent in most cases of cancer of the pancreas, because it is almost never a question of a *total* degeneration of the gland. The surface of the carcinomatous tumour of the pancreas is usually uneven and its consistence hard. But if, on the other hand, the surface of a tumour in the region of the pancreas is smooth and its consistence soft and fluctuating, we may think of the presence of

CYSTS OF THE PANCREAS

The diagnosis of this condition has already been correctly made several times before the operation of the cysts, and has recently gained considerable surgical interest. The size of the tumour may reach that of a child's head and even more and occupy the entire abdominal cavity. It is situated between liver and spleen, pushes the stomach or the transverse colon before it (which can be made particularly distinct by inflation of these organs with carbonic-acid gas), the stomach moves upward, the colon downward, does not show any or but very little respiratory locomotion, but, eventually, pulsation transmitted from the aorta. Important, above all, are the considerable, rapidly progressing emaciation of the body and the, sometimes periodically occurring, paroxysms of pain which are of diagnostic significance in so far as the symptoms of cœliac neuralgia do not occur in any other cyst of the epigastrium. The coincidence of pancreatic cyst and diabetes mellitus which has been observed several times, is to be connected with the experimental total extirpation of the pancreas. The *differential diagnosis* wavers principally between echinococcus of an abdominal organ, ovarian cysts, and hydronephrosis. The characteristics mentioned of pancreatic cysts, the first occurrence of the tumour in the epigastric region should be considered above all.

Results of Puncture of the Cysts.—In doubtful cases it may be possible that *exploratory puncture* will decide regarding the origin of the cyst, inasmuch as the contents of the cyst, if resulting from a pancreatic cyst, will be composed mostly of epithelia, blood corpuscles, and *pancreatic ferments*, the ferment which transforms starch into sugar and, what is more important, emulsifies fat. The fluid of the cyst is also very albuminous, in contrast to the fluid originating in echinococcus cysts which is free from albumin and contains echinococcus hooklets.

The absence of *fatty stools* cannot at all be made use of diagnostically against the existence of a pancreatic cyst, because the cysts, *usually situated in the body and tail* of the pancreas, do not entirely suspend the secretion of pancreatic juice and its passage into the intestine.

STONES OF THE PANCREAS.—PANCREATIC STONE COLIC

The diagnosis of pancreatic stone disease, a rare affection with less than 100 cases on record, may eventually be made correctly, as is proved by a

case of Lichtheim, in which the stones, which were diagnosticated *intra vitam*, were found post mortem in the dilated duct of Wirsung. The most important symptoms are the *attacks of colic* which run a course similar to that of gall-stone colics, and are associated with vomiting and fever. They are distinguished from colic in cholelithiasis by the concentration of the pains in the epigastrium or *left hypochondrium*, by the addition of pronounced *salivation* and the *absence of jaundice*. The latter symptom, however, is not conclusive, for jaundice fails to appear in quite a number of cases of gall-stone colic and, on the other hand, may occur in stones of the pancreas if the ductus choledochus is compressed by a stone (p. 212). But some other symptoms may support the diagnosis of pancreatic stone disease—*diarrhœic, fatty stools* and, above all, the occurrence of *sugar in the urine*. The mellituria appears at first to occur to a slight extent and intermittently, to become permanent later on, when, owing to the irritation of the glandular ducts by the stones, a chronic interstitial pancreatitis has been brought about to take the course of a severe diabetes. The diagnosis gains in certainty if *pancreatic stones* are excreted in the fæces or perforate externally through an abscess, and prove to be such. They are either hard, mortar-like, and consist principally of calcium phosphate or carbonate, or they are half firm, amorphous, light-gray, and especially rich in organic matter and can, in that case, hardly be confounded with stones of other origin.

If all the above symptoms, or at least the majority of them, are found in a case, the diagnosis may be made with great probability, in fact with certainty. But it must not be concealed that, in spite of considerable accumulation of stones in Wirsung's duct, all diagnostic points of support may be absent, such as vomiting, fever, diabetes, stearrhœa and pancreatic stones in the stools, except the epigastric pains, which, of course, are of no value in the diagnosis (case of Nicolas).

To diagnosticate other diseases of the pancreas, such as acute and chronic tuberculous, syphilitic or purulent *pancreatitis, atrophy of the pancreas, even with a degree of certainty*, is *a priori* a *failure*, according to the present status of our knowledge of the diagnostic significance of the above-mentioned symptoms pointing to an affection of the pancreas. It can here only be a question of *conjecture*, and even this is only permissible in cases in which several of those manifestations which favour disease of the pancreas in general, coincide, and a careful diagnosis by exclusion points to an affection of the pancreas.

DIAGNOSIS OF DISEASES OF THE SPLEEN

Introductory Remarks.—Although we know but little more of the physiological significance of the spleen than that it bears a certain relation to the formation of the blood; that the organ, owing to its unstriped muscular fibres, may easily change its volume upon external stimulation of certain drugs, and may be entirely absent or be removed by operation without injury to the life and the functions of the organism, yet the diseases of the spleen are much better known and much more certain of diagnosis than the affections of the pancreas. This is undoubtedly due to the fact that, in the first place, affections of the spleen are considerably more frequent than those of the pancreas, and, secondly, changes in the spleen are much more accessible to physical diagnosis than the diseases of the pancreas. For even slight enlargements of the spleen can be recognised as such with absolute certainty, less by percussion than by *palpation*, which, in examinations of the spleen, I consider the only method which gives actually reliable results. Statistics collected in my clinic of results obtained upon palpation and percussion of the spleen, in comparison with the measurements of the size of the organ at autopsy, showed the uncertainty of the findings by percussion in 40 per cent of the cases, while the result of palpation did not correspond with the autopsy findings in only about 20 per cent. In the latter cases an enlargement of the spleen was found post mortem which had not been determined during the course of the disease, but never a normal size of the spleen if the organ had been considered enlarged *intra vitam*.

Method of Palpation of the Spleen.—It is true, to recognise a small increase in the volume of the spleen by palpation, requires practice and the employment of certain advantages in placing the patient in a proper position. He is to be placed in the half right lateral position—i. e., to assume a posture midway between lateral and dorsal, and to place the left arm, or the left hand, upon the head. The patient is also made slightly to flex the thighs, to obtain as thorough as possible a relaxation of the abdominal walls, slightly to press the back of the head against the pillow, and, finally, to draw a very long and deep breath. The physician stands at the right side of the patient, grasps, with the left hand from the axillary line, the lowest portion of the left half of the thorax and exerts a pressure to the right, while his right hand, which is placed quite flatly upon the abdominal wall (about in the direction of a line from the right pubic crest to the left costal arch), gradually enters softly below the costal arch and examines whether the apex of the spleen can be felt especially at the moment of deepest inspiration. The organ will almost always, if it is a question of an enlargement of the same, spring over the fingers of the examining physician; at the same time consistence and eventual changes in the form of the organ should be observed. *I consider, supported by an experience of many years, that enlargements of the spleen which can be demonstrated by percussion only, and not also by palpation, are at least doubtful*; for this reason I never make a positive diagnosis of enlargement of the spleen in such cases, and, with this reservation, I have avoided many wrong diagnoses, as proved by autopsy.

HYPERÆMIA OF THE SPLEEN—HYPERPLASIA OF THE SPLEEN

There are no reliable symptoms which are of value in the diagnosis, except palpable swelling of the spleen, in this affection of the organ. It is therefore only necessary to *demonstrate*: (1) *Whether the organ which was felt to be enlarged actually is the spleen,* and (2) all affections of the spleen being accompanied by enlargement of the organ, *whether there are any ætiological factors, and if so, which, to allow the diagnosis of hyperæmia of the spleen or simple splenic hyperplasia.*

In regard to the *first* point, the differential diagnosis will not meet with great difficulties in contrast to the diagnosis of other abdominal tumours. Mistaking it for other tumours can be much better avoided than in the case of tumours of the kidney, intestine, and other abdominal organs. The situation of the organ, above all, protects against errors. The tumour of the spleen grows, from the left hypochondrium, and that *diagonally in the direction towards the right pubic crest; its surface can be distinctly* followed from below to the costal arch, *and never admits at this locality of an overlapping of the upper border of the tumour,* because with its upper portion it is completely embedded in the upper vault of the diaphragm (an exception to this rule occurs only when the enlarged spleen happens to be a floating organ). *The position of the organ changes very easily on deep inspiration;* we then generally feel the enlarged spleen advanced far downward, if the fingers are slightly pressed against the anterior point of the organ and the patient is now made to breathe as deeply as possible. To determine the form of the tumour is almost as important as the determination of its position. The form is more or less oval; very characteristic is the blunt *point* which is advanced towards the pubic crest, and which is always *easily grasped*; notches of the border, the so-called crenæ lienis, are rarely to be felt, according to my experience.

Differential Diagnosis.—It is not easily possible, therefore, to *mistake* a tumour of the spleen; at most it may be confounded with a *carcinoma of the gastric fundus.* A wrong diagnosis is avoided, in such cases, by remembering that the upper roundish contour of the tumour can almost be grasped still below the costal arch or in the hypochondrium (upon the entrance of the fingers below the ribs). Besides, the other symptoms of gastric carcinoma are also to be considered in the differential diagnosis. Errors are more easily possible if the *left lobe of the liver is much prolonged to the left* or even, which happens often enough, actually spans the spleen. In this case we should pay especial attention to the *continuous transition of the tumour into the border of the liver in the epigastrium,* to the decidedly rounded point of the organ in enlarged spleen, and to the direction into which the tumour is displaced upon deep inspiration. *This respiratory displacement is always more diagonally in splenic tumours, more straight downward in tumours of the liver.* But I must admit that errors in this respect cannot be avoided in rare cases with tense, thick abdominal walls.

Ætiological Diagnosis of Splenic Tumours.—After it has thus been demonstrated that the enlarged organ actually is the spleen, a microscopical examination of the blood should be made to exclude, under all circumstances, the presence of a leucæmia, and then it should be considered, by careful observation of the *ætiological* factors, which special variety of splenic enlargement may be present. The following facts may be regarded as the leading points in this respect.

Engorgement of the spleen is to be assumed if, besides the splenic tumour, circulatory disturbances prevail in the region of the portal vein or in the liver. Affections of the liver which are causes of such disturbances are especially cirrhosis, rarely syphilis of the liver, and unilocular echinococcus hepatis (relatively often, in about 90 per cent of the cases, is splenic enlargement found in multilocular echinococcus of the liver), and, finally, hyperæmia of the liver, in which latter affection the deflux of the portal-vein blood meets with resistance from the other side owing to congestion of the capillaries. These difficulties, however, are comparatively slight, so that enlargements of the spleen due to engorgement, which occur in chronic affections of the lungs and of the heart from the inferior vena cava, are not as frequent as might be expected theoretically. But if splenic enlargement is found in emphysema of the lungs, in cirrhotic processes of the lungs, or in cardiac affections, the first thought should be of engorgement of the spleen, and the diagnosis of this condition should not be abandoned until very special reasons, such as an overcome malaria, favour another cause of the enlarged spleen. But it is always necessary for a positive diagnosis that the spleen has enlarged owing to stasis from the inferior vena cava, that the liver be found in a condition of passive hyperæmia.

Enlargement of the spleen is found quite regularly in some affections of the liver without the possibility of demonstrating a stenosis of the portal-vein region as cause for the enlargement of the spleen. This is the case in connective-tissue hyperplasia of the liver, in amyloid liver, in acute yellow atrophy of the liver, and in abscess of the liver. The cause of splenic enlargement in hypertrophic cirrhosis is probably a proliferation of connective tissue in the spleen which corresponds to the process in the liver, in amyloid liver a simultaneous amyloid degeneration of the spleen. Acute yellow atrophy of the liver and abscess of the liver (in which, however, the spleen is not always enlarged) evidently give rise to splenic hyperplasia only by the general *infection* connected therewith.

Infectious Enlargement of the Spleen.—The same cause is to be applied to splenic tumours in the numerous cases in which the organ becomes enlarged in the course of the *various infectious diseases.* It is of no value to enumerate the latter individually; it may only be stated that enlargement of the spleen is found most constantly in *malaria* and *enteric fever*, and that the *typhoid spleen*, in spite of its softness, is palpable *almost without exception and with little difficulty*, whereas I, supported by a very large clinical experience and in opposition to others, must state *that I have found infectious swelling of the spleen in croupous pneumonia only in at most one fourth of the cases, in recent syphilis, however, but very rarely.*

Chronic Enlargement of the Spleen Due to Malarial Infection.—*Chronic*

swelling of the spleen occurs very often in malarial districts in persons who have recovered from various attacks of intermittent fever, as well as in such, according to my experience, who have never been subject to a febrile attack. Under such circumstances splenic enlargement is, as a rule, combined with anæmia, whereas in such localities as are absolutely free from malaria, the combination of splenic enlargement and anæmia is rarer.

Frequent as *chronic enlargement of the spleen* is in slowly developing malarial infection, as relatively *rarely* does it develop in the later stages of *syphilis* and in chronic tuberculosis.

Blood, constitutional, and metabolic affections: leucæmia, pseudoleucæmia, rhachitis, etc., also give rise to the formation of splenic enlargement, as will be explained in the discussion of the respective diseases. The diagnosis of such splenic enlargements as are produced by neoplasms, parasites, amyloid, etc., localized in the spleen, will have to be discussed separately.

Finally, there exist in districts which are entirely free from malaria, "idiopathic" enlargements of the spleen, which, as even the autopsy does not give any explanation as to their genesis, are probably due to toxic-irritative processes.

EMBOLISM OF THE SPLEEN—HÆMORRHAGIC INFARCT OF THE SPLEEN—ABSCESS OF THE SPLEEN

So soon as an opportunity is given for the formation and carrying into the arterial circulation of coagula, consequent in certain affections of the lungs, in endocardial diseases of the left auricle or ventricle, aneurysmal dilatation or arteriosclerosis of the aorta from its origin to the splenic artery, an embolus into the spleen and the formation of a hæmorrhagic infarct in the organ may occur. Statistics have taught us that, under such conditions, if emboli are at all formed, the spleen is most frequently affected by the embolus next to the kidneys. It is rarer that local thromboses form in the vessels of the spleen (mostly due to weakness of the heart), especially in cachectic conditions and in certain infectious diseases, most frequently in the course of enteric fever.

Diagnosis of Hæmorrhagic Infarct of the Spleen.—Sometimes the onset of an embolism of the splenic artery is announced by *chills* and vomiting; if a hæmorrhagic infarct develops, soon afterward (mostly also on palpation) a *painful swelling* of the spleen occurs. If this symptom-complex is found in the morbid picture of a cardiac defect, of an aneurysm of the aorta, etc., the diagnosis of embolus of the spleen may be made with a certain degree of positiveness, especially if signs of embolism appear in other organs of the body: Pain in the renal region and hæmaturia, hemiplegia, etc.

Diagnosis of Abscess of the Spleen.—If the thrombus or embolus is saturated with a suppurative virus, *abscess of the spleen forms.* Its *diagnosis* is based upon the following manifestations: Repeated chills, fever with marked remissions and intermissions, severe disturbance of the general health—symptoms which render a suppuration in the body probable. If a swelling of the spleen, which did not exist before, points to the local affection, and if undoubted causes prevail for the formation of an embolism, the diagnosis of abscess of the spleen can be made with great proba-

bility. This diagnosis becomes *certain,* if a *fluctuating area* is observed *on the surface of the spleen,* or if the previously enlarged spleen suddenly diminishes and pus is discharged with the fæces, urine, in the vomitus or sputum. It may also occur that pus perforates into the peritoneal cavity; in this case the signs of peritonitis follow, which usually becomes general and lethal, but eventually also may be circumscribed and terminate in a cure, as I have experienced in a recent case.

With the above-described acute or subacute course and the severity of the morbid picture, if the splenic abscess leads to fluctuations on the surface of the organ, a confounding with other fluctuating accumulations of fluid in the spleen, especially with echinococcus, is impossible, and can only become feasible if the echinococcus sac itself forms pus.

SPLENITIS

Excepting that variety of splenitis which is caused by embolism there is another inflammation of the spleen which is considered as a result of a dissemination of the inflammation from the adjacent organs—from the stomach, from the connective tissue surrounding the left kidney, from the peritonæum or the lungs. It will scarcely be possible to diagnosticate this variety of splenitis with certainty; but it may be assumed with a degree of probability if pain in the region of the spleen and enlargement of the organ can be determined, and if the above-mentioned neighbouring organs are in a condition of intense inflammation or gangrene, or if a traumatism preceded which has led to contusion of the spleen and thus has created a soil favourable for the attack of generators of inflammation.

RUPTURE OF THE SPLEEN

A very intense traumatism may also, instead of causing splenitis, lead to *rupture of the spleen.* The capsule of the spleen is very apt to rupture if it has been excessively stretched by previous swelling of the spleen. The occasional cause of traumatism or of an increase of the intra-abdominal pressure upon straining, cough, vomiting, etc., is not at all necessary for this accident to occur; it may even happen in acute splenic tumours (especially those caused by malarial infection) that it tears solely as a consequence of the rapidly increasing swelling of the parenchyma. The picture of the, upon the whole, rare affection is that of an internal, rapidly fatal hæmorrhage—collapse, acute pallor, absence of pulse, etc. The spleen as cause of these hæmorrhage symptoms is indicated by the statement of the patient that "something has burst in his body," and the concentration of the pains in the left hypochondrium, and especially the fact that the severe morbid picture occurs suddenly in people in whom an acute swelling of the spleen had unquestionably been present until then. However, the diagnosis will hardly ever be more than provisional. [Rupture of the spleen has occurred upon several occasions, due to careless and rough palpation of the spleen in enteric fever, etc.]

AMYLOID SPLEEN

Amyloid degeneration of the spleen is characterized by the formation of a *hard, smooth, thick* enlargement of the spleen. But, as it is *not* distinguished by its hardness from other swellings of the spleen, the diagnosis becomes possible only when reasons for amyloid degeneration in general and other signs of the same are present. It is advisable never to make the diagnosis of amyloid spleen unless long-lasting suppurations, caries or necroses of the bones, phthisis pulmonum, or persistent syphilis preceded the affection of the spleen. This ætiological basis being determined, the

occurrence of a hard swelling of the spleen is very suspicious of amyloid disease. But the diagnosis becomes certain only if, at the same time, the liver enlarges and becomes hard, if the urine contains albumin and presents the other signs of amyloid urine, and if œdema can be demonstrated. The spleen often being the first of those organs which are affected by amyloid degeneration, the above-mentioned other manifestations need not be present besides splenic tumour; but the assumption of amyloid spleen is, at most, a provisional diagnosis.

NEOPLASMS OF THE SPLEEN—SYPHILITIC CHANGES OF THE SPLEEN

Neoplasms of the spleen are rare and can with difficulty only be distinguished from simple hyperplasias of the spleen, *intra vitam*; the diagnosis is possible only if very favourable circumstances coincide. Neoplasm, not a chronic enlargement of the spleen, is, above all, to be thought of *if the surface of the enlarged organ is uneven*, respectively if it shows large, prominent nodes. Not until then is it permissible from a differentio-diagnostic standpoint to consider sarcoma or carcinoma of the spleen, isolated large tubercular nodes, and gummata of the spleen.

Tuberculosis, Carcinoma of the Spleen.—Tuberculosis is probable if it is a question of phthisical patients, especially children, carcinoma, if primary carcinomata exist elsewhere, in the stomach, liver, etc., *sarcoma* if the nodes of the spleen have formed *primarily*, as I have recently seen in a case, or if, a less rare occurrence, general sarcomatosis may be demonstrable and gummata excluded at the same time.

Syphiloma of the Spleen.—The relatively most frequent neoplasm of the spleen is *splenic syphiloma*, as syphilis in general very often leads to changes in the spleen. Besides hyperplasia and amyloid degeneration, already mentioned as originating on the basis of syphilis, gummatous nodes also occur as a result of persistent syphilis. The same as all gummatous nodes, those localized in the splenic parenchyma may also be reduced by proper therapy, and then indentations instead of nodes will be palpable upon the surface of the spleen. These indentations, brought about by connective-tissue hyperplasia and atrophy, occur also as an independent affection of the spleen, as *syphilitic sclerosis of the spleen*, especially in congenital syphilis. The diagnosis of the above-named syphilitic changes of the spleen is facilitated not only by the history (anamnesis), but, above all, by the fact that these changes, amyloid, gummata, and connective-tissue induration, can be simultaneously demonstrated in the liver.

PARASITES OF THE SPLEEN

Echinococcus of the Spleen.—Only echinococcus offers any diagnostic interest, and even this in a very inferior degree, as it occurs rarely alone, but is usually combined with echinococcus of the liver. The diagnosis of splenic echinococcus is based upon the eventual demonstration of a tumour of the spleen showing fluctuation. If the tumour, from the repeatedly described symptoms, has been recognised as a tumour of the spleen and if fluctuation can be demonstrated, it can only be a question of abscess, of a suppurating echinococcus cyst, or of an echinococcus cyst with clear fluid; the first two are accompanied by fever occurring at the formation of pus. Exploratory puncture will reveal the character of the fluid, as has been explicitly discussed in echinococcus of the liver; in fact, regarding the details, I must refer to the diagnosis of the latter affection.

PERISPLENITIS

Diagnosis.—This malady is rarely observed as a consequence of traumatism in the region of the spleen, more frequently in connection with the above-referred-to diseases of the splenic parenchyma (especially hæmorrhagic infarct of the spleen and syphilitic affections of this organ) and with peritonitides which affect the capsule of

the spleen. The diagnosis is easy if the *coarse friction is felt* which is characteristic in respiratory displacements of the spleen. If this friction is only *heard*, the diagnosis is less certain, because then it cannot be distinguished from dry pleurisy in the pleural sinus, in the region of which the spleen is almost entirely placed, in cases in which the spleen is not enlarged. At most, if the sound is stronger in the lower half of the splenic dulness than in the upper, and if, placing the stethoscope below the costal arch, the friction sound is heard louder than upon auscultation of the thoracic wall, can perisplenitis be surmised. It is irrelevant for the diagnosis whether the patient feels pain in the splenic region, especially upon respiration. Perisplenitis which takes a chronic course may cause adhesions with the surrounding organs to such an extent that the spleen becomes firmly coalescent with the thoracic wall and is no longer capable of locomotion.

CHANGES OF FORM AND POSITION OF THE SPLEEN

FLOATING SPLEEN

We have already referred to *changes of form of the spleen* by neoplasms and processes of shrinking due to syphilis; but, besides, there are sometimes found congenital anomalies in the shape of the spleen, especially lobulations by deep fissures, which, in case an organ enlarges which is formed so out of the ordinary, are felt and may occasionally be misinterpreted, unless we are aware of the occurrence of such anomalies of form. There also occur accessory spleens which are suspended from the lower end of the spleen and which sometimes attain the size of a walnut.

Of greater importance for the diagnosis are *changes in position of the spleen*, caused by pressure from above (pleural exudates, etc.) or from below (due to tympanites, neoplasms, etc.). This variety of dislocation cannot be diagnosticated, according to my experience; this holds good not only for a spleen which is displaced upward, but also for the organ pressed downward, because, in the latter case, the spleen, if not enlarged at the same time, does not appreciably descend farther upon respiration and therefore does not become palpable.

Floating Spleen (Lien Mobilis).—On the other hand, the diagnosis of a spleen which, owing to stretching of the gastrosplenic ligament and of the splenic artery and splenic vein, has become extremely displaced, so-called *floating spleen (lien mobilis)*, can be made with ease and certainty. At the location of the splenic dulness, therefore, between the ninth and eleventh ribs, there is found a tympanitic sound which is replaced by a dull sound if the spleen situated at another part of the abdomen is pushed back to the left hypochondrium. The movable organ is usually easily palpable below the left hypochondrium, rarely farther down in the umbilical region or in the left iliac region, or even in the true pelvis! It can be palpated as an oval tumour of the shape of the spleen; eventually, the notch of the spleen may be recognised and the splenic artery palpated. The tumour can be displaced without difficulty in the abdominal cavity, especially also be replaced into the left hypochondrium if the dislocated organ has not become coalescent with adjacent organs in its new location.

The disturbances caused by floating spleen are of a very variable character; sometimes the patient does not complain of anything at all, at other times of feelings of tearing and pressure, of a desire to urinate, constipation, etc. But these symptoms cannot be used in the diagnosis.

Differential Diagnosis.—Floating spleen may be confounded with fæcal tumours of the transverse colon. But the fact that, in this case, *the splenic dulness can be constantly demonstrated in its normal position*, easily guards against a wrong diagnosis, aside from the doughy consistence of fæcal tumours. The same is the case with other movable abdominal tumours of the size of the spleen. Eventually, as I have seen years ago, a *carcinoma* of the pylorus may become extremely movable; in the case I speak of it could easily be displaced into the left hypochondrium(!), but the splenic dulness remained within its normal boundaries. Usually, still other symptoms allow of a positive diagnosis of an affection of the stomach, in spite of the unusual movability of the pyloric tumour. Floating spleen can, according to the

above-mentioned diagnostic rules, also be easily differentiated from movable kidney, which otherwise may cause similar manifestations, as does movable spleen. Only when both organs become movable at the same time, as I was able to ascertain in a recent case, it becomes doubtful whether the organ felt in the abdomen is the spleen or the kidney.

Finally, it must not be forgotten, that a dislocated spleen enlarges for some reason or other, or that a normally located, enlarged spleen, which for this reason has become heavier, is more apt to descend and become floating. It is true that it is no longer possible in such cases to apply the normal contours of the spleen as a criterion whether the palpable organ is the spleen or not; however, here also the enlarged organ can be replaced into the left hypochondrium, and, if the large spleen has descended, the splenic dulness is always wanting at its proper place.

DIAGNOSIS OF DISEASES OF THE DIGESTIVE TRACT

DISEASES OF THE ORAL CAVITY

THE diagnosis of affections of the oral cavity is usually very easy, as the organ which is to be examined is accessible to direct inspection and palpation.

Coated Tongue.—The tongue is normally rose-coloured anteriorly, whereas posteriorly it appears white, even yellowish-white; in some individuals an insular formation of *coating* is shown. Diseases of the oral cavity alter the appearance of the tongue, as do also affections of the stomach in a reflex-nervous manner. *However, it is not permissible to draw diagnostic conclusions from the coating of the tongue regarding gastric and other affections*; for, according to the investigations of J. Müller and Fuchs, coating of the tongue is also found in many quite healthy individuals. Neither does the coating of the tongue bear relation to local affections of the oral cavity (gingivitis, caries of the teeth), unless these interfere with the ingestion of food or with the movement of the tongue. In the normal adult coating of the tongue is found mostly in middle age (in about 60 per cent), in children not as frequently, in the aged only in about 33 per cent. Coating of the tongue in the well is caused by a more marked development of the fascicle-like, horny processes of the filiform papillæ. These latter decrease in length towards the point and borders of the tongue, for which reason the coating of the tongue, as stated, is almost always thickest at the base of the tongue. The horny layer of the filiform papillæ is being shed continually; very large masses of basement epithelia are cast off, especially during mastication of solid food, as has been proved by experiment. If this normal shedding is suspended, in affections which interfere with the ingestion of food or place the tongue in a position of rest by painful affections of the mouth, numbness, etc., a coating is formed by the growing of the fascicular processes and more marked proliferation of the horny layer accompanied with bacteria. The nightly rest is sufficient in some persons to thicken visibly the coating of the tongue. The various coats of the tongue are due to the food ingested, possibly to proliferation of fungi. It might be thought that the reaction of the fluids of the mouth exerts an influence upon the formation of the coating of the tongue, inasmuch as greater alkalinity facilitates the shedding of the horny basement epithelia; but numerous investigations of J. Müller and Fuchs did not lend support to this hypothesis. The correctness of the views developed regarding the cause of coating of the tongue was supported by a statistical investigation of more than 1,800 case histories. Coating of the tongue is found *more often* than normally, and that in about equal frequency, in acute gastritis, disease of the tonsils, enteric fever, scarlatina, and pneumonia, and in about *equal* percentage as in the healthy and chronic affections of the stomach (chronic gastritis, gastric ulcer, carcinoma). It is probable that, in chronic affections of the stomach, the reduction of the general condition of nutrition is accompanied with a slower growth of the horny layer of the filiform papillæ.

Saliva.—*The reaction of the saliva* is always *alkaline*, according to the investigations of J. Müller and Dieminger, who employed better methods than were formerly in use. It is true, the amount of $NaHCO_3$ (which always has an alkaline reaction) in the saliva was often reduced in affections of various kinds; but the reaction of the saliva was, nevertheless, never found acid. The *quantities of saliva* which are se-

creted due to certain irritants fluctuate considerably in various normal individuals, the same as in the sick. A decrease in the amount of saliva is usually found in all diseases which reduce the general nutrition of the patient, and also, although not at all constantly so, in febrile conditions.

Catarrhal Stomatitis.—All portions of the oral cavity are flushed and swollen, with a thickly coated tongue, in *catarrh of the mouth, catarrhal stomatitis*, caused by mechanical, chemical, and thermic irritants affecting the mucous membrane, and some infectious diseases. At the same time there exists first dryness of the mouth, then more marked secretion of mucus, and a pappy taste. It is always easy to recognise catarrhal stomatitis, only sometimes certain difficulties are encountered in finding the cause. It may be brought to mind here that teething in the suckling is accompanied physiologically with a mild or sometimes pronounced catarrh of the mouth.

If a powerful irritant acts, or—and that is undoubtedly the main fact in the majority of cases—if the teeth exert an intense pressure upon the swollen, inflamed mucosa, defects of the mucous membrane are brought about, and now this affection of the mouth presents itself as *stomatitis ulcerosa* (stomacace).

Stomatitis Aphthosa.—The milder form of this affection is the so-called *stomatitis aphthosa*, in which rounded, grayish-white and yellowish-white plaques are found, which are surrounded by a narrow areola, are attached to their base, the papillæ of the mucous membrane of the mouth, and are either cast off entirely within a few days or gradually thin out and disappear. At the point of stratification there remains sometimes a defect in the epithelium, a flat, small ulcer, which heals without forming a cicatrix. The secretion of mucus and saliva is increased, marked fœtor from the mouth is absent, but slight fever may exist; recovery follows in a few days. The aphthæ occupy the entire cavity of the mouth, preferably the tongue, particularly the point of the latter, furthermore on the inner surface of the cheeks and lips, especially in the sulcus gingivarius. Aphthous stomatitis occurs principally in childhood, but may occur in adults also; certain persons become afflicted with it at regular intervals, especially women during menstruation, during pregnancy, and during lactation. In aphthous stomatitis it is a question, according to the anatomical investigations of E. Fränkel, of a fibrinous exudate with gangrene of the epithelia which, being in the process of coagulation necrosis with unaltered leucocytes or such as have become denucleated, permeate the above-named fibrin trabeculæ (pseudodiphtheria). A confusion is scarcely possible, at most with *herpetic ulcers, which, however, are always due to small vesicles*, whereas this never happens in aphthous stomatitis. The aphthous form of stomatitis is differentiated from the graver variety of this disease by the fact that the ulcers of the latter invade more deeply, show a dirty, disintegrated base, and are located especially at the border of the gums and at the opposite part of the cheek; at the same time there exists a marked fœtor *ex ore* in stomacace, while a fœtor is scarcely noticeable in aphthous stomatitis.

Stomacace.—*Ulcerative stomatitis* is principally an affection of childhood, the same as aphthæ, but it occurs sporadically in adults, sometimes also endemically and, very frequently, is due to mercurial poisoning (*stomatitis mercurialis*). The symptoms of the affection are very marked, so that errors in diagnosis are rare. It is characterized by marked fœtor from the mouth, swelling and reddening of the gums around the teeth, gangrenous disintegration of the gums at these places and formation of deep-reaching, easily bleeding ulcers with a dirty, grayish-yellow base either at the margin of the gums or at those places of the labial or buccal mucous membrane which correspond to the row of the teeth; at the same time there exists *salivation* and swelling of the tongue with marginal ulcerations, while the pharynx remains unaffected.

Differential Diagnosis.—It is very apt to occur that ulcerations in stomacace are mistaken for syphilitic ulcers of the mouth, especially if

specific and mercurial ulcers are simultaneously present in the mouth of a syphilitic. The location of the ulcer is decisive; in syphilis the ulcers are also found on the palate and in the pharynx, and especially in the corners of the mouth; they are less apt to bleed and are of a less diffusely gangrenous character, but, on the other hand, if they reach deeper, easily give rise to perforations. It is also possible that a physician who never or only rarely has an opportunity to see scurvy may confound it with this latter affection.

SCURVY OF THE ORAL CAVITY

The general affection scurvy is principally localized in the oral cavity, especially at such regions of the gums in which teeth are found. Scorbutic affection of the mouth is characterized by a considerable, bluish-red, *puffy* swelling of the gingivæ, loosening of the teeth, slow formation of ulcerations (which, with extensive ulceration, also occur on cheeks and lips), especially in those portions of the gums which rise between the different teeth, and by pronounced tendency to hæmorrhages. Tongue and pharynx are free from ulcers, but ecchymoses are found at those areas that are free from ulcers. There exists at the same time salivation, fœtor *ex ore*, but less intense than in stomacace, except if gangrene supervenes in the severest cases of oral scurvy upon the above-described changes. Other symptoms of scurvy are simultaneously found at various parts of the body, especially hæmorrhages in the subcutaneous tissue and in the muscles, particularly in those of the calves, etc. (See Hæmorrhagic Diathesis.)

Tuberculous Ulcers of the Mouth.—*Tuberculous ulcers* occur rarely *in the oral cavity,* relatively oftenest on the *tongue.* They represent *flat, reactionless ulcerations which, later, sometimes become deep-seated, and then simulate tertiary syphilitic ulcers,* and in the base of which tubercle bacilli may sometimes be demonstrated. This makes the diagnosis positive; but generally it may be made from the above-described appearance of the ulcers and from simultaneous, usually far advanced, pulmonary phthisis.

Noma, Gangrene of the Cheeks, Cancrum Oris.—This very rare affection is characterized by hard infiltration of the cheek near the corners of the mouth, becoming gangrenous internally and externally, and leading to perforation of the cheek and casting off of the gangrenous tissue in large, black portions; the jaw also becomes necrotic. The process is concentrated upon *one* half of the face and occurs almost only during middle childhood. Healthy, vigorous children are not affected, but only weak individuals who live under bad hygienic conditions.

Noma is distinguished from anthrax by the strict localization, the comparatively insignificant disturbance of the general health, and, above all, by the absence of anthrax bacilli; from stomacace it is differentiated by the deep and rapid spread of the gangrenous process to the external skin and by the perniciousness of the process. Noma almost without exception terminates in death.

DEVELOPMENT OF FUNGI IN THE MOUTH—BACTERIAL DISEASES OF THE MOUTH

The cavity of the mouth forms a soil which is very favourable for the development of micro-organisms, as it communicates with the external conditions of the individual, its temperature approaches that prevailing within the organism, and it contains masses of necrosed epithelia. It is, therefore, not to be wondered at, that a very great variety of microbes has been found in the oral cavity. It is certain that some of the pathogenic micro-organisms which enter the body are, previous to their later invasion of the interior of the body, lodged and, eventually, pre-cultivated in the cavity of the mouth, thus probably in scarlatina, acute rheumatic fever, etc. (See Infectious Diseases.) Some of those microbes of the mouth cause processes of fermentation in the mouth, favour the development of dental caries, and play a certain part in mouth affections in general. Only some of the more important of the very considerable number of micro-organisms which have been positively demonstrated in the oral cavity, may be briefly mentioned here.

1. **Pathogenic Bacteria in the Cavity of the Mouth.**—Various pathogenic bacteria which, inoculated into animals, produce septicæmia (Kreibohm) and more or less rapidly cause the death of the animals. Furthermore, are found in the oral cavity, respectively in the saliva, of the healthy —Fränkel's pneumo-bacteria, sometimes also Friedländer's pneumo-bacillus, etc.

2. **Leptothrix buccalis** can as yet not be grown in pure culture, forming long thin threads, the contents of which are coloured violet by iodine and acids. They are probably the expression of thread formation of various varieties of bacilli occurring in the mouth, and play an important *rôle* in the genesis of caries of the teeth.

3. **Thrush fungus, oidium albicans,** identical, according to Plaut's investigations, with the mould fungus *monilia candida*, grows in filaments of mycelium, and the proliferating cells may produce distinct alcoholic fermentation. The fungus, in its proliferation in the oral cavity, forms first lentil-sized white, later more yellowish points which cover the mucous membranes of the cheeks and the surface of the tongue in a hoop-like manner, and gradually spreading to the pharynx, œsophagus, and the opening of the larynx. The fungous layer can partly, but always with very great difficulty, be wiped off from the base. The adjacent mucous membrane is usually not inflamed, but sometimes slightly reddened or even slightly ulcerated and sensitive to the touch; the secretion of the mouth is of an acid reaction. The thrush fungus occurs principally in children, but often also in adults if they have become cachectic, usually shortly before death. I have also observed an endemic dissemination in a military hospital. The diagnosis can be made from the above-mentioned appearance of the mouth, and it becomes positive by the microscopical demonstration of filamentous mycelium and proliferating cells.

DISEASES OF THE PALATE AND PHARYNX

Diseases of the soft palate often become affections of the pharynx [owing to continuity of structure]; their diagnosis, therefore, is best discussed together.

ANGINA—PHARYNGITIS

Simple inflammation of the palate and pharynx is one of the most easily diagnosticated diseases; at most some difficulties are encountered in becoming familiar with the nomenclature which has been established by various authors to designate the different grades and positions of the angina.

The best method of classifying the chaos of designations of the anginas is to differentiate between *acute* and *chronic*, and *superficial* and *deep-seated* inflammations (*angina superficialis* and *angina phlegmonosa*). If the changes are concentrated upon the lacunæ of the tonsils, it is permissible to establish, as a variety of the superficial inflammation, a *lacunar* angina, to which special attention has recently been paid.

Acute superficial angina is characterized by the following manifestations; reddening and swelling of the mucous membrane of the soft palate, of the surface of the tonsils and uvula, sometimes of all these structures, at other times individual affection of only one of these structures. Usually a grayish mucus is attached to the surface of the inflamed parts; it is rare that smaller losses of epithelium and formations of flat erosions occur in the neighbourhood of the tonsillar lacunæ, or that œdema forms simultaneously as the expression of a more violent inflammation. In other cases the epithelium of the excretory ducts of the mucous glands swells, and the secretion is retained; the swollen mucous glands then project as small nodes from the surface of the mucous membrane (*glandular angina*), and lead, after they have broken, to the formation of small flat ulcers. The contents of the glands, owing to its tenacity, remains in place in the shape of small gray points and patches, and is often difficult to remove, and impresses the inexperienced observer as a diphtheritic membrane.

Angina Lacunaris.—If the inflammation affects the lacunæ of the tonsils in particular (*lacunar angina*), they become filled with a secretion which is gelatinous, whitish at the onset and, later, inspissated to a cheesy consistence; the microscopical examination of the plugs proves that they consist of accumulated masses of bacteria, fat and leucocytes. This causes the more or less inflamed tonsil to assume a mottled appearance; the number of the thus changed openings of the lacunæ varies greatly. Lacunar angina is often confounded with diphtheria, because the whitish-yellow plugs eventually protrude above the surface, can be wiped off with difficulty or not at all, and sometimes spread membrane-like over the surface of the tonsil. Wrong diagnoses are best guarded against by the observation of the fact that the white membranes are restricted to the lacunar openings, that they usually protrude as small plugs upon slightly increased pressure upon the neighbourhood of the lacunæ, and can then be easily removed. *However, lacunar angina can be differentiated from tonsillar diphtheria with certainty only by the microscopical examination for diph-*

theria bacilli; it should not be forgotten that there are cases of genuine diphtheria which, during their entire course, show the clinical picture of a lacunar angina.

Chronic Superficial Angina.—If a *superficial* angina becomes chronic, and if the *lacunar* form of angina prevails, a further inspissation of the yellowish-white plugs occurs, and with it are brought about a calcification of the secretory masses and the formation of tonsillar concretions, or even putrefaction of the masses giving rise to fœtid breath. The stagnating secretion leads to new inflammatory changes and often to hypertrophy of the tonsils. It is also possible that erosions form at the openings of the lacunæ with ensuing coalescence of the opposite walls and cyst formation or obliteration of the affected lacunæ.

If it is not a question of *lacunar*, but of *diffuse chronic superficial angina and pharyngitis*, the mucous membrane of the parts affected appears in a more dirty red discoloration with abundant secretion of a thickish mucus, which is sometimes mixed with particles of blood. The secretion sometimes causes the pharyngeal wall to appear as though varnished; if the secretion dries, the posterior laryngeal wall seems to be covered with greenish-gray crusts.

Chronic Granular Pharyngitis.—If a chronic pharyngitis concentrates itself upon the mucous glands or upon the adenoid tissue of the pharynx, and if simultaneously an inflammatory hyperplasia of the follicular apparatus sets in, the picture of *granular pharyngitis*, a quite frequent obstinate affection, develops, with the formation of low, reddish-gray, nodular projections between which the chronically inflamed, bluish-red mucous membrane is visible. If the epithelium proliferates more markedly, this becomes noticeable in the form of white discolorations. Similar changes are found if the chronic inflammation principally affects portions of the palate. Swelling of the mucous glands and of the solitary follicles of the palatine arches, constant reddening of the uvula, of the palatine arches, and tonsils is noted.

The other symptoms—fever in the acute form, difficulty in deglutition, disturbances of mastication and speech, which assumes a nasal quality, tickling, hawking, etc.—are to be considered only in a secondary degree, in contrast to the above-mentioned objective changes which are visible on inspection of the pharyngeal cavity and can be determined with certainty.

Acute Phlegmonous Angina.—*Deep-seated, phlegmonous* acute angina is differentiated from the superficial form by the fact that the inflammatory process principally affects the submucosa and shows a tendency to suppuration. The swelling of the affected parts is quite considerable, in keeping with the more intense inflammation; it may occur especially that the arches of the palate and the uvula swell to the thickness of a thumb. But, above all, the tonsils are enormously enlarged, so that they, almost touching each other, nearly completely obstruct the entrance to the pharynx. Now the process either recedes, or suppuration sets in in the tonsils, in the interlacunar connective tissue and in the follicular wall, with perforation of the abscesses to the surface of the tonsils or into their lacunæ.

Then we observe one portion of the surface of the tonsils to be more prominent, and we feel on palpation that it is softer than the surrounding tissue, in fact, fluctuates distinctly before the pus perforates. Besides, œdema may occur in the organs which are contiguous to the phlegmonously inflamed area (œdema of the glottis); furthermore, a spread of the phlegmons to the cellular tissue of the throat (angina Ludovici) or a descent of the pus into the mediastinum, sometimes gangrene of the palate, may supervene as a dangerous complication. It goes without saying that the general state of health is severely disturbed in phlegmonous angina and that the local difficulties of deglutition, speech, etc., may reach a high degree. Such severe phlegmonous inflammations of the pharynx occur either as metastatic processes, especially in the course of puerperal fever, or in connection with scarlatinal angina or scarlatinal diphtheria or erysipelas, or it may also occur primarily, to which fact Senator has recently drawn attention. The latter form, in the severer grades, is accompanied with marked general symptoms; with fever between 103° F. and 105° F., disturbances of the sensorium, with enlargement of the spleen, with nephritis and with gastritis, and is distinguished by the spreading of the phlegmonous inflammation to the neighbouring organs, especially to the larynx, and by its pernicious, fatal course. Milder forms of "*acute infectious phlegmonous pharyngitis*" may be considered such cases of angina, in which, although a pronounced diffusely suppurative infiltration of the mucous membrane and of the subcutaneous tissue of the pharynx does not occur, but only a considerable swelling of the pharyngeal organs takes place, in which, in contrast to ordinary superficial angina, as I have often observed, splenic enlargement and nephritis occur and persist for some time after recovery of the patient.

Retropharyngeal Abscess.—Phlegmonous inflammation of the mucosa and submucosa of the pharyngeal cavity is not very frequent; on the other hand, it is well known that circumscribed abscess formations which are located in the retropharyngeal connective tissue—*retropharyngeal abscesses*—are found quite often. Their symptoms are: difficulty of deglutition and respiration, especially in the dorsal position, a visible and, particularly, palpable tumour at the posterior pharyngeal wall, which is resistant at first and later becomes soft and painful to the touch, simultaneous enlargement of lymph glands of the throat. The diagnosis, accordingly, can be made easily and with certainty, especially if the ætiology of retropharyngeal abscess is also taken into consideration, i. e., the fact that they occur in the course of severe infectious diseases (pyæmia, scarlatina, etc.) or of caries of the cervical vertebræ. It is true, retropharyngeal abscess also occurs spontaneously, especially in children.

Chronic Phlegmonous Angina.—This affection presents itself in the form of permanent inflammatory hyperplasia of some portions of the palate, thus of the uvula and, above all, of the tonsils, which may become hypertrophied by new formation of reticular tissue in the train of a phlegmonous angina in which no suppuration sets in. Similar conditions prevail in phlegmonous pharyngitis which becomes chronic, inasmuch as here, also, hypertrophies or even atrophic processes may take place as consequence of a deep-reaching inflammation in the pharynx. The local disturbances

in this form of angina and pharyngitis are the same as in acute catarrh, only less violent; nor is fever present in chronic phlegmonous angina.

DIPHTHERIA OF THE PALATE AND PHARYNX

While local changes predominate in the inflammations of the palate and pharynx so far discussed, the general infection and the demonstration of the specific bacillus determine the diagnosis in the diphtheritic inflammation. Local changes in the palate and pharynx, which may present very varying forms are to be noted. As specifically diphtheritic only that form may be designated which is accompanied with the deposition of peculiar white membranes, whereas the infectious diphtheritic angina which does not lead to the formation of membranes, on the one hand, and gangrenous palatine and pharyngeal diphtheria, on the other hand, do not differ in any way, so far as the local manifestations are concerned, from a common catarrhal angina or from gangrene of the palate or pharynx brought about in some other manner.

Infectious (Diphtheritic) Simple Angina without Membrane.—The possibility that an apparently innocent catarrhal angina may be of diphtheritic origin is shown by the fact that, in families in which diphtheria prevails, sometimes one child is afflicted with the common form of diphtheritic disease of the pharynx, another child at the same time shows gangrenous, a third one again a simple angina, and, further, that this latter may cause an infection which, in the infected individual, takes the course of a severe pharyngeal diphtheria. But the proof that an apparently simple catarrhal angina may be of a diphtheritic character, has recently been rendered absolutely certain by the fact that in such cases the presence of genuine virulent diphtheria bacilli has been determined on the pharyngeal mucous membrane. If, therefore, the general symptoms in simple angina are disproportionately severe—i. e., if decided fatigue and weakness as well as intense fever are present—if, besides, the affection is associated with vomiting and swelling of lymph glands of the neck, if the patient recovers but slowly, and if nephritis or even paralysis appears in connection with the angina, the probability may be considered that the latter is of the *infectious* diphtheritic variety, especially if it can be proved that there was a possibility of contagion. But the diagnosis always remains provisional in such cases if they occur isolatedly, unless the microscopical examination for diphtheria bacilli gives a positive result.

Finding of Bacteria in Diphtheria.—*For the bacillus which was first described by Klebs in 1883, and first obtained in pure culture by Löffler in 1884, can to-day be considered with absolute certainty as the sole generator of genuine infectious diphtheria.*

In referring, regarding the morphological and biological properties of the diphtheria bacillus, to the details given later in the description of the infectious disease diphtheria, I will state here only that, besides the specific diphtheria bacillus, various other pathogenic bacteria are found in the diphtheritic membranes: streptococci, staphylococci, a short-rod bacilius analogous to Fränkel's pneumo-bacillus, and to the bacillus coli communis. Streptococci are the most important in so far as they, in contrast to the diphtheria bacilli, enter deeply into the tissue, into the lymph and blood channels, and cause a septicæmic infection. The membranes proper, produced by the effect of the diphtheria bacilli upon the epithelia and a supervening fibrinous exudation, consist of more or less thick layers of fibrin in which degenerated epithelia, single round cells as well as bacteria (towards the mucous membrane the specific diphtheria bacilli) can be demonstrated.

Character of the Diphtheritic Membrane.—If the diagnosis of diphtheria is to be certain, the microscopical demonstration of the presence of the Klebs-Löffler's bacillus must be made. But the presence of diphtheria can, in many cases, be surmised with at least a great deal of probability from the changes in the palate and pharynx in the form of the well-known *whitish-gray depositions* (pseudo-membranes). They are most frequent on the tonsils, often commencing directly at the openings of the follicles; in other cases the starting-point of the diphtheritic exudation is in the uvula or in the posterior pharyngeal wall.

The pseudo-membranes are either of a *dull white* or of a *lard-like gray* or *yellowish* colour, which usually cannot be wiped off; if they can be wiped off, sometimes slight losses of substance remain which then bleed and are rapidly covered again with new membranes. In cases in which the local changes are more prominently developed, the various *plaques* soon become confluent, and at the same time enter beneath the surface. The tissue then becomes necrotic and pulpy, and smaller or larger pieces of the tonsils, of the arches of the palate or of the uvula, become detached by gangrene (*gangrenous diphtheria*).

Painful, often considerably enlarged lymph glands are found, besides the changes in the pharynx, externally at the throat, especially at the angle of the lower jaw.

Differential Diagnosis.—It is generally easy to recognise the diphtheritic character of the plaques, unless it is a question of very insignificant membranous formation; but the diagnosis is often difficult at the onset of the affection. A *confounding* of this condition with the secretory plugs of *lacunar angina* is especially apt to occur. But the latter are situated exclusively in the openings of the follicles of the tonsil, whereas the diphtheritic membranes, although at first also are sometimes located at these places, are not precisely limited to the same in the further course of the affection. It is true exceptions to this rule occur in both directions; membrane-like spreading in non-diphtheritic lacunar angina and, on the other hand, genuine diphtheria resembling the clinical picture of lacunar angina without diffusion of the membrane (see p. 238).

Besides, anginas are sometimes observed which, in their external appearance, are in no way different from diphtheritic anginas, *but, in the membranes of which, diphtheria bacilli cannot be demonstrated either in the microscopical examination or in plate cultures* ("*diphtheroid*"). Here either no micro-organisms are found at all, or, as a rule—streptococci, staphylococci and diplococci, or non-virulent diphtheria bacilli (pseudo-diphtheria bacilli, see Differential Diagnosis of Diphtheria).

Corrosive crusts, due to silver nitrate or acids, sometimes also resemble diphtheritic membranes, but they are easily recognised as artificial productions if the delicately white colour of the corrosive crusts and their superficiality are sufficiently observed.

A positive decision whether diphtheria or only a diphtheria-like affection prevails, is only brought about by the microscopical examination of a particle of membrane for the specific bacilli, which should always be made in doubtful and in unquestionable cases.

Scarlatinal Diphtheria of the pharynx (*scarlatinal diphtheroid*) is to be differentiated from common diphtheria as an affection of the pharyngeal formations which varies in character from genuine diphtheria. Pathological-anatomical and clinical facts are in favour of the correctness of this assumption. The "diphtheritic" pharyngeal changes in scarlatina *usually* represent, in contrast to the usual condition in infectious diphtheria, actual crusts of the mucous membrane, and in these only cocci, especially streptococci, were found, and not the Klebs-Löffler's bacillus. In some cases of scarlatina with pharyngeal diphtheria, however, genuine diphtheria bacilli are found; but then it is a question of a mixed infection of scarlatina and diphtheria. It should be emphasized, clinically, as a differential diagnostic characteristic, that genuine diphtheria spreads to the air passages, scarlatinal to the middle ear, rarely affecting the larynx to a marked degree (necessitating tracheotomy); furthermore, paralyses, which are characteristic of genuine diphtheria as subsequent affections, are almost never observed after so-called scarlatinal diphtheria.

TUBERCULOSIS OF THE PALATE AND PHARYNX

Sometimes, although rarely, we may find in tuberculous patients, at the arches of the palate, at the tonsils and in the pharynx, cheesy infiltrations which principally affect the adenoid tissue, and which lead to the formation of ulcers of varying depth which show no tendency to cicatrization; these ulcers have sharp, eroded margins with flabby granulations and a smeary base. In these ulcers or in their surroundings, sometimes tuberculous nodes are found and, upon microscopical examination, *tubercle bacilli* are detected. This is the final criterion, to differentiate tuberculous from syphilitic ulcers for which they may sometimes be mistaken.

SYPHILIS OF THE PALATE AND PHARYNX

Syphilis in its various forms preferably localizes itself at the palate and in the pharynx.

Syphilitic Angina.—*Syphilitic angina and pharyngitis* do not differ in any manner from the non-specific variety. At most, the obstinacy of the catarrh and the insignificant disturbances connected with it arouse suspicion that it is a question of a syphilitic affection. Besides, there may be whitish discolorations, caused by thickening of the epithelium, in some places of the inflamed mucous membrane.

Condylomata and Simple Syphilitic Ulcers.—On the other hand, there can no longer be any doubt as to the syphilitic character of the palatine-pharyngeal affection, if pea-sized, rounded, pale prominences of the mucous membrane, so-called *plaques muqueuses*, caused by cellular infiltration, are found. These prominences, the analogon of the broad condylomata of the external skin, may ulcerate and form sharp-edged, eventually deep ulcers, sometimes even ulcers which possess diphtheria-like membranes, especially on the tonsils and on the uvula. They are early symptoms of syphilis, but sometimes form the transition stage to gummata of the palate and pharynx, the characteristic manifestations of the later phases of syphilis.

Gummata of the Palate and Pharynx.—*Syphilitic gummatous nodes* represent extensive infiltrations which start from the mucosa or submucosa, sometimes also from the periosteum of the posterior pharyngeal wall and of the hard palate. They show a great inclination to disintegrate and form deep ulcers and perforation at the palate, but, above all, at the velum near the uvula so that the latter may be partly or totally destroyed thereby. The appearance of such ulcers is not absolutely characteristic; but, usually,

their comparatively very rapid formation from an infiltration, the synchronous presence of other manifestations of syphilis, the swelling of the cervical, and especially of cubital glands, leave no doubt as to the nature of the ulcerations. If cicatrization occurs, there arise, according to the depth of the ulcerating surface, flat or radiating cicatrices with coalescence and distortion of the various parts of the palatopharyngeal space. These cicatrices, as well as the persisting perforations and defects, are usually characteristics of recovery from syphilis, which last for life.

It is not easily possible to confound the formation of gummatous nodes with *carcinoma* of the palate and pharynx, if attention is paid to the steady growth and spread of the new formation besides the ulceration, and if, eventually, the carcinomatous character of the neoplasm is determined by microscopical examination of excised portions.

DISEASES OF THE ŒSOPHAGUS

ŒSOPHAGITIS

The inflammatory conditions of the œsophagus are well characterized in a pathological-anatomical respect and occur in the various varieties which are also observed in the other organs, as catarrhal, follicular, pustulovariolous, phlegmonous, croupous inflammations; clinically, however, œsophagitis offers few points of support for the diagnosis. The latter, therefore, is usually very uncertain, unless an examination is made with the œsophagoscope, the application of which in œsophagitis, however, is connected with such disproportionately great inconveniences to the patient that, except in rare cases, it is not well to resort to this diagnostic aid.

Those symptoms which are to a certain extent considered diagnostic are, primarily, the pains which are felt along the œsophagus, and which are increased by the descent of food, but especially also by an eventual examination with the stomach-tube (the rubber tube should always be used, but even this only in urgent cases). Reflex muscular spasm causes during deglutition of food a lodging, sometimes a forcing upward of the food. The vomited masses may be enveloped in mucus, etc.

If, according to the above remarks, it is risky to diagnosticate œsophagitis with certainty, then diagnoses which concern the special form of inflammation of the œsophagus, are constructed almost entirely theoretically. If *fibrinous* masses are forced up in pharyngeal diphtheria, this does not prove anything at all regarding their origin in the œsophagus; it is different however if, as has been observed in some rare cases, a tube-like membrane—i. e., a complete fibrinous cast of the œsophagus—temporarily obstructs the lumen of the œsophagus and is vomited. It is obvious that the, theoretically to be expected, expulsion of *purulent masses* in *phlegmonous* inflammation of the œsophagus does not give any positive assurance as to the existence of this rare disease, as the pus may have perforated from adjacent organs into the œsophagus.

ULCERS OF THE ŒSOPHAGUS

They arise under the most varying ætiological conditions (ulcers due to corrosion, mechanical pressure, syphilitic, tuberculous, peptic ulcers) and occur in the greatest variety of forms. Here, too, the diagnosis is supposed to be indicated by difficulties of deglutition, pain, forcible ejection of food which may be impregnated with blood. It may be especially mentioned that *syphilitic ulcers* occur extremely rarely on the mucous membrane of the œsophagus (in contrast to other mucous membranes), and furthermore, that at least *one* ulcer is easily to be diagnosticated, on account of its

high seat, without the application of the œsophagoscope, namely the ***decubital ulcer of the œsophagus***. The latter occurs in debilitated, bedridden patients during the last weeks of life due to the pressure which is exerted by the larynx (respectively the plate of its cricoid cartilage) which sinks posteriorly, owing to the general muscular weakness, upon the adjacent œsophago-pharyngeal wall, so that the two surfaces of the mucous membranes are pressed upon each other and upon the vertebral column. Thus there are formed at the lowest part of the pharynx, at the boundary between the latter and the œsophagus, two small, corresponding necrotic areas (at the anterior and posterior walls of the lowest portion of the pharynx), which ulcerate and which can be seen with the laryngoscope as ulcerations.

Peptic Ulcer of the Œsophagus.—In the lowest portion of the œsophagus there occurs, in rare cases, an ulcer which must be considered as *peptic*, caused by the fact that, after the circulation in the lowest end of the œsophagus has, for some reason or other, become disturbed, the acid or hyperacid gastric juice erodes, upon regurgitation into the œsophagus, the locality which is inclined to digestion. The symptoms must be the same as in ulcer of the cardia; the diagnosis of this œsophageal ulcer, therefore, coincides with that of the latter.

NEOPLASMS OF THE ŒSOPHAGUS

Only *carcinomata of the œsophagus* are of clinical interest among the neoplasms which occur in the œsophagus, if we do not include those rare cases of polypi of the œsophagus, which, commencing in the lowest portion of the pharynx, are accessible to surgical treatment.

Carcinoma of the Œsophagus is a relatively frequent affection, and is found in the œsophagus almost always as a primary carcinomatous growth in the form of an epithelial cancer. The seat of the tumour is, in by far the majority of cases, the middle, and particularly the lower, third of the œsophagus. At the onset only affecting the smallest portion of its wall, the cancer gradually becomes *girdle-like* and thus causes a stricture which narrows the lumen of the œsophagus more and more, and only temporarily becomes slightly permeable again by ulceration of the carcinomatous locality. If the ulceration spreads beyond the wall of the œsophagus, perforations occur into the air passages, into the pleural and pericardial cavities and into the large blood-vessels. In other cases the carcinoma proliferates into the posterior mediastinum or into the vertebral column, thus causing symptoms of pressure. Angina pectoris with severe attacks of dyspnœa (pressure upon the vagus), as I have seen in an extraordinary example, furthermore, paralysis of the vocal cord (by pressure upon a recurrent nerve, especially the left), paralysis of the lower extremities, etc. An œsophageal carcinoma may also, although rarely, proliferate towards the stomach; in a case which I observed recently it happened that the undoubted secondary development of the gastric carcinoma was much more extensive than that of the primary carcinoma of the œsophagus.

Disturbance of Deglutition.—The diagnostically important symptoms are obvious from the above remarks. The swallowed foods finding an obstacle in the œsophagus, they are regurgitated, sooner or later, according to the higher or lower seat of the cancer—if putrefaction has set in, covered with blood and fœtid material. The same as in all carcinomata, a pernicious effect upon the constitution does not fail to appear in carcinoma of the œsophagus, in this case more so because the ingestion of food

gradually becomes more and more deficient. At the onset of the affection, so long as the patients are still able to ingest soft food without difficulty, the apparently absolutely good nutrition contrasts very markedly with the objective finding which carcinoma of the œsophagus shows even in this stage. Later the cachexia attains the highest development; eventually, metastases occur in the liver and in other organs, especially in the lymph glands. But, as usually those do not swell which are situated in the axilla, in the supraclavicular and infraclavicular regions, but those situated at the hilum of the lung, this metastatic infiltration of the glands is, as a rule, of no avail diagnostically.

Stenosis of the Œsophagus.—The principal point in the diagnosis is always the determination of a *stenosis of the œsophagus*, and, furthermore, its differentiation from other forms of œsophageal narrowing, so that it appears more proper to discuss further particulars of the diagnosis in the diagnosis of stenosis of the œsophagus to which we shall presently pass.

STENOSIS OF THE ŒSOPHAGUS

Stenosis of the œsophagus is the most frequent and practically most important affection of the œsophagus. Its diagnosis does not present any difficulties as regards the determination of a narrowing of the lumen of the œsophagus, but often almost insurmountable obstacles in regard to the discovery of the cause and character of the stenosis in the given cases.

Difficulties of Deglutition—Regurgitation of Food Particles.—The first symptom of a narrowing of the œsophagus is the disagreeable sensation that larger morsels of solid food do not pass down quite unobstructedly. After a short while the patient observes that they become lodged and that they regurgitate, the sooner, the higher the location of the stricture; this occurs a little later, if the narrowing is situated more towards the cardia. A longer retention of food in the œsophagus is also favoured especially by the fact that that position of the œsophagus which is situated immediately above the narrowed area, becomes dilated. The appearance of the ejected masses, unless a distinctly coloured food has been partaken of, is grayish white; they contain particles of food of the last or of a previous meal, with abundant masses of mucus and fungi, rarely blood. The amylaceous substances are partly transformed into sugar, the albuminous substances, if they remain more than one day, are putrid; the regurgitated mass has an insipid, sometimes also a penetrating fœtid odour.

Masses which are brought up from the œsophagus do not contain, in contrast to masses that are vomited from the stomach, pepsin, free hydrochloric acid, peptone and constituents of bile. The reaction of the regurgitated mass is almost always neutral.

Auscultatory Phenomena.—Auscultation of the œsophagus should be performed at the throat to the left of the trachea, farther down on the left side of the vertebral column to about the tenth thoracic vertebra. Upon swallowing of fluids by the person to be examined, we hear at this place, normally, a kind of ripple, caused by the passing down of the fluid swallowed, which, in less than half a second, is "squirted" as far as the cardia. After having remained for a few seconds above the latter, it passes, mixed with air, about six to seven seconds after the beginning of the act of

deglutition, to the entrance of the stomach with a gurgling sound ("pressing-through sound"), which is heard, upon auscultation of the gastric region, to the left of the xiphoid process.

If a stenosis of the œsophagus is present, we can observe that the œsophageal deglutition sound becomes softer or is suddenly interrupted, and may entirely miss the "pressing-through sound" or observe it later. However, these auscultatory œsophageal phenomena are by no means certain signs of stenosis of the œsophagus. Much more reliable results are obtained with the direct *examination of the stenosed area with the stomach-tube.*

Sounding of the Œsophagus.—For this first sounding I employ, under all circumstances, a rubber tube; the latter is, supplied with the mandrin, passed over the plate of the cricoid cartilage, then the mandrin is removed and the tube is passed down until it stops at the obstruction. Now the place is marked on the tube at which it is touched by the incisors. If, from the above mark, the tube is externally placed upon the cheek and the lateral portion of the throat, we may be able approximately to determine the location of the obstacle. This *harmless* mode of examination is usually fully sufficient. It is also satisfactory to me, above all, when I have reason to surmise a carcinoma of the œsophagus. I have been taught by years of experience that the generally practised use of thin *stiff* sounds causes more harm than good in most cases of carcinoma of the œsophagus. Only, if no traces of blood can be found on the sound after withdrawal, in repeated sounding with a thin rubber tube, I employ in carcinoma also stiff bougies, the use of which in other cases (e. g., in cicatricial strictures or in nervous-spasm strictures of the œsophagus) is, of course, imperative therapeutically.

Disturbances of Nutrition.—Deficient ingestion of food is bound, in the course of time, to injure the nutrition of the patient. But it should be stated that, unless it is a question of carcinomatous stenosis of the œsophagus and fluids still pass the narrow area, the emaciation is relatively very insignificant, as even a person who until then was well nourished, may be kept at his former weight by the sole administration of sufficient quantities of milk. I knew a patient who lived exclusively on milk for three years and who not only lost nothing of his weight of about 165 pounds, but actually gained 9 to 11 pounds. But if, on the other hand, the passage is so narrow that no fluids can pass, or only in insufficient quantities reach the stomach, the patient emaciates rapidly and the gastric region retracts in a trough-shaped manner.

Nature of Stenosis of the Œsophagus.—The diagnosis of œsophageal stenosis can be made without difficulty from the above-described signs. As easy as it is, accordingly, to determine a stenosis of the œsophagus, so difficult is it sometimes to ascertain *the character of the stenosis.* But this is the most important demand made upon a diagnostician, because not until a decision is arrrived at as to the cause of the stenosis of the œsophagus, will prognosis and therapy take a distinct direction. To attain this object, the following method had better be observed:

Extra-Œsophageal Causes.—At first it should be determined whether *causes situated outside the œsophagus* exist which may give rise to the occurrence of a stenosis. The first object, therefore, is the external examination of the throat, to decide the question whether a swollen lymph gland of the throat, an infiltration of the cellular tissue of the throat, etc.; or,

the most frequent finding, a *goître* presses upon the œsophagus, to which fact simultaneous dyspnœa points by compression of the trachea. In connection with the above, percussion of the region of the manubrium sterni should also be done to find whether a substernal goître may be the cause of the compression. If dulness is found here, we must consider further whether the presence of an *aneurysm* is likely, we must auscultate for eventual murmurs, compare the radial pulses, etc. I almost habitually examine in such a manner in all cases of œsophageal stenosis, before introducing the sound for the first time. Of course, the consideration whether a tumour narrowing the lumen of the œsophagus at the throat or an aneurysm, etc., is the cause of the stenosis, depends primarily upon the height at which the stenosis is located. If it may be assumed that the latter is situated in the intrathoracic portion of the œsophagus, there are further to be considered, besides aneurysm, *mediastinal tumours* or a large *pericardial exudate*, diseases the diagnosis of which has been explicitly discussed. The last-named intrathoracic affections, upon the whole, rarely give rise to dysphagia. Furthermore, we must examine the vertebral column for deviations and painfulness of the vertebræ, as an excessive degree of lordosis, a hypostatic abscess originating in a carious vertebral process, etc., may cause stenosis of the œsophagus. After thorax, vertebral column, and throat have been examined in such a manner—in rare cases, if the location of the obstacle is very high, a laryngoscopical examination is also to be made—and if nothing supports the assumption of a certain cause of stenosis of the œsophagus, then a last extra-œsophageal factor is to be thought of, which acts as a compressor, viz.:

A Diverticulum of the Œsophagus.—We shall fully discuss the diagnosis of this condition in dilatations of the œsophagus; here mention may be made only of the fact that in diverticulum, according to the amount of food which is contained in it, large quantities of food may eventually be forcibly ejected and the œsophageal tube may at times be compressed laterally and impermeably, at other times, after evacuation of the diverticulum, it may be passed so that the sound, which previously encountered an insurmountable obstacle, now passes the place without hindrance. This change in the possibility of entering the œsophagus with a bougie, which is of the greatest importance for the diagnosis of a diverticulum, is, however, not pathognomonic of the formation of a diverticulum alone; similar conditions are also found with foreign bodies in the œsophagus which change their position—in fact, in the most varying forms of stenosis, if a larger morsel of food temporarily lodges in the opening of the stenosis, but, above all, in

Spastic Stricture of the Œsophagus.—In *stenosis of the œsophagus caused by spasm of the œsophageal musculature* it is always possible, according to my experience, with patience to overcome the obstacle, in employing a not too thin, hard sound. It is true, in some cases a certain amount of force is required to push the sound down and to bring it up again. The sound in such a case is retained, and then, as can be distinctly felt, suddenly released, eventually to encounter no obstacle at any part of the œsophagus at the following sounding. Sometimes, according to my experience,

a spastic stenosis of its lumen is found in the entire course of the œsophagus. Then we feel, especially upon withdrawal of the elastic tube, how the latter is firmly grasped spasmodically for a long distance, and even may be rather drawn out—i. e., stretched—than brought up. Usually other signs of hysteria exist besides spastic stricture; not always, it is true, as is proved by the fact that I have sometimes found spastic strictures of the œsophagus in robust men. Sometimes it occurs that a spastic narrowing of the œsophageal lumen supervenes secondarily upon organic changes of the œsophageal wall, upon ulcerations, tumours, or upon the lodgment of foreign bodies. However, the latter condition is *very rare*, and the change of the permeability of the œsophagus in these cases may be explained in a different manner.

Intra-Œsophageal Causes of Stenosis of the Œsophagus—Foreign Bodies.—This naturally leads us to the diagnosis of *intra-œsophageal* causes of stenosis of the œsophagus.

A *wedged-in foreign body* is to be thought of as cause of the occlusion of the œsophagus, if the stenosis occurred *acutely* and causes violent pain. Usually the patient himself states that this or the other object has been carelessly swallowed by him. But it should not be forgotten that, occasionally, upon the swallowing of hard food, the patient becomes aware, for the first time, of an organic change of the œsophagus which has been present for some length of time, and which is erroneously interpreted as the cause of the œsophageal stenosis. The rare pediculated *polypi of the œsophagus* may also be counted in the category of foreign bodies. They usually originate from the lower part of the pharynx and are suspended from here into the œsophagus. It is further possible that the presence of very considerable masses of the *oidium albicans* in the œsophagus may cause its occlusion. A *careful* sounding is indicated in such cases; the obstacle is easily overcome in the last-mentioned case, not so, however, in the presence of swallowed foreign bodies in the œsophagus; then the application of Duplay's sound may give direct information whether or not a foreign body is present in the œsophagus.

A *cicatricial stricture* will gradually form, if the foreign body has entered the œsophagus some length of time previous and if its presence has caused a defect, or if a corrosion of the œsophageal wall has taken place by acids, alkalies or other caustic substances. A cicatricial stricture may develop from any kind of ulceration, syphilitic and diphtheritic included—I once saw a cicatricial stenosis of the œsophagus follow a pharyngeal diphtheria—but both are rare causes of cicatricial stricture. The presence of the latter is pointed to by, above all, the history of the case, by the slow development of the dysphagia, the marked, uniform consistence of the obstacle upon introduction of the tube, the absence of blood on the tube upon withdrawal and the possibility of dilating the stenosed area.

I can only advise, always to go through this entire course of diagnostic calculation, from beginning to end, and only after all the above-mentioned causes of stenosis, at any rate the most important of them, have been gone over and have been excluded, to concentrate the diagnostic considerations upon the most frequent cause of œsophageal stenosis, viz., *carcinoma*.

Carcinoma the Cause of Œsophageal Stenosis.—If we begin in the inverse direction, i. e., with the consideration whether a carcinoma may be present and whether there is anything opposed to the presence of such a condition, we are very apt to be satisfied with a certain probability of diagnosis of cancer of the œsophagus. It is true that, owing to the very frequent occurrence of carcinomatous stenosis of the œsophagus, a correct diagnosis will be made even in this manner; but in some of the cases the diagnosis will prove to be wrong, causing injury to the patient and mortification to the physician. If a gradually developing stenosis of the œsophagus is found in a patient above forty years of age, and if, although he is still able to pass liquid food without difficulty, an increasing emaciation and cachexia become manifest, it is almost certain from the beginning that he suffers from carcinoma of the œsophagus. If then, upon subsequent examination with the sound, an obstacle can be determined, and if the sound upon withdrawal is covered with masses of mucus and *blood* (in putrefying carcinoma ichorically fœtid masses), although a soft sound has been employed and no pressure has been exercised with the hard tube, the suspicion of carcinoma becomes confirmed. Sometimes the sound, unless too thick a one has been selected, advances to the locality of the stenosis in a narrow channel and is retained in the latter; upon withdrawal it is then felt how the sound is firmly grasped by the annular, long-stretched neoplasm. In rare cases we find in the tube-window pieces of tissue, the microscopical examination of which removes any doubt as to the diagnosis of cancer. The diagnosis is further confirmed by the occurrence of carcinomatous metastases in lungs and liver, also by paralyses of the recurrent nerves and by other symptoms of compression caused by the growing tumour, which, although occurring in other forms of œsophageal stenosis, also, are of particular frequency in carcinoma of the œsophagus.

DILATATIONS OF THE ŒSOPHAGUS

Of dilatations of the œsophagus there are distinguished the *diffuse*, or *total*, and the *circumscribed* dilatations, which are limited to a small area in the œsophagus.

Diffuse Dilatations of the Œsophagus.—As to the former, *primary, total dilatations*, they (only a very small number of cases have as yet been observed) manifested themselves by severe dysphagia, inasmuch as the food ingested did not pass down but was forced up again after ingestion. A *positive* diagnosis of these rare conditions is scarcely possible; but complete dilatations of the œsophagus may at least be surmised, if, upon the presence of dysphagia, the demonstration of the absence of a stenosis can be furnished by means of a sound, which instrument, in contrast to the condition in diverticulum, always slides down without obstruction, in fact, abnormally easily, upon repeated examination.

Partial Dilatation of the Œsophagus.—The diagnosis can be made with much more precision in *partial dilatation* of the œsophagus, either because the circumscribed dilatation refers to the entire circumference of the œsophagus above a stenosis, or because it only affects one part of the wall and causes a sacculation of the organ, a *diverticulum*. *To a partial dilata-*

tion affecting the entire circumference of the œsophagus there points the fact that more or less copious masses of food ingested and retained above the stenotic area, are forcibly ejected at once, furthermore that, after evacuation of the same, large masses of fluid can be introduced and withdrawn from the area of the stenosis, which is demonstrated at the same location by sounding, without the tube ever reaching the stomach. However, in rare cases it may be possible that the sound may, in spite of a uniform dilatation of the œsophagus over the cardia, reach the stomach without obstruction, namely, when the cardia at times only contracts spastically, or if its reflex relaxation, respectively opening, is only impeded upon deglutition. The food will, in both cases, remain in front of the cardia and gradually dilatation of the œsophagus will occur.

Œsophageal Diverticulum.—Diagnostic reasoning takes another direction if it is a question of *lateral* sacculations, so-called *diverticula. Of both kinds of diverticula, traction and pulsion diverticula, only the latter variety is accessible to diagnosis.*

Traction diverticula, arising in consequence of peri-œsophageal processes of wasting, especially in the region of the bifurcation of the trachea, are small prominences of the œsophageal wall, half a centimetre to, at most, one centimetre in size, which are not capable of being diagnosticated because the sound, as well as the food, passes them without encountering an obstacle. The presence of a traction diverticulum may be, at most, surmised, if at certain places, far down in the œsophagus, large, hard morsels become lodged by becoming attached to the diverticulum, while no stenosis can be demonstrated with the sound and a spasm of the œsophagus may be excluded in the given case, or if, in connection with those vague disturbances of deglutition, a perforation of the œsophagus occurs due to another, inexplicable cause.

Pulsions Diverticulum.—The diagnosis of a so-called *pulsation diverticulum*, studied particularly carefully by Zenker, offers less difficulties. These diverticula are situated almost exclusively *at the uppermost boundary of the œsophagus*, therefore *behind the plate of the cricoid cartilage,* and originate in the posterior wall of the œsophagus.

Their origin is probably due to the fact that the wall becomes more yielding at some place at the boundary between œsophagus and pharynx, in such a manner, for instance, that a foreign body pushes aside a few muscular fibres, and that now the mucous membrane is forced towards this yielding space by the morsels of food, which always experience a slight stoppage behind the plate of the cricoid cartilage, sacculating the mucous membrane and gradually forming a continually growing pouch. The latter is situated between vertebral column and œsophagus, and continually forces the latter forward; the axis of the œsophagus thus becomes kinked, whereas the axis of the diverticulum becomes gradually situated in the direction of the pharyngeal axis. Thus it occurs that the food, upon deglutition, is more and more caught by the diverticulum, and that but very little or nothing reaches the œsophagus and stomach; and the same will occur with the œsophageal sound if introduced for diagnostic purposes in such cases. It always enters the diverticulum, *is bound* to, according to the anatomical conditions, to enter the same. Only when the pouch is completely evacuated, which, however, is very rarely the case in larger diverticula, will the sound pass the entrance of the diverticulum and enter the œsophagus, and now even a thick sound may be advanced without obstruction down into the stomach.

The changing permeability of the œsophagus for the tube is an extremely important factor for the diagnosis of the diverticulum; however, this diagnosis cannot be made so simply as is usually assumed theoretically. For this change in the possibility to sound the œsophagus is found, as has been stated, in other conditions also; besides, the total evacuation of the sac, which is the indispensable condition for the entrance of the tube into the œsophagus, is a rare occurrence.

Employment of the Diverticulum Sound for the Diagnosis of a "Pulsions Divertikel."—Zenker and I, therefore, have constructed a sound which, in contrast to that tube which usually becomes lodged in the diverticulum, allows of the entrance of the instrument into the œsophagus (diverticulum sound) through a bending of the point, which can be brought about at any desired place, in an angle forward around the edge. As we know the place at which the entrance of the diverticulum can be found, viz., behind the plate of the cricoid cartilage, the possibility is given to do the bending of the point of the tube at the proper moment when the sound is introduced. Whereas, therefore, the diverticular sound, not bent, enters the diverticulum and, according to the length of the latter, advances more or less far down to become lodged at its blind end, it becomes possible to force the same sound upon withdrawing it, if the point is bent forward behind the plate of the cricoid cartilage, into the œsophagus and then to advance it, without obstacle, into the stomach. I have succeeded in such a manner in two cases to make the diagnosis of a "Pulsions Divertikel."

Although the sac is rarely entirely emptied, yet, upon partial evacuation of the same, and with temporarily suspending compression of the œsophagus in such a manner, food may occasionally pass without obstruction; but the greatest part of the food ingested goes into the diverticulum. As the latter, originating at the uppermost boundary of the œsophagus, sinks between the vertebral column and the œsophagus, upon more marked development of the sac, *a distinctly visible and palpable tumour becomes prominent externally at the throat, near the trachea*; this tumour decreases in size after evacuation of its contents by regurgitation or by pressure and stroking from outside, increases again upon ingestion of food, exerts a pressure upon its surroundings, the trachea and nerves of the throat, and it may eventually give rise to a violent paroxysm of coughing upon change of posture of the patient.

RUPTURE OF CONTINUITY OF THE ŒSOPHAGUS

The diagnosis of ruptures and perforation of the œsophagus is always difficult.

Spontaneous Rupture of the Œsophagus.—Commencing with *spontaneous rupture*, which, without traumatism, affects the lowest portion of the apparently, until then, entirely healthy wall of the œsophagus, the pathological picture has been a suddenly occurring, extremely severe one in the few cases which have been observed up to this time. The patients complained of violent pain at the location of the rupture; they had the distinct sensation that some laceration had taken place in their interior, after inclination to vomit or retching had preceded, in which cases blood was sometimes thrown up. Soon collapse of a high grade, anxiety and dyspnœa predominated in the pathological picture. The diagnostically most important symptom was a *cutaneous emphysema*, which originated in the supraclavicular region and rapidly spread over the body; it was evidently due to the entrance of air into the mediastinum, originating from this point. As a tear of the pleura was also found in several cases, probably only explicable by an œsophagomalacia originating during life, it is probable that a pneumothorax accompanying the cutaneous emphysema, also supports the diagnosis. But it is always necessary in this diagnosis that, not a spon-

taneous *perforation*, but one which occurs gradually, owing to an anatomical change situated in the wall of the œsophagus or in its neighbourhood, can be excluded.

Perforations of the Œsophagus.—These *perforations* occur much oftener than spontaneous ruptures. Their causes are: diverticula ulcerating in consequence of wedged-in foreign bodies (carcinomatous and peptic) ulcers, erosions of the œsophageal wall, and sharp-edged foreign bodies. A perforation from without to within is brought about by suppurating bronchial glands, suppurative mediastinitis, gangrene of the lungs, aortic aneurysms, etc., causes of perforation of the œsophagus which must be known to the diagnostician. It is obvious from the above that perforations are much more frequent in the lower than in the upper portion of the œsophagus. The perforation developing gradually and chronically inflammatory coalescences having formed, mostly in the neighbourhood of the œsophagus, some time previous to the perforation, the symptoms are different from those discussed in spontaneous rupture. The cutaneous emphysema, which is of such importance in the latter affection, is, therefore, usually absent; on the other hand, owing to diffusion of the ulceration, *mediastinal abscesses* occur, and, in connection with them pleurisy, pericarditis, pneumonia with abscess formation, pneumothorax and pneumopericardium of a putrefactive or non-putrefactive character.

As to the latter, I have seen that air may be present in the pericardial sac without a complete perforation of the external pericardium being the necessary consequence, if only the latter has become gangrenous and thus, apparently, permeable for air.

If it is possible, in consideration of the ætiology and of the secondary intrathoracic, usually ichoric, inflammatory process, to make the diagnosis with a great deal of probability, it becomes still more certain if, for instance, ingested food, eventually recognisable by its colour, is coughed up, or if a stenosis of the œsophagus which has existed so far, apparently becomes passable, inasmuch as the food finds another outlet. We have already, in a former chapter (see p. 99), treated of the consequences of a communication between œsophagus and bronchus which is brought about by perforation. It may be remarked that in such cases it may be heard on auscultation how the fluid that is partaken of, enters the lungs, and that this fluid is expectorated owing to the cough which is produced by the act of deglutition; this fluid, if it has been previously coloured, can easily be proved to be that which has entered the trachea, respectively bronchus, through the œsophagus. If the abscess which forms in the neighbourhood of the perforated œsophagus, breaks through the skin, particles of food will appear in the external opening of the fistula.

Hæmorrhages of the Œsophagus.—Of those diseases of the œsophagus which have not yet been discussed, *hæmorrhage of the œsophagus* is of no great diagnostic interest, as the diagnosis can properly be made only by exclusion—i. e., the source of the hæmorrhage should be looked for in the œsophagus only after a gastric or intestinal hæmorrhage can be positively excluded as the cause of hæmatemesis and eventually of the black stools, and if, on the other hand, certain reasons (the presence of a carcinoma, of an aneurysm, etc.) point to the likelihood of a hæmorrhage from the œsophagus. A positive diagnosis is almost never possible. But we shall still mention

neuroses of the œsophagus which are more readily diagnosticated and which are of some importance.

NEUROSES OF THE ŒSOPHAGUS

Spasm of the œsophagus, *œsophagismus*, manifests itself by *dysphagia*, especially by *pain, a sensation of constriction* (in the throat or low down in the chest), and *regurgitation* of the ingested food. At the same time the peculiarity often prevails that solid morsels can be swallowed better than liquids, a symptom which is decidedly in favour of the fact that no organic, but a nervous stricture is present. This is also proved by the introduction of the sound which actually encounters, sometimes at a higher, at other times at a lower, portion of the œsophagus, an obstacle, which, however, can soon be recognised as one that is not permanent, as it can always be overcome with a little patience. It is possible, in most cases, from the intermittent character of the dysphagia which may be caused by the process of deglutition as such, but also by mental emotions, etc., to decide upon the nervous character of the affection, especially if other symptoms of nervousness and hysteria are present besides spasm of the œsophagus. In other cases the spasm of the œsophagus may persist for months, as I have seen several times, and the picture of an organic stenosis is, according to my experience, also simulated especially by the fact that a complete condition of inanition may occur with it. The examination with the stomach-tube is sometimes neglected in such cases by the physician, because he encountered an apparently insurmountable obstacle at the first attempt and caused a laryngospasm, etc., thus rendering a further attempt at sounding appear dangerous. And yet, a single introduction of the sound, done without consideration, will at once cure the affection after it has lasted for years, as a case in my practice has taught me. An idiopathic(?) spasm may only be assumed when organic changes of the œsophageal wall (which, in their part, may reflexly cause spasm of the œsophagus), such as ulcerations, etc., may *with certainty* be excluded.

Paralytic Dysphagia.—*Paralysis of the œsophagus*, finally, *dysphagia paralytica*, is very difficult to diagnosticate; lodging of food in the œsophagus, in spite of absence of any obstacle upon sounding, and regurgitation of the morsel characterize this rare affection. Here, also, larger morsels are often swallowed better than small ones; the deglutition of liquids is sometimes accompanied with loud rumbling. Of course, nervous paralysis of the œsophagus cannot be differentiated from a diffuse dilatation of the œsophagus which is caused by a (partly anatomically justified) lessened muscular contractility, at most by the fact that the former sometimes develops suddenly, whereas diffuse dilatation of the œsophagus always develops slowly.

DISEASES OF THE STOMACH

ANATOMICO-PHYSIOLOGICAL PRELIMINARY OBSERVATIONS

The most important portion, physiologically, of the wall of the stomach is the *mucous membrane*. It is composed of an epithelial layer of the tunica propria, the muscularis mucosæ, and of the submucosa.

Structure of the Wall of the Stomach.—The *epithelium* of the gastric mucous membrane is a simple cylindrical epithelium; its product is the gastric mucus, the secretion of which, coming from the cells, gives to the latter the appearance of beaker cells. The *tunica propria* consists of elastic and connective-tissue fibres with embedded leucocytes, which sometimes also occur in dense conglomerations and form solitary nodules. The latter occasionally ulcerate in the course of enteric fever, similar to the solitary follicles of the intestine, and such typhoid ulcers of the stomach may then, as I have seen, give rise to fatal hæmorrhages. The main part of the tunica propria are the gastric glands which enter into pit-like depressions of the surface of the mucous membrane ("stomach cells"). The glands are so numerous at

most parts of the stomach that they almost cause the tissue proper of the tunica propria to disappear entirely; only towards the pylorus the glands are less approximate, so that, here, the tunica propria appears more developed and sometimes forms actual villi.

Two forms of glands are distinguished (1) *glands of the fundus and* (2), *glands of the pylorus.* The former, the real gastric or lab glands, possess two kinds of cells: main cells and parietal cells [marginal cells, border cells], of which the former represent simple cylindrical cells which everywhere border upon the lumen of the glands, whereas the parietal cells are in part pushed more towards the periphery, so that they are connected with the lumen of the gland only by small side branches. These side branches carry the secretion of the parietal cells to the lumen of the glands and are themselves excretory ducts of the "capillary system of secretion," which embraces the parietal cells in a basket-like manner, respectively permeates them. The pylorus glands almost exclusively contain main cells, or at least cells which are very similar to them, and but very few parietal cells.

The *muscularis mucosæ* consists of smooth muscular fibres, which in part ascend between the glandular tubules, the *submucosa* of the elastic and the connective-tissue fibres.

Physiological Details.—The gland cells undergo morphological changes during the process of digestion; both varieties of cells become darker, and especially the parietal cells are larger. The product of their activity is the *gastric juice.*

The latter consists of two ferments, the proteolytic *pepsin* (Schwann, 1836) and the *lab-ferment* (Hammarsten, 1872), which causes casein to coagulate. While pepsin and lab-ferment are secreted by the cells of the pylorus glands and by the main cells of the fundus glands as products of secretion, the third constituent of the gastric juice, *hydrochloric acid,* is formed exclusively by the parietal cells, and that by the specific activity of the same from the chlorides of the gastric fluid contents. This has been positively proved by experiment, as, upon long-lasting sodium chloride hunger of the gastric glands, pepsin is always found, but acid is absent (and at that, neither hydrochloric nor lactic acid). If soluble chlorides are administered to a dog, the gastric juice of which has become free from acid by absence of sodium chloride, an abundant secretion of *hydrochloric acid* from the gastric mucous membrane commences at once. This proves that, if *lactic acid* can be demonstrated in the contents of the stomach, it cannot be formed by the activity of the gland cells. Lactic acid is formed by bacterial fermentation from the carbohydrates which are present in the stomach. For, if food is ingested which does not contain generators of lactic acid, it is possible eventually to demonstrate hydrochloric acid in the stomach contents, but never lactic acid. In contrast to this fact, we find only lactic acid in the stomach contents of human beings who have ingested carbohydrates, in the first half hour after the meal; the lactic acid disappears later except traces, whereas more *hydrochloric acid* continually appears. On account of the latter preventing the saccharificating action of the *saliva,* the transformation of amyl into sugar through the ptyalin in the stomach continues only for a comparatively short while—i. e., according to the given case—at most one to four hours.

Hydrochloric acid, therefore, prevents amylolysis, and at the same time serves *to prevent abnormal fermentation and putrefactive processes in the stomach,* because, in the presence of free hydrochloric acid, the acetic-acid fermentation, and also, although to a lesser degree, lactic-acid fermentation are prevented or, at least, reduced. It is true, this is not the case at the onset of digestion, because the secreted quantities of hydrochloric acid are bound either by basic salts or by the albuminous bodies of the food. The formation of lactic acid, therefore, continues until free hydrochloric acid predominates in the contents of the stomach. If, on the other hand, as is usually the case in patients suffering from gastric disease, a reduction of hydrochloric acid exists, the respective bacteria continue to develop, and the processes of fermentation caused by the same take place without obstruction. The carbohydrates and albuminous bodies ingested decompose under these circumstances, and a considerable formation of gases (H_2,CO_2, etc.), especially after the ingestion of carbohydrates, takes place, as will be explained in greater detail in the discussion of the various diseases of the stomach.

Pepsin.—Besides its antifermentative properties, hydrochloric acid plays a very important *digestive* part in its relation to pepsin. Pepsin as well as lab-ferment is contained in the main and pyloric duct cells, not as such, but as the digestively inactive pepsinogen and labzymogen. The latter, however, is rapidly transformed into the active enzymes by acidulation, especially by hydrochloric acid. Pepsin digests most energetically with 0.2 to 0.4 per cent of hydrochloric acid, much less so with other acids—phosphoric, acetic, lactic acids, etc. Pepsin is rapidly destroyed even in weak solutions of soda, so that gastric juice, which has been rendered alkaline outside of the body, remains digestively inactive, even if it is again acidulated fifteen seconds after having been made alkaline. Pepsin digestion is also impeded by salts and by alcohol; pepsinogen, the same as pepsin, is not resistant to the latter, and that still less so than pepsin.

Peptones.—Pepsin with hydrochloric acid develops an especially proteolytic action and then transforms albuminous bodies into *peptones*. The process is as follows: To begin with, the albuminous bodies swell and dissolve and are "denaturalized" to *syntonin*. This denaturalization, which is caused even by hydrochloric acid alone, but still more readily by hydrochloric acid and pepsin, is now followed by the splitting of the albumin molecule by hydration. In this process—i. e., by incorporation of the elements of water—there arise from the albuminous substances, respectively syntonines, at first albumoses and therefrom, by further splitting off, *peptones*, which finishes the action of the gastric juice upon the albuminous substances (whereas by the pancreatic digestion, especially by the action of trypsin, the splitting of the albuminous substances continues, inasmuch as amido acids, leucin, tyrosin, and asparaginic acid, besides small amounts of ammonia, are formed thereby).

Albumoses are much more soluble than albuminous bodies, but slightly less soluble than *peptones*, which also, probably owing to the smallness of their molecules, diffuse very easily, in contradistinction to albumoses. Both combine with hydrochloric acid, forming salt-like combinations; it is necessary, therefore, in order to keep the process of digestion going, always to administer new quantities of hydrochloric acid to the material which is to be digested. Besides, experience teaches that *the hydrolytic processes are delayed and, finally, entirely suspended by the products which accumulate in the further course of digestion.* Such a suspension, however, of the digestive process does not occur in the stomach under normal conditions, because the hydrolytic products are always removed at once.

If gastric digestion is entirely suspended, the albumin-containing food is, nevertheless, still utilized, as now the secretion of the pancreas assumes proteolysis alone. This has been ascertained both by experiment and by clinical experience, inasmuch as recently the entire human stomach has been extirpated without injury to the general nutrition, on the contrary, a gain in weight of several kilos was observed in the patient affected; dogs also, whose stomachs were totally extirpated, remained in nitrogen equilibrium, with sufficient nutrition from the duodenum. It must be assumed, accordingly, that the stomach is not *absolutely* necessary for digestion, although it always aids digestion for the very reason that it assists in proteolysis, and only transmits such quantities of digestive material to the intestine as the latter is conveniently able to assimilate.

Casein Digestion.—Casein of milk occupies a peculiar position among the albuminous bodies which enter the stomach, inasmuch as it is coagulated by *lab-ferment*, and then only is transformed into syntonin and peptone. The meaning of this, at first glance, is apparently superfluous; but the process becomes comprehensible by the fact that casein, if it is resorbed directly, would be excreted from the blood through the kidney as a non-assimilable protein substance—i. e., as a foreign body—and therefore would be lost and not utilized in metabolism.

Inversion of Cane Sugar.—*Cane* sugar is inverted in the stomach, as is positively demonstrated by my own experiments of 1882. It is beyond question that hydrochloric acid of the stomach plays an important *rôle* in the inversion. But it should be remarked that, in control experiments, hydrochloric acid alone acts less rapidly and energetically than gastric juice containing the same amount of acid. It is to be surmised, therefore, that another inverting ferment is active in the gastric juice, but that its definite properties have as yet not been determined.

Fats are not changed at all in the stomach, or but very slightly. However, according to Cash, a splitting of the fats, although scarcely worth mentioning, into glycerin and fatty acids, is said to occur in the stomach.

Effect of the Nervous System.—The secretion of the gastric juice is undoubtedly dependent upon the *nervous system*. This secretion begins with deglutition of food, even when the morsels are regurgitated through an œsophageal fistula—i. e., do not reach the stomach at all—whereas this stimulation of the gastric secretion does not occur if the vagi are severed. Furthermore, it has recently been successful in experiment to increase the secretion of gastric juice by direct stimulation of the peripheral portion of the vagus (Schneyer), so that it is unquestionably demonstrated that the *vagus* carries the secretory fibres to the stomach centrifugally, and is the nerve of *secretion for the stomach*. But secretion of gastric juice takes place even after severing the vagi, if food reaches the stomach. It may be assumed, therefore, that even without the intercession of nerves, a secretion of gastric juice is brought about owing to a direct stimulation of the gland cells by the ingested food. It is also known that gastric juice may be secreted upon the action of psychical impressions.

Motor Function of the Stomach.—The *motility of the stomach* plays quite an important *rôle* in digestion, besides the active chemical factors of the gastric juice. The *rotary movements*, caused principally by the *musculature of the fundus*, have the object of mixing the ingesta intimately with the gastric juice, whereas another muscular movement, the *peristalsis* proper of the stomach, forces the contents of the stomach interruptedly into the duodenum. The pylorus is usually in a state of tonic contraction during digestion, but it relaxes from time to time in order to permit the passage of food which has undergone the necessary change in the stomach. Another function of the musculature of the stomach is, in my opinion, the promotion of *resorption* of the constituents of the gastric contents by the fact that contractions of the musculature cause a more marked movement of lymph vessels and blood vessels. The normal stimulation of the gastric musculature appears to issue from the ganglion cells situated in the wall of the stomach. However, the movements of the stomach are also influenced by the central nervous system, and in the various portions of the same. In the cerebral cortex, in the corpus striatum, in the corpora quadrigemina, in the medulla oblongata, and in the spinal cord, centres have been found which (in the tracts of the vagus and sympatheticus) act upon the muscular movements, either stimulating or retarding them. It has also been ascertained by von Mering's very interesting investigations that the *filling of the small intestine reflexly prevents the emptying of the stomach*. If, namely, in a dog, the lower portion of the duodenum, which is severed and with the ends sewed into the abdominal wall, is filled with milk or a similar absorbable material, water introduced into the stomach will remain in the latter until the duodenum is again empty. There exists, therefore, an arrangement which, from the intestine, regulates the emptying of the stomach and prevents an overfilling of the intestine.

Resorption on the Part of the Stomach.—*Resorption* of dissolved substances takes place to a limited extent in the stomach; at any rate, the surface of the stomach represents a far less satisfactory field for resorption than the intestine. Besides watery salt solutions, peptones, solutions of dextrine and of grape sugar, it *is particularly alcohol which is absorbed to a considerable degree by the stomach*, and the quantity of the substance absorbed generally increases with the concentration of the solutions. The resorption of the substances is accompanied with a secretion of water into the stomach adequate to the quantity of absorbed substance. *But, on the other hand, the stomach does not resorb any appreciable quantities of water*, as has been strikingly demonstrated by von Mering, because all the water introduced into the empty stomach after a short while flows out through a duodenal fistula. As in von Mering's experiments the escaping water often was not acid, this fact proves, incidentally, that movements and an emptying of the stomach take place even without acid reaction of its contents, an observation which is of significance in explaining the fact that, in cases of subacidity or even inacidity of the gastric juice, the emptying of the stomach may still take place in the regular time.

Digestion is *retarded*, as has been demonstrated first by R. Fleischer, by ingestion of alcohol, also by the partaking of food "difficult to digest," e. g., greasy fish, cer-

tain kinds of vegetables (beans), etc., as may be assumed according to *my own* and Penzoldt's experiments regarding the digestibility of certain food materials (considering the time they are retained in the stomach).

DIAGNOSTIC PRELIMINARY REMARKS

In keeping with the plan of this work, I do not intend fully to discuss here the methods which concern inspection, palpation, percussion, and auscultation, but rather occasionally to mention them later on. But, on the other hand, in order to define my standpoint and my experience in regard to certain points, especially as to the chemical diagnosis of diseases of the stomach, it seems necessary to me at least to premise some few diagnostic remarks before discussing the various affections of the stomach.

There cannot be any doubt that at present we are able, owing to the employment of the stomach-tube, and to the chemical examination of the stomach contents for diagnostic purposes, which were first recommended by me, to diagnosticate the various diseases of the stomach with considerably more precision than formerly.

Description of the Stomach-Tube.—In order to examine the stomach, I exclusively employ Nélaton's elastic *stomach-tube* with a closed lower end and two lateral openings near the latter. I do not permit the tube, at least not the first time, to be simply swallowed, because there are some persons who do not succeed in accomplishing this at the first attempt, but I introduce the tube. As a mandrin I employ a thin rattan about half as thick as the lumen of the tube, which, therefore, can be *very easily* withdrawn at any time. Long years of experience have taught me that this sort of tube fully answers all requirements, and that it is also preferable to a simple rubber tube. For, although I willingly admit that the latter fully answers the purpose in many cases—i. e., can be introduced without any difficulty—yet this is not the case in every instance. In a certain number of patients it is not possible, at least not the first time, to push the simple rubber tube beyond the plate of the cricoid cartilage; whereas the above tube, supplied with a mandrin, can, *without exception*, easily be introduced beyond this place. As soon as the latter is passed, the mandrin is withdrawn, and the, now unarmed, tube slides down into the stomach and offers all the advantages of the simple stomach-tube—softness, etc. Neither can I deny that it causes me uneasiness in some cases if the lower end of the tube is open and if its wall, as has become customary recently, is disproportionately thick and stiff. I am not able, since I was unfortunate enough, many years ago, to tear out with a hard, stiff tube, the lower end of which was carefully rounded but open, and which acted like a gouge, a piece of gastric mucous membrane which upon withdrawal of the instrument was fastened in the lower opening of the latter corresponding exactly to its lumen, to overcome a certain suspicion of all tubes similarly constructed.

Employment of the Tube for Various Diagnostic Purposes—Lavage.—To decide the question how to proceed in employing the tube for diagnosis depends upon the purpose in view. *It is necessary, in order to examine the function of the stomach in regard to the time required to finish a meal partaken of*—i. e., the time after which the latter has completely left the stomach—*to practise lavage of the stomach.* The funnel is to be filled twice with about half a litre of tepid water each time, as I have often observed that the water, after the first lavage, may be returned almost clear and only after the second lavage may contain undigested remnants of food.

Expression of the Stomach Contents.—*If, however, it is the intention to determine the acidity of the stomach contents, Ewald's expression method is preferable to lavage,* because with this method we obtain undiluted stomach contents. The latter method is *not* a dangerous manœuvre, as is proved by thousands of cases; but, on the other hand, it is not so *absolutely* devoid of danger as is usually stated. I have noticed several times that some blood was expressed with the gastric juice, and have found in one case even a piece of gastric mucous membrane in the opening of the withdrawn tube. To avoid the latter possibility, I now combine with the instrument a tube and funnel; after the stomach contents have been expressed, I introduce some water through the funnel, and not until then do I withdraw the tube from the stomach, during the time that the water flows into the stomach, with the funnel in

a high position. For a number of years I have employed this precautionary measure *without exception* in every withdrawal of the tube from the stomach, because only in such a manner are we certain to avoid an eventual aspiration and a tearing of the gastric mucous membrane.

Another much-discussed question is to decide which stimulant should be employed to determine the *motor, resorbing,* and *secretory* power of the stomach and, incidentally, its conduct in a sensory respect during the process of digestion. A number of years ago I recommended, in order to examine the ability of the stomach to overcome a certain quantity of food within the time usually required for its conversion and expulsion from the stomach—i. e., to examine the "*motor power*" of the stomach—to have the patient partake of a certain *test meal,* as much as possible in keeping with the usual demands upon the stomach (consisting of a plate of soup, a beefsteak, a biscuit, and a glass of water; more exactly defined, according to Riegel, of 400 cc. of bouillon, 200 gm. of beefsteak, 50 gm. of bread, and 200 cc. of water), and then, after six to seven hours, to wash out the stomach. I am still of the opinion to-day that this method best answers the accustomed working conditions of the stomach and yields relatively the most reliable results as to the entire work performed by the stomach (in a secretory and especially in a motor respect). These results, taken of different individuals, are comparable among each other, because it has been found that, although the task is not always exactly the same, owing to the complicated condition of the test meal, a healthy stomach, nevertheless, with a few very rare exceptions, has always *surely* expelled the food after seven hours. Riegel has further employed this method to diagnosticate the chemical conditions of the digesting stomach, in examining the chemical changes of the stomach contents about four hours after ingestion of the test meal—i. e., during the height of digestion. There is no doubt in my mind that this method allows of a good insight into the secretory power and into the chemistry of gastric digestion, particularly as it has the great advantage over others that it considers the demands made upon the activity of the stomach by a larger meal partaken at the accustomed hour. However, I do not employ the test meal in the *especial* chemical examination of the stomach contents, because it appears to me that the stimulation of such a test meal is too dissimilar in the various cases to allow of a comparison of the results obtained one with another, and because a single washing out is not sufficient, as a rule.

Ewald's Test Breakfast.—This latter condition is not quite so much the case with Ewald's test breakfast, consisting of 35 gm. of a roll and 300 cc. of water (or a weak infusion of tea); but even this test breakfast is subject, although to a smaller degree, to the inequality of the stimulant. A test stimulant which excludes every objection has not as yet been found. It may be that possibly barley, which I proposed, acts more uniformly as a mechanical stimulant, while as a thermic stimulant ice water gives more uniform results, than Ewald's test breakfast. The objection which was made to the ice-water method, that it furnishes diluted gastric juice and, therefore, unsatisfactory results, does not affect the ice-water method, if the stomach contents are expressed in it, any more than other test meals, and, for this reason, I do not understand the animosity of some authors in rejecting this method. But, nevertheless, as the majority of physicians have adopted Ewald's test breakfast as the test of the secretion, I regularly employ the same for chemical diagnosis, because it introduces a stimulant which is at least nearly uniform, and to which the empty stomach is accustomed, and, being adopted generally by physicians, it permits, in the most cases, a comparison of the results obtained by the various investigators.

Chemical Examination of the Stomach Contents.—The *chemical examination of stomach contents* obtained by means of one of the above-mentioned methods embraces the qualitative, eventually the quantitative demonstration of hydrochloric acid, pepsin and peptones, lab-ferment, and lactic acid. The most important, at least, of the methods in use for this purpose is to be mentioned here.

The hydrochloric acid secreted by the gland cells, as soon as it appears at the surface of the gastric mucous membrane, is at once taken possession of, bound, by saliva, mucus, cast-off epithelia, but, above all, by the albuminous substances of the food and alkalies which may eventually have been introduced with the same. This combination of HCl to the albuminous substances is associated with a gradual

transformation of the latter into syntonin, albumoses, and, finally, into peptones. The amount of HCl which is not used in this chemical process remains in excess in the stomach as "*free*" *hydrochloric acid*, and as such it serves other (not less physiological) purposes, in particular displays antifermentative and antibacterial effects (see above). The acid reaction of the stomach contents at the height of digestion is principally due to the excess of HCl; but, at the same time, also by organic acids (above all, lactic acid) and acid-phosphate salts.

Qualitative Demonstration of Free Hydrochloric Acid in the Stomach Contents.—Quite a number of methods have been recommended and employed for the *qualitative demonstration* of free HCl. The method most in use is the examination of the stomach contents with *Congo paper*. The latter is not turned blue by the hydrochloric acid bound to organic matter, but actually only by the excess of HCl; however, free organic acids, if present in considerable quantities, will also give a positive reaction with the same. This is true of the tropæolin test; Congo and tropæolin, therefore, are reagents for *free* (inorganic and organic) *acids*. But other reagents are to be employed, if the presence of an excess of free *hydrochloric acid* (not in combination with other organic acids) is to be demonstrated with certainty. The phloroglucin-vanillin (Günzburg) and the resorcin (Boas) tests have proved to be useful in this respect, inasmuch as neither hydrochloric acid combined with organic bases and albuminous bodies, nor organic acids influence the reaction, which is brought about solely by free mineral acids—i. e., HCl. However, the test with Congo paper is undoubtedly sufficient for practical purposes, inasmuch as, although the turning blue of this paper by free organic acids is theoretically possible, this would require the presence of so large a quantity of free organic acid in the stomach contents as is very improbable ever to occur in practice.

Quantitative Demonstration of Free Hydrochloric Acid in the Stomach Contents.—Various methods are in use, according to the object in view, for the *quantitative* determination of HCl in the stomach contents:

(*a*) To ascertain the *total acidity*—i. e., the aggregate of substances causing the acid reaction (free HCl, combined HCl, organic acids, and acid salts)—it is best to employ a simple titration of a certain quantity of stomach contents with a decinormal soda solution with the addition of a few drops of a phenolphthalein solution (which is turned an intense red by alkalies) as an indicator. The amount of sodium consumed in titration is usually not calculated into the equivalent amount of acid; but the degree of acidity is simply expressed by the amount of cubic centimetres of decinormal soda solution necessary to neutralize 100 cc. of stomach contents—for instance, upon employment of 10 cc. stomach contents and consumption of 5.5 cc. decinormal soda solution, the degree of acidity is designated as 55. The latter fluctuates, under normal conditions, after a test breakfast between 40 and 60.

(*b*) To determine the quantity of *free hydrochloric acid* alone, decinormal soda solution is added to a certain amount of stomach contents until Günzburg's reagent no longer gives a reaction with a drop taken from the fluid to be examined (Mintz); if for this purpose we use, for instance, upon employment of 5 cc. stomach contents, 1.4 cc. of decinormal soda solution, this corresponds to a percentage of free hydrochloric acid of $1.4 \times 0.00365 \times 20 = 0.10$ per cent.

(*c*) Quite a number of methods have been recommended to determine the quantity, not only of the excess of free HCl, but also of that which is combined with organic substances and which has entered upon physiological action—i. e., the *total physiologically active hydrochloric acid*. I have employed, for a number of years, almost exclusively, the Braun method, having satisfied myself of the efficiency of the same.

The mode of procedure is as follows: At first, for the purpose of detection, the total acidity is ascertained of a certain quantity of filtered stomach contents (usually, 5 cc.), by means of titration with decinormal soda solution (see above). Then we add to another equal quantity of stomach contents (5 cc.) a few cubic centimetres more of decinormal soda solution than correspond to the neutralization of the first test. The alkaline fluid is now carefully dried in a platinum dish and reduced to ash in an open crucible, causing the organic acids to be evaporated in the form of carbonic-acid gas. The ash is then diluted (the dilution heated to drive off the free CO_2)

with as many cubic centimetres of decinormal acid solution (sulphuric acid preferably) as were previously added to cubic centimetres of decinormal soda solution to render the test alkaline, and, after this, titrated with decinormal soda solution with the addition of 2 drops of phenolphthalein solution. If, for instance, the amount of decinormal soda solution is X, this would correspond to an amount in the gastric juice of free HCl and of HCl combined with organic substance of $X \times 0.00365$ per 5 cc. $= X \times 0.00365 \times 20$ per cent.

Example 1. Test for total acidity: 5 cc. of stomach contents require for neutralization 3 cc. decinormal soda solution; this, therefore, would correspond to an amount of HCl of $3 \times 0.00365 \times 20 = 0.22$ per cent.

2. Test (direct determination of hydrochloric acid). To 5 cc. stomach contents are added 6 cc. of decinormal soda solution; this is reduced to ash, etc. To the ash are added 6 cc. of decinormal acid; then titrated with decinormal soda solution. Therefore of the latter are required 2.5 cc.—i. e., amount of hydrochloric acid $2.5 \times 0.00365 \times 20 = 0.182$ per cent.

The time required for this procedure is about half an hour.

The amount of HCl determined in this manner from gastric contents, expressed about one hour after ingestion of the test breakfast, varies in *healthy persons* quite considerably, 0.15 to 0.25 per cent. But, at any rate, it can be maintained that, under quite normal conditions, the amount of HCl should not deviate materially either above or below these figures.

An important fundamental question which concerns the generally employed method of quantitative examination of the stomach contents for acid, has recently been raised by Bourget and by R. Geigel. The latter demonstrated that the figures obtained in that determination of hydrochloric-acid percentage, which until now was exclusively employed, do not at all always run parallel with those which represent the *absolute* amount of HCl in the stomach—i. e., that amount which is present at a certain time upon a certain stimulation. He therefore keeps the relative and absolute acidity of the gastric juice strictly apart; he determines the latter in such a manner that, after a small quantity (2 cc.) of undiluted stomach contents has been expressed, one hour after the ingestion of an Ewald test breakfast (300 cc. fluid), and has been examined in the usual manner for its hydrochloric-acid percentage, now the stomach is washed out and 50 cc. of the entire washed-out water (about 2 litre) are treated in order to determine their contents of HCl. This will show the absolute amount of HCl which was present in the stomach, but at the same time also, from a comparison of the absolute and percentage quantities, show how much fluid was still present in the stomach at the time of examination. For example, determined by percentage, an amount of 0.35 per cent was found, therefore a relative hyperacidity; but the absolute quantity of HCl was only 0.45 gm. (while about 0.5 gm. is found in the normal), the gastric fluid therefore $45 : 0.35 = 130$ cc.

The test for *lactic acid* in the stomach contents is as follows: The fluid to be examined is first thoroughly shaken with ether free from alcohol, and then Uffelmann's reagent is added to the residue of evaporation of the ether containing the lactic acid. The reagent consists of a mixture of 10 cc. of a 4 per cent (about) carbolic-acid solution with 20 cc. of water, to which is added 1 drop of the tincture of perchloride of iron; the amethyst-blue colour of the mixture is changed to a "canary yellow" or "greenfinch colour" upon the presence of lactic acid. Lactic acid is *not* formed in the stomach under normal conditions (see above); if it is found in the gastric contents, it is either ingested with the food or it is the product of fermentation of carbohydrates in the stomach by bacteria, which can be isolated from the sputum of the mouth and from the gastric contents, and which possess the property of forming lactic acid from sugar.

The test of the gastric contents for albumin, syntonin, albumose and peptones, and the separation of the various products of digestion of albuminous bodies from one another does not present any difficulties. It is also easy to demonstrate by adequately performed digestion experiments whether the sample of gastric-fluid contains a sufficient amount of pepsin. However, the tests for peptones and pepsin action have as yet not attained the diagnostic significance of the acid determinations.

Test of the Rate of Absorption of the Gastric Mucous Membrane.—Specially to

test the *rate of absorption* of the gastric mucous membrane, Penzoldt's potassium iodide method may be employed, which is to incorporate potassium iodide in gelatine capsules into the stomach and to examine the saliva of the patient every five minutes. Thus an insight is gained into the rate of resorption of the gastric mucous membrane, at least as regards the above iodine salt, and at least a probable conclusion may be drawn as to the rate of resorption of the gastric wall in general. The first iodine reaction appears in the saliva within a quarter of an hour under normal conditions.

Another method, which especially takes into account the natural conditions of absorption, has recently been recommended by von Mering. According to this method measured quantities of two substances, one of which is surely absorbable whereas the other is absolutely inabsorbable, are introduced into the stomach; after some time the contents of the stomach are expressed and the quantitative proportion of mixture of both substances is determined. An emulsion of yolk of egg with sugar-water is most appropriate for the purpose. If, for instance, a healthy person takes, on an empty stomach, 250 cc. of an emulsion of yolk of egg containing 37 grape sugar to 10 fat, there will be, after the emulsion has been retained in the stomach for two and a half hours, only 31 grape sugar to 10 fat. As it is impossible that either a secretion in the stomach or a passage of the stomach contents into the intestine can alter the original proportions of the two substances to one another, the change in proportions of the mixture undoubtedly proves the *resorption* of the sugar by the wall of the stomach, and we are entitled to draw our conclusions as to the rate of resorption of the stomach wall from the proportionate decrease of the sugar in comparison to the fat.

We shall now pass on to the *diagnosis of the various affections of the stomach.* The differentiation of the various pathological conditions of the stomach has become decidedly more positive than has been the case formerly, thanks to the modern mode of examination by means of the stomach-tube, the main characteristics of which we have just enlarged upon. However, it goes without saying, that *many subjects are still a matter of controversy* in a domain in which only since a comparatively short time work has been done according to distinct principles, and, on the other hand, the danger is obvious that what has been found to be new in some patients may be too much generalized by the respective author. This occurred with the alleged excessive acid secretion in ulcer, with the absence of hydrochloric acid in carcinoma of the stomach, etc. *Thus especially a great deal is also very problematical in regard to the functional conduct of the gastric mucous membrane in gastritis,* because it has been entirely too little controlled as yet, whether the functional disturbances which are referred to gastric catarrh, actually are disturbances of a gastric mucous membrane which, by gastroscopical examination—for the present a *pium desiderium*—or post mortem, really proves to be inflammatorily affected. We should be prepared, therefore, that one or the other of those assumptions which to-day are considered to be correct or very probable, may prove to have been erroneous or may be essentially modified in certain respects.[1]

[1] I have endeavoured, in the discussion of the different affections of the stomach, to define their characteristics very precisely and purposely paid as little attention as possible to the *exceptions to the rule.* I consider it necessary, in view of the enormous, almost chaotic accumulation of individual observations and individual statements regarding the (especially chemical) conduct of the stomach in the various gastric diseases, to proceed in the diagnosis from those rules which hold good

CATARRH OF THE STOMACH—GASTRITIS IN ITS VARIOUS FORMS

We differentiate quite appropriately *acute* and *chronic* forms of gastritis, and in the complete picture of the former, according to form and intensity of the inflammation, the simple, diphtheritic, and phlegmonous varieties. Besides, a form of gastritis is also defined which is brought about by local poisoning, and, owing to the peculiar anatomical changes caused thereby, as well as on account of its typical development, it is specially described as toxic gastritis.

The most frequent, the simplest form of inflammation of the stomach is *acute "catarrh of the stomach."* The general covering of the free surface of the mucous membrane of the stomach and of the relatively deep stomach cells, with exquisitely muciparous cylinder epithelia, justifies the designation catarrh of the stomach. But it should not be overlooked that the inflammatory process of the gastric mucous membrane affects not only the muciparous elements of the mucous membrane, but also the glands of the stomach which enter the stomach cells and furnish the specific secretion of digestion, manifesting itself in granular cloudiness, fatty degeneration, and wasting of the cells. Besides, more or less marked hyperæmia of the mucous membrane is found with desquamation of the epithelia, eventually also an accumulation of round cells in the interstitial tissue. The effects of this pathologico-anatomical conduct of the gastric mucous membrane in acute gastric catarrh are changes in the production of mucus and secretion of gastric juice as well as of the functions of the stomach in general.

Dyspepsia.—Thus, *disturbances of the digestive activity of the stomach* become manifest in all directions—i. e., in *secretory, absorptive, motor,* and *sensory* respects. The total effect of the latter regarding the process of digestion is designated as impairment of digestion—*"dyspepsia."*

Symptoms of Dyspepsia.—This affection manifests itself by subjective and objective symptoms; the former are: Nausea, want of appetite or perversion of the latter for unusual dishes, increased thirst, pulpy, rarely bitter taste, cardialgia (pyrosis), feeling of fulness and general discomfort; furthermore, giddiness or actual headache, sometimes vertigo, general fatigue, and psychical depression. Of *objectively* demonstrable symptoms, I mention: Distention of the gastric region, yawning, usually coated tongue, herpes labialis (although very rarely), increased secretion of saliva, fœtid breath, sour or bitter eructations, and vomiting. *The examination of the vomitus or of the stomach contents obtained by means of the stomach-tube* after a test breakfast *will show in catarrh* of the stomach: *deficiency of hydrochloric acid*, lactic acid in more or less considerable amounts according to the food last partaken of, fatty acids, mucus and remnants of

---

at least in the majority of cases, unless the beginner is to lose an insight in diagnosis of gastric affection and—the enjoyment of diagnoses. I would ask therefore kindly to consider this point of view in forming an opinion regarding the various statements in the following chapters.

food which remained longer in the stomach than normally. *The examination as to the time of digestion by means of a test meal shows a retardation of digestion,* so that, after seven hours, undigested masses are found in the washed-out water.

Conduct of the Pulse.—Acute catarrh also affects the frequency of the *pulse,* inasmuch as the latter usually increases in frequency, then to fall after several days, as I believe, having observed this in the majority of cases, to normal or *below normal.* Often *defecation* is not normal either, sometimes it may be retarded, at other times diarrhœic; *jaundice* may also set in if the inflammation spreads to the duodenum or if a duodenal catarrh is caused by the irritation of the abnormal chyme entering the duodenum. The excretion of *urine* is usually scanty with a large amount of urates. The *temperature of the body* is mostly *not* raised. I give the diagnostic advice, whenever fever exists, to think of other sources of the fever than of acute gastritis, especially because most febrile diseases are associated with the symptoms of an acute gastric catarrh which is usually designated as secondary or sympathetic. It cannot at once be decided whether, in the latter case, an acute catarrh of the stomach always prevails which can be demonstrated anatomically (by accumulation of round cells in the interstitial tissue, uniform granulation and wasting of the gastric gland cells and marked secretion of mucus of the mucoid goblet cells, or whether the dyspeptic disturbances may not sometimes only be due to a nervous reflex action of the stomach upon the fever, which occurs in some individuals. It is certain, however, that fever in acute catarrh of the stomach is much oftener caused by other affections than by the latter. On the other hand, cases also occur, as can be easily determined, in which no other cause for the fever can be detected than the acute gastric catarrh. But they are very rare, according to my experience in such cases it is possible that the disease may commence with severe febrile symptoms, with chills and pronounced exhaustion, and the temperature may rise to 102.2° F. and above.

Ætiologico-Diagnostical Points.—The *ætiology* may also be of value in the diagnosis of acute gastric catarrh. The onset of acute gastritis is usually preceded by a *marked dietetic error,* by partaking of dishes very difficult to digest or indigestible by a sensitive stomach, or of tainted food and drinks. The cause of an acute gastritis can often be traced to the *excessive indulgence* in alcohol, especially if committed by people not accustomed to alcohol. It occurs rarely that an *excessively high or low temperature* of the ingesta, and excessive quantity of the same, or an exquisitely coarse condition of the food, *mechanically* irritating the surface of the stomach, give rise to gastric catarrh; but it may happen the affection is brought on by the swallowing of *foreign bodies* or chemically different substances (certain medicines, decomposed pus in gangrene of the lungs, etc.). Those gastric affections which occur in the course of certain *infectious diseases* (*influenza, erysipelas, scarlatina, etc.*), can be explained either as sympathetic dyspepsia (see above) or as actual (i. e., confirmed by autopsy) gastritides which are generated by the bacterial or chemical virus of these affections. Finally, manifestations of gastritis (loss of appetite, coated tongue, etc.) also occur after violent *mental emotions.* I, for my part, do not consider the very pronounced, often severe dyspeptic symptoms to be the expression of an acute gastric catarrh, but the result of nervous functional disturbances of the stomach, because I have observed that *they disappear immediately if the cause of the severe annoyance, etc., is suddenly removed.*

Differential Diagnosis.—The *diagnosis of acute gastritis* is not very difficult, accordingly; the symptoms of dyspepsia and the above-mentioned result of the examination of the gastric contents will usually not permit the thought of another affection to become prominent. If acute gastric catarrh commences with chills or less sudden onset of fever, it may be confounded with any number of *infectious diseases* which run their course without pronounced initial symptoms, such as enteric fever, small-pox,

malaria. In such cases, then, it will be absolutely *impossible* to make a *positive diagnosis.* All that can be done will be to wait and to pay attention to the course of the fever, to the eventual occurrence of an exanthema, etc., in order to substitute for the provisional diagnosis of acute gastric catarrh (which is almost always wrong in the presence of high fever), or rather the diagnosis "febrile, as yet indefinable affection," the positive diagnosis enteric fever, etc. The acute course of the affection also precludes the confounding of acute gastric catarrh with *other diseases of the stomach.* Nor is a confusion very likely with acute affections of other abdominal organs, although acute gastritis is often associated with acute enteritis. *Gall-stone colic* causes considerably more pronounced pains—*attacks* of pain—so that this affection is much more apt to be mistaken for gastric ulcer or gastralgia than for acute gastric catarrh, in which the pains in the epigastrium are always insignificant and should never be taken into account in the diagnosis. The same refers to the question whether a beginning *peritonitis* may simulate an acute gastric catarrh. Here, also, the pains almost always from the onset predominate in the pathological picture, but, besides nausea and vomiting, which may remind us of a gastric catarrh, other symptoms of peritonitis, painful urination, and especially collapse, become manifest; furthermore, the rapidly developing severity of the pathological picture and the direct demonstration of the exudate will soon disperse any doubt as to the nature of the affection.

This is different, however, in other forms of acute gastritis the course of which is often accompanied with very severe manifestations, and the diagnosis of which, therefore, should be specially enlarged upon. The picture of *peritonitis* is called to mind principally by

PHLEGMONOUS GASTRITIS—INTERSTITIAL PURULENT GASTRITIS—ABSCESS OF THE STOMACH

The principal symptoms of this very rare, *diffuse, purulent infiltration of the wall of the stomach,* in which the latter shows sieve-like perforations, are: vomiting (it should be well understood that pus has almost never been found in the vomitus as yet), pain in the epigastrium, which is scarcely increased by pressure, abdominal pains, mostly also meteorism and diarrhœa, collapse, delirium, fever, small, irregular, frequent pulse. These symptoms are evidently but little significant, and may all be ascribed to peritonitis which may accompany the disease, *so that a differential diagnosis cannot be made.* Not even when pus appears in the vomitus in this morbid picture, is it permissible to make a diagnosis of phlegmonous gastritis, as is proved by a case of acute *purulent* gastritis which I have observed. The principal symptoms of phlegmonous gastritis were all developed in this case; the severe pathological picture accompanied with collapse, severe pain in the gastric region, intense vomiting, fever, small, irregular, accelerated pulse. The vomitus contained pus, besides gastric epithelia, very numerous mycelium threads, and bacteria; yet no gastritis submucosa was found post mortem, but simply a high-graded inflammation of the gastric mucous membrane with unusually marked mucopurulent secretion on the free surface of the wall of the stomach.

Only a *probable diagnosis,* therefore, would be permissible, if the above-described pathological picture becomes exceptionally prominent in the course of *pyæmia, puerperal fever, or a severe infectious disease,* because the metastatic occurrence of phlegmonous gastritis, particularly in these diseases, has been demonstrated. However, this form of phlegmonous gastritis occurs far less frequently than the *primary* affection, the ætiology of which is as yet unknown.

The prospect of a diagnosis is more favourable of phlegmonous gastritis if it does not occur in the form of a diffuse infiltration, but as a circumscribed abscess of the stomach. It is true, the symptoms are the same, in general, as in the diffuse form, but eventually a tumour may be felt which disappears with vomiting of pus. But the diagnosis will always be doubtful, as it may be a question of an abscess which developed in the neighbourhood of the stomach and perforated into the latter, and which, as is obvious, cannot be differentiated from an abscess of the wall of the stomach.

If we exclude the *diphtheritic* form of acute gastritis, which is of no clinical but only of pathologico-anatomical interest, there remains the discussion of another form of acute gastritis which occurs quite often and which, as a rule, does not offer any difficulties, viz.,

TOXIC GASTRITIS

This affection represents an acute inflammation of the wall of the stomach, which, according to the quantity of poison acting in the stomach, is more or less severe. The poisons usually to be considered are: sulphuric acid, nitric acid, oxalic acid, rarely hydrochloric acid, corrosive alkalies, furthermore, concentrated alcohol, copper sulphate, phosphorus, arsenic, corrosive sublimate, potassium chlorate, nitrobenzol, etc.

The action of these poisons upon the gastric wall varies according to the nature and concentration of the noxa. Sometimes, as in poisoning with phosphorus, arsenic, and antimony, which are not actual corrosive agents, and also in alcohol intoxication, it is a question of cloudy swelling and fatty degeneration of the gastric gland cells, which may be supervened by peptic ulcers (if the mortification of the cells has assumed larger dimensions and extravasations of blood occur in the mucous membrane of the stomach due to the brittleness of the fattily degenerated vascular wall); at other times we may have to deal with severe anatomical changes of the gastric wall, actual corrosive effects, as in poisonings with concentrated acids and alkalies. Accordingly, the clinical picture changes in every instance; but a general pathological picture for the diagnosis of toxic gastritis may be described as follows: severe pains in the epigastrium, increasing upon external pressure, frequent vomiting of mostly blood-coloured masses generally giving no relief, unquenchable thirst, severe disturbances of the general health, small, frequent pulse, accelerated, superficial, thoracic respiration, collapse, cyanosis, sticky perspiration, disturbed sensorium. This may be followed by peritonitis and, according to the character of the poisoning, albuminuria, hæmaturia, petechiæ, jaundice, etc. It may occur that, as an effect of corrosive action, shreds of mucous membrane may become detached and be vomited, or an atrophy of the gastric mucous membrane with its sequelæ may remain. It is a diagnostic rule in all suspicious cases to examine the oral and pharyngeal cavities for corrosions, to control the smell of the breath, and chemically to examine the vomitus or the washed fluid for the presence of the poison.

Still another form of gastritis is to be mentioned additionally, the occurrence of which has been maintained recently, viz.,

MYCOTIC GASTRITIS

Although microbes which pass from the oral cavity into the stomach are not, as was assumed formerly, destroyed in the acid gastric juice, yet their growth is decidedly retarded, according to recent investigations. Yeast and mould fungi are more resistant to acid than the schizomycetes which develop better in gastric juice deficient in acidity. Therefore, it depends, above all, upon the prevailing deficiency of acid, upon the time of retention of the contents in the stomach, and upon the quality of the ingesta, which fungi develop in the stomach and in what quantities. We usually find yeast and mould fungi, sarcinæ, oidium albicans, and various kinds of schizomycetes (anthrax bacilli, etc.). But as all these fungi almost never exert a directly injurious action upon the gastric mucous membrane, and their presence in an affected stomach does not materially counteract a recovery, we must rather con-

sider the occurrence of fungi in the stomach contents as an accidental occurrence. It appears, however, that certain micro-organisms are able, in rare cases, to cause inflammation and abscess formation in the stomach; but so few unquestionable anatomical observations have so far been made of *mycotic gastritis* that it is advisable to refrain from the description of a clinical picture and from a diagnosis of the same.

CHRONIC GASTRITIS—CHRONIC CATARRH OF THE STOMACH

If it is possible to determine an injury to the structure and function of the stomach-gland cells in acute gastritis, this is much more markedly the case in chronic gastritis, in which the change of the gland cells (cloudy swelling, granulation, fatty degeneration, and wasting to obliteration of the epithelia) is a permanent one. Besides, there occurs considerable infiltration in the interstitial tissue and excessive secretion of mucus of the cells.

Excessive Production of Mucus—Reduction of HCl—Retention of Food in the Stomach.—*Accordingly, we find as principal symptoms of chronic catarrh of the stomach the presence of abundant mucus in the stomach and deficient production of digesting glandular secretions.* The consequence is that the ingesta are quite insufficiently digested and are caused to ferment and putrefy. This, it is true, could be prevented if the muscular action of the walls of the stomach would interfere compensatorily and expel the chyme undigested into the intestine within the normal time. But experience teaches us that this does not occur, or happens only very imperfectly, because the energy of the musculature is also affected by the inflammatory infiltration of the gastric wall. Thus a third principal manifestation of chronic inflammation becomes associated with reduction of secretion of gastric juice and abundant formation of mucus, viz., *retention of the ingesta in the stomach.*

These severe disturbances of gastric digestion can be easily demonstrated. The retention of the ingested food by the results of a test meal, the reduction of hydrochloric acid and of pepsin by the result of a test breakfast, the abundance of mucus by inspection of the vomitus and of the washed-out fluid, the decomposition of the ingesta by chemical examination of the stomach contents, in which, besides reduction of HCl and of ferments, the presence of butyric acid, alcohol, acetic acid, of micro-organisms, and of the products of albumin putrefaction can be determined.

Pathological Picture.—*The pathological picture* of chronic gastritis is in general an increase of the symptom-complex of acute gastritis. The principal manifestations are: pulpy, insipid taste, increased thirst, want of appetite or perversion of the same, coated tongue, acid or rancid eructations, pyrosis, nausea, yawning, increased secretion of saliva, vomiting which, in some instances, may occur at any time, while in others it takes place, with a certain regularity, during the morning hours and which causes the passage of a considerable amount of mucus (vomitus matutinus). Poor sleep, heaviness in the head, general fatigue, dislike of work, disgust of life and hypochondriasis may follow. The abnormal processes of fermentation, which develop gases, especially hydrogen and carbonic acid, in the stomach, cause distention of the epigastrium, a feeling of fulness in

the gastric region—in fact, actual pain of tension. The latter is increased by external pressure, but it is *diffuse,* not restricted to one small locality. Severe headache, vertigo and agoraphobia may occur, due to a reflex action upon the central nervous system transmitted by irritation of the gastric nerves; a reflex action upon the heart may produce cardiac asthma (dyspepticum), palpitation of the heart, and arrhythmic pulse. Constipation generally prevails, and the urine is sometimes scanty in the course of the disease.

Condition of the Stomach Contents in Chronic Catarrh of the Stomach —Mucus.—Besides the above-named symptoms, it is necessary, above all, to consider the result of the examination of the stomach contents obtained by means of the stomach-tube.

The most important constituent of the same in chronic catarrh of the stomach are more or less copious *masses of mucus* which are intimately admixed with the undigested food, and which appear in the washed-out fluid either as small shreds or in the shape of a large, diffuse, viscid conglomeration of mucus. Considerable amounts of mucus are also found —contrary to the condition of the healthy stomach—in the *empty* stomachs of patients with chronic gastric catarrh. It is true, smaller quantities occur also in the contents of a healthy empty stomach—in fact, even larger amounts are found in some instances—but it seems to depend principally upon the fact whether a healthy empty stomach also secretes, besides the mucus, larger amounts of HCl, which, as has recently been demonstrated by A. Schmidt, is able to digest the mucus. In contradistinction to this, mucus originating in the stomach of patients with chronic gastritis generally appears in the shape of swollen glassy masses, which are the more abundant in the gastric contents the lower the acidity of the stomach. Mucus secreted by the pharynx and œsophagus or by the bronchi, or swallowed mucus are easily differentiated from gastric mucus, because the former kinds are admixed to the washed-out fluid in isolated globules and the microscopical examination betrays their origin from the first digestive tracts or the bronchi by the presence of basement epithelium or of alveolar epithelium.

Reduction of Hydrochloric Acid.—Another important finding in the stomach contents of patients suffering from chronic gastritis is the *reduction* of pepsin and lab-ferment, and especially of *hydrochloric acid.* This is unquestionably the rule in by far the majority of cases of chronic catarrh of the stomach. However, exceptions occur, i. e., cases in which large amounts of HCl are excreted.

For this reason it has recently become customary to differentiate *acid* from *simple* catarrh as a special form of chronic gastritis, based upon the chemical examination of the stomach contents.

Acid Catarrh of the Stomach.—Regarding *excessive secretion of gastric juice,* those cases should be excluded, in the first place, which should not be classified as gastric catarrh, but as neuroses—i. e., as chronic hypersecretions (see below)—and in which the dyspeptic symptoms—acid eructation, pyrosis, sensibility of the epigastrium, etc.—should be regarded as secondary to the action of the acid upon the wall of the stomach, respectively its nerves. But there exist also undoubted cases of gastric catarrh, according to my experience, which are accompanied with decided

hyperchlorhydria. I leave it undecided, however, whether the still intact glandular regions secrete excessively in a compensatory manner, or whether it is a question of proliferation and multiplication of the parietal cells, as has recently been maintained by Mayens. But the deficient formation of digestive glandular secretion is always the rule in gastritis, the *typical* effect of chronic gastric catarrh. I also consider it just as unnecessary and going too far in specialization, owing to the prevalence of a production of mucus (besides a reduction of the digesting gastric secretions), to distinguish a *mucous* from "simple" chronic catarrh of the stomach as a *special* form, much as I value the endeavour more and more to define by precise examinations of the stomach contents the diagnosis of chronic catarrh of the stomach which, until recently, has been so frequently employed as an auxiliary diagnosis.

Another manifestation of chronic gastritis which can be determined in the examination of the stomach contents is the *longer retention of the ingesta* in the stomach. This is certainly the usual condition, caused, on the one hand, by the reduction of the secretion of gastric juice and by the secretion of considerable mucus, which prevents the saturation of the food with the digestive juices, and, on the other hand, by the inflammatory serous saturation, or even inflammatory atrophic degeneration of the musculature, which is followed by a weakening of the motor activity and by an extensive dilatation of the stomach. All these pathological factors act in the same direction—they cause an abnormally long retention of the ingesta in the stomach, and thus produce decomposition and abnormal fermentation of the food, thus giving rise to a further continuation of the gastric catarrh—i. e., creating a vicious circle. If we also consider that absorption is impaired in chronic gastritis, it appears obvious that abnormally long retention of the stomach contents is one of the typical symptoms of chronic catarrh of the stomach. Exceptions to this rule may occur if the musculature develops compensatorily increased energy, forces the remnants of food into the intestine before they are fully transformed and gradually hypertrophies, before, in the further course of the affection, it becomes subject to degeneration and atrophy.

Differential Diagnosis.—The diagnosis of chronic gastritis is not difficult in most cases in consideration of the above-mentioned symptoms, and especially of the last-referred-to result of the diagnostic lavage of the stomach. But it should not be forgotten that chronic catarrh of the stomach sometimes complicates *gastric ulcer* and is the usual companion of *carcinoma of the stomach and of gastrectasis. I have therefore always made it a diagnostic rule to assume simple gastritis only when the last-named morbid conditions may be completely excluded.* The differential diagnosis, however, will not be entered upon until we discuss the various other diseases of the stomach. I only wish to state here that cases of *gastric neurosis* also may often give rise to differentio-diagnostical considerations. Marked changes in the intensity of the dyspeptic symptoms, especially temporary presence of a good appetite, furthermore, complete emptying of the stomach after test meals within the normal time, and the absence of large quantities of mucus in the washed-out stomach contents allow, at least in most cases, the positive exclusion of gastritis, and make the diagnosis of neurosis of the stomach possible.

Ætiologico-Diagnostical Points.—The consideration, finally, of the *ætiology* of chronic catarrh of the stomach may also give some support to

the diagnosis. A long-lasting, or often, in short intervals, repeated action of noxæ especially affecting the stomach: Habitual excesses in eating, and particularly in drinking, insufficient mastication, etc., is the most frequent cause of chronic catarrh of the stomach. A predisposition is further created by other affections of the stomach, especially often by carcinoma, either by general maladies (anæmia, chronic tuberculosis, Bright's disease, etc.) or by long-lasting stases in the venous system, both in the system of the cava (in affections of the heart and lungs) and also in particular in the portal-vein system, to which facts we have repeatedly referred in the discussion of the diagnosis of diseases of the liver. Often following chronic catarrh of the stomach as sequelæ are *gastrectasis* and *atrophy of the gastric mucous membrane.*

ATROPHY OF THE MUCOUS MEMBRANE OF THE STOMACH—ANADENIA OF THE STOMACH—ACHYLIA GASTRICA

Atrophy of the Gastric Mucous Membrane.—We owe the precise knowledge of this pathological condition to the investigations of Fenwick, Quincke, Ewald, and others. The results of autopsies have demonstrated that the glandular tubules with their cells are destroyed by small-cell infiltration and parenchymatous degeneration which occur in the mucosa or, in other cases, by interstitial connective-tissue processes of proliferation, gradually involving also the muscularis in the atrophic process. Anadenia is usually, as stated previously, the effect of severe, especially toxic, gastritides, or of carcinoma of the stomach. But general constitutional diseases, such as diabetes mellitus, carcinoma of organs other than the stomach, may also give rise to atrophy of the gastric mucous membrane. It has been particularly observed that the affection has taken its course in the picture of pernicious anæmia, and it sometimes remains questionable whether achylia always represents the primary disease or whether, *vice versa*, severe anæmia, the same as in other constitutional affections, may not cause the development of atrophy of the gastric mucous membrane.

It might be expected that the symptoms of the disease are very pronounced, and that the diagnosis therefore does not present any difficulties. But this is not always the case, inasmuch as, when the mucous membrane alone atrophies and the musculature is not also affected, the deficiency of secretory function of the stomach is regulated by the compensatorily interfering activity of the muscles—i. e., if the passing of the undigested food from the stomach takes place within the normal time and the process of digestion is more or less incumbent upon the intestine. But so soon as the motor power becomes impaired, the signs of dyspepsia set in. The appetite is decreased, and there exists particularly an aversion to meat; fulness and pressure in the gastric region are noted, sometimes increasing to marked diffuse pains, also eructation and vomiting (but always without admixture of blood)—in short, the entire complex of dyspeptic symptoms manifests itself. Naturally! For in total atrophy of the mucous membrane there can no longer be any question of secretion of gastric juice. A digestion of the ingesta is impossible; they remain in the stomach and decompose unless the muscular activity interferes compensatorily.

Stomach Contents in Anadenia.—Accordingly, we find the ingested food undigested, in the process of fermentation, when examining the washed-out contents of the stomach; the fluid contains more or less lactic acid, but is *free from hydrochloric acid, pepsin, and lab-ferment;* the microscopical examination can demonstrate neither gastric epithelia nor blood corpuscles, at most round cells, and, of course, bacteria. *Mucus is also absent in the stomach contents* if the entire mucous membrane has been destroyed, whereas in the earlier stages of the disease, contrary to the above, larger quantities of viscid mucus are found which, with the simultaneous total absence of hydrochloric acid and pepsin, points distinctly to the presence of an atrophy of the mucous membrane. As determined by A. Schmidt, at the atrophied areas of the mucous membrane, a metamorphosis occurs of the common epithelium of the stomach into an abnormal form of epithelial cells with interspersed goblet cells which secrete abundant mucus, and not until this epithelium has also been destroyed does the formation of mucus cease entirely.

Effects of Atrophy of the Mucous Membrane.—Another effect of atrophy of the gastric mucous membrane is that, upon cessation of the HCl secretion, the acidity of the urine does not decrease as under normal conditions during digestion. Gradually the general nutrition becomes greatly impaired, especially when the changes in the stomach become complicated by a similar process in the intestine (a wasting of the glandular apparatus and of the villi), if diarrhœas set in and the intestine is no longer able to assume the vicarious digestion of the ingesta which remained undigested by the stomach. I have already mentioned that the course of some cases runs entirely in the picture of severe progressive pernicious anæmia.

Differential Diagnosis.—As easy as it is to diagnosticate this affection from the above-described syndrome in the far-advanced cases of atrophy of the gastric mucous membrane—i. e., from the presence of a severe disturbance of the general health and of pronounced dyspepsia with constant total absence of hydrochloric acid and digestive ferments in the gastric contents—as difficult does the diagnosis become in those cases in which, although the findings in the gastric contents as washed out after a test breakfast are negative, yet the food leaves the stomach within the normal time (see above). Here the question suggests itself whether or not a nervous affection of the stomach prevails with total suspension of the function of the secretory factors of the stomach brought on under nervous influence. Such cases of *nervous achylia* occur, as clinical experience teaches us, although observations which are entirely unobjectionable, and autopsy reports are still lacking. The differential diagnosis, therefore, can only be made from the result *alone* of the stomach contents washed out after a test breakfast, if a large quantity of viscid mucus can be determined in the stomach contents, with absence of hydrochloric acid and digestive ferments, which would speak directly in favour of atrophy of the mucous membrane, especially if the masses of mucus disappear in the further course of the disease —i. e., if, finally, the formation of mucus becomes suspended. But if cases come under observation in which the latter event has already occurred, it

is only possible to answer the question whether atrophy of the mucous membrane is present, or nervous achylia (without anatomical changes in the stomach), from the history (anamnesis) and the accompanying symptoms. To atrophy of the mucous membrane point: the development of the affection from chronic, respectively toxic gastritides and carcinoma, the synchronous presence of a severe, progressive, pernicious anæmia or cachexia. The presence of a purely nervous achylia, on the other hand, is favoured by the fact that the above-mentioned ætiological factors in the history are absent, but nervous-dyspeptic and pronouncedly hysterical disturbances were marked from the beginning. The diagnosis becomes absolutely certain if repeated examinations of the gastric function give sometimes absolutely negative, at other times positive results—i. e., if at one time no digestive juice, at another time hydrochloric acid and ferments can be demonstrated in the gastric contents. However, only the positive result—i. e., determination of such a change in the intensity of the secretion, and not a constantly negative finding—must be utilized in the differential diagnosis, because clinical experience teaches that the character of neuroses of secretion in general is quite usually adhered to with great tenacity in the same direction and to the same extent in the given case.

The differential diagnosis between atrophy of the mucous membrane and certain cases of gastric carcinoma may sometimes present great difficulties. Common to both is the deficiency of hydrochloric-acid reaction as well as the absence of pepsin and lab-ferment in the stomach contents examined. It is obvious that in the greatest majority of cases the tumour, the coffee-ground vomit, the pains in the stomach, etc., do not admit of any doubt that we are dealing with carcinoma and not with anadenia of the stomach. However, cases exist in which these pathognomonic symptoms of carcinoma are absent for some length of time. Under such circumstances it is necessary to leave the diagnosis in suspense until a tumour becomes palpable, or even until the positive demonstration of cancer elements by microscopical examination of the stomach contents renders the character of the gastric affection indubitable.

Amyloid degeneration of the gastric mucous membrane, finally, may give rise to confusion with atrophy of the mucous membrane. Amyloid degeneration of the mucous membrane may also lead to the cessation of gastric-juice secretion, as was first determined in Riegel's clinic. The differentiation of both conditions is in reality not difficult, however, because simultaneous amyloid degeneration of spleen, liver, and kidneys, with their marked symptoms, and the demonstration of tuberculosis, syphilis, and long-lasting suppurations as ætiological factors will lead the diagnosis in the right direction.

ULCUS VENTRICULI PEPTICUM—GASTRIC ULCER

Diagnostically Serviceable Symptoms of Gastric Ulcer.—The *diagnosis* of *gastric ulcer* is a task which often confronts the physician. It can often be made with ease and precision; in other cases it must be left in suspense, and sometimes becomes certain only by the therapeutic success of a cure

which was directed against the ulcer. If we were to make the diagnosis of gastric ulcer only in those cases in which its principal symptoms, pain and gastric hæmorrhage with or without perforation are present, it would only be possible in the smaller number of cases of ulceration. Only the *pain* is almost constant, and in the vast majority of cases the only symptom which can be determined by the physical examination of the ulcerated stomach. The palpation of a thin tumour, corresponding to the thickened round and hard borders of the ulcers is successful only in the rarest cases, according to my experience; at any rate, the possibility of palpating such thickened ulcerated localities presupposes very favourable conditions for palpation and great technical skill. Of course, it is easy to palpate secondary muscular hypertrophy at the pylorus or tumour-like peritoneal masses of exudate in the neighbourhood of the ulcer. Gastric hæmorrhage is relatively rare, perforation still rarer. Let us briefly sketch the picture in which the ulcer usually takes its course, and let us investigate which symptoms are suitable in rendering the diagnosis of gastric ulcer feasible.

The patients complain, the same as other patients suffering from gastric troubles, of *dyspeptic* symptoms, but *sensations of pressure* in the gastric region are prevalent; but, besides changes of appetite, acid eructations, pyrosis, nausea, and vomiting are frequently present.

Vomiting.—The vomitus mostly has an acid reaction and shows, upon a test with Congo paper, and especially on a more precise chemical examination, an *increased amount of hydrochloric acid*; the latter is absent, of course, if vomiting occurs shortly after the ingestion of food.

The vomitus contains *blood* in a great many instances. This to physicians who attend only to private practice appears to be a rarer symptom in the course of gastric ulcer than to hospital physicians, simply because hæmatemesis is an event the severity of which is the first instigation to the patient to consider the affection more seriously and to go to the hospital. Thus, for instance, a compilation of the cases clinically treated by me shows the presence of hæmatemesis in more than one half of the cases. The dejecta are black after a marked hæmorrhage into the stomach has taken place; the tarry condition of the fæces is, in some cases, really the only symptom of the gastric hæmorrhage, inasmuch as the entire blood extravasated into the stomach is evacuated through the intestines, and no hæmatemesis therefore occurs.

The vomited blood can almost always be at once recognised as such. It rarely assumes a coffee-ground appearance, unless the blood which extravasated into the stomach has remained there for some length of time and the hæmoglobin has been under the influence of the gastric acid and transformed into hæmatin. The examination with Almén's reaction or, better, Teichmann's hæmin test, will decide in a doubtful case whether the masses suspected to contain blood actually do contain blood. Hæmatemesis is sometimes accompanied with a slight rise of temperature.

Pain.—Vomiting usually occurs soon (half an hour to one hour) after the meal the same as the *pain*. The latter, otherwise the most unreliable point of support for positive diagnoses, is necessarily to be utilized in the diagnosis of gastric ulcer. The pain occurs in paroxysms and is usually produced by ingestion of particularly indigestible, cold or hot food, or by

the irritation of the accumulating of more than abundant acid, in which case the administration of sodium bicarbonate may cause a temporary cessation of the pain; external pressure generally increases, very rarely ameliorates it. *The observation of the pain slightly gains in objectivity* by the fact that it is usually *concentrated upon one certain locality of the gastric region,* either in the epigastrium or near the vertebral column between the scapulæ, but, above all, by the other fact that *the intensity of the pain depends in a very great number of cases upon the posture of the patient,* thus, for instance, appears constantly only when the patient is in the right lateral position. I wish especially to emphasize the last-named conduct of the pain, because it has often helped me to make a diagnosis in doubtful cases. Tugging of nerves in the ulcer or direct chemical or mechanical irritation of the surface of the ulcer by shifting of the stomach contents are the causes of such a variation in the intensity of the pain upon change of posture of the patient. The *seat* of the pain is usually in the pit of the stomach immediately below the xiphoid process, more rarely to the right or even to the left of it.

Sometimes the so far doubtful diagnosis becomes certain by a supervening perforation of the ulcer into the pleural or pericardial cavities, into the peritonæum, into the skin, etc.; in other cases the ulcer heals, but the cicatrix causes conditions which produce a symptom-complex, and which require a special diagnosis, thus *gastrectasis due to cicatricial stenosis of the pylorus and gastralgias due to cicatrization.*

Absence of Pain after Complete Cicatrization.—The latter, however, is, according to my experience, a *very rare* source of pain and dyspeptic manifestations, and I wish to caution most emphatically, not to make the diagnosis of cicatrization and its consequences, in the interest of the patient's and our own comfort, if after a Carlsbad or rest cure some pains or some dyspeptic symptoms have remained. These diagnoses are almost always wrong, in my experience. Contrary to the usual assumption, I maintain that cicatrices of gastric ulcer, except in very rare cases, do *not cause pains,* but that, rather, if pains remain in the course of treatment of gastric ulcer, they are almost without exception due to the fact that the ulcer has not completely healed or that a relapse has occurred. Another repetition of strict dietary measures will undoubtedly clear the situation in such cases and lead to the desired therapeutic end.

The condition of the *stools* of patients suffering from gastric ulcer is of no value diagnostically, because they are not constantly the same. This holds true of the condition of the *urine,* although at the height of digestion it shows, contrary to the condition in other gastric affections, decreased acidity, and less of it is voided in cases in which the patients vomit considerably.

The diagnosis of gastric ulcer can be made with certainty in a great number of cases, especially by reason of the bloody vomit; in a still greater number of cases, especially in private practice, it remains a provisional diagnosis, because it is principally dependent upon the presence of a more or less pronounced dyspepsia and of gastric pains. Lately it had seemed that the diagnosis of gastric ulcer would considerably gain in certainty as it was believed that an excessive formation of hydrochloric acid could be demonstrated as a *constant* manifestation of gastric ulcer.

Excessive Formation of HCl in Gastric Ulcer.—Were this true it would be of the greatest significance in the diagnosis of the ulcer. However, recent experiences have proved that such a hyperchlorhydria, although frequent in gastric ulcer, is by no means a constant symptom.

Besides, in the greatest majority of cases a determination of the gastric acidity by means of the stomach-tube must not be undertaken. I am not in a position to change my opinion that the employment of the tube for diagnostic purposes is not devoid of danger in ulcer, and is unnecessary in most cases, because a dietetic treatment of ulcer, based upon a provisional diagnosis, can never do any harm but only be of advantage. I do not mean to imply with these remarks that I do not consider the finding of gastric superacidity in ulcer of the stomach to be very meritorious and appropriate essentially to enhance our knowledge regarding the symptomatology and cause of this affection; on the contrary, I consider the now positive demonstration of the frequent coincidence of ulcer and superacidity a very gratifying confirmation and establishment of the views stated by me twenty-one years ago, regarding the origin of gastric ulcer (especially of the postulate regarding the origin of gastric ulcer of a temporarily abnormal acidity of the gastric juice). Only, from my standpoint, I am not in a position to recommend the diagnostic sounding of the stomach just for this affection. This at once leads us to the question whether the ætiology of ulcer will supply us with certain points for the diagnosis which render the assumption of gastric ulcer *a priori* probable in the given case.

Ætiological Points of Support.—As such may be considered: violent blows which strike the external abdominal walls and with it the wall of the stomach (I have seen several very conclusive examples); furthermore, irritations affecting the *mucous membrane* of the stomach of a mechanical, chemical, or thermic character, and, finally, emboli originating in the heart, a very rare cause, in my experience, for the formation of gastric ulcer, in spite of the great importance which was attached to this ætiological factor in particular from an experimental standpoint.

In all these cases it is a question of a lesion of the gastric mucous membrane with or without preceding hæmorrhage, or an interruption of the blood supply at a circumscribed locality of the wall of the stomach, upon which then, of course, the gastric juice exerts its peptic action, causing a loss of substance of the mucous membrane of the stomach. The question, then, in the given case will be, whether the lesion heals rapidly, as was observed in stomach wounds which were caused accidentally or experimentally, or not. The latter will be the case if any factor exists which retards recovery. Such a factor is undoubtedly *anæmia*, respectively chlorosis, in which, as is well known, the development of gastric ulcers occurs extremely often, and which, artificially produced, evidently retards the healing of losses of substance, as has been proved experimentally.

The explanation why this is the case meets with certain difficulties. The gastric juice does not digest the wall of the stomach under *normal* conditions. It is true, as the experiment teaches us, a living frog undoubtedly shows the process of digestion by the gastric juice, but the *wall of the stomach* itself is not affected, owing to its inherent (possibly adapted) resistant property. It is different, however, if the circulation is interrupted in some portions of the same, and thus its nutrition is locally injured (as is the case by traumatism, embolus, etc., possibly also, according to the experiments of Talma, by excessive tension of the gastric wall, especially also a spasm of the pylorus), and if these conditions are aggravated by an increase of hydrochloric acid in the gastric juice. There can be no doubt at present that such an increase is of great significance in the pathogenesis of ulcer of the stomach, after it has been proved experimentally that the protoplasm of the gastric wall is the more corroded and destroyed at weaker portions of the gastric wall, the more HCl is contained in the gastric juice acting upon the same. Now, it is well known that hyperchlorhydria of the gastric juice is of frequent occurrence in the course of chlorosis. But it must not necessarily be primary, but may, as I must assume according to my experience, supervene secondarily upon ulcer as a subsequent manifestation and favour its continuation.

The diagnosis of ulcer of the stomach is easy in some cases, difficult in others, especially because certain other diseases present symptoms in their course which are similar to those of ulcer. Most frequently to be considered differentio-diagnostically are: gastralgia, also intercostal neuralgia, carcinoma of the stomach, gall-stone colic, and duodenal ulcer.

Differential Diagnosis—Duodenal Ulcer.—Regarding the latter, it is impossible in most cases to differentiate ulcer of the stomach from that of the duodenum. The symptoms in the latter must be the same as in gastric ulcer if situated in the neighbourhood of the pylorus. Some support, although by no means positive, is rendered by the seat of the pain which, in duodenal ulcer, is usually more towards the right parasternal line, while in ulcer of the pylorus it is mostly concentrated upon the epigastrium in the median line or in the right sternal line. The fact that vomiting is usually absent in duodenal ulcer, and that, if hæmorrhages occur in the latter case, the blood originating in the ulcerated region is usually evacuated only towards the intestine, should also be considered in the diagnosis, so that, in cases in which repeated hæmorrhages occur through the intestine *only*, the suspicion, at least, of duodenal ulcer is justified. The complication of jaundice with the latter is too rare to be of diagnostic value. The determination of superacidity of the gastric juice may possibly be of importance in the future as being more in favour of gastric ulcer, although, theoretically, this should also be assumed in the formation of a duodenal ulcer.[1]

But, on the other hand, the examination of the gastric juice as to its HCl acidity, which, in particularly doubtful cases, unless a tendency to hæmorrhage prevails, is also exceptionally made by me, is of determining significance in the *differential diagnosis between gastric ulcer and carcinoma of the stomach. An increased amount of hydrochloric acid is decidedly in favour of gastric ulcer*; although normal or even decreased acidity may occasionally also occur in gastric ulcer, but is rare in any case, and entire absence of free hydrochloric acid has, to my knowledge, never been observed in a case of ulcer of the stomach. In carcinoma, however, an abnormally high or a normal, in fact an acidity of the gastric juice that is but slightly diminished, are great exceptions—I can only quote very few examples from my clinical material—the rule in carcinoma is rather absence of free hydrochloric acid or, at least, reduction of the same to a minimum. The extremes, hyperchlorhydria and entire absence of free hydrochloric acid, are therefore certainly of use in the differential diagnosis, whereas acidities midway between these extremes are of less value in diagnosis.

But I wish to emphasize regarding the extremes also, that they only, as a rule, determine the diagnosis ulcer or carcinoma. For, as a considerable catarrh of the stomach may become associated with gastric ulcer, it is not surprising that, in some cases of ulcer, the, originally increased, secretion of HCl is materially reduced later; and, on the other hand, formation of carcinoma is relatively often the direct result of ulcer, so that it is quite conceivable why even hyperchlorhydria has been found in some instances in a recently developing carcinoma. I have once determined,

[1] A case in my practice of duodenal ulcer that terminated in death by hæmorrhage showed a low amount of hydrochloric acid, viz., 0.16 per cent HCl.

in a case in which formation of carcinoma had begun at the base of two open ulcers, in masses vomited six hours after the last meal, 0.27 per cent of free hydrochloric acid! Nevertheless, such exceptions will not undo the average rule, and the above-mentioned diagnostic conclusions, to be drawn from the existing gastric acidity, will remain in force.

As to the other differentiating symptoms between ulcer and carcinoma, they are of little value diagnostically, unless they are symptoms, like a palpable tumour, which speak directly in favour of carcinoma,[1] in which case a confusion is absolutely impossible. In general, *in favour of ulcer* and against carcinoma are primarily the *youthful age* of the patients, inasmuch as three fourths of the cases of ulcer occur before the fortieth year, whereas carcinoma is found, *vice versa*, in three fourths of the cases after forty, and especially often not until after the sixtieth year of life. Exceptions to this rule occur in both directions; thus, for example, I have treated a patient of twenty-six years of age who suffered from carcinoma of the pylorus, as was shown by autopsy; cancer of the stomach was several times observed even in children. Furthermore, in a doubtful case, a point against carcinoma and in favour of ulcer is *the slight emaciation and absence of cachexia* (striking exceptions are found here also. I have seen a patient suffering from ulcer who weighed about 200 pounds, lose about 100 pounds, and patients with carcinoma gain several pounds per week) *vomiting of pure blood*, while in carcinoma larger masses of unaltered blood are almost never vomited, but only the well-known ominous coffee-ground masses. Finally, ulcer is generally favoured by perforations if they occur in early stages of the disease, whereas they do not make their appearance until after the affection has lasted for some time, and relatively early only when a carcinoma develops on the base of an ulcer. The character of the pain, the appearance of the tongue, the condition of the unbloody vomitus, the appearance of the stools, etc., are all too changeable and too unreliable ever to make a diagnosis dependent upon them.

Differential Diagnosis between Ulcer and Neuralgias.—Easier is the differentiation of gastric ulcer from *gastralgias* and *unilateral intercostal neuralgias*. The decidedly *paroxysmal* occurrence of the pain, the frequently long duration of the interval between two attacks, the irregular effect of irritants acting directly upon the wall of the stomach (for instance, that even food very difficult of digestion can be partaken of without disturbances), the synchronous presence of hysteria, neuralgias, uterine diseases, speak decidedly in favour of the gastralgic character of the pains, which is still more favoured by the constant absence of bloody vomit and of any kind of dyspepsia during the painless time, which does not very often occur in ulcer, at least according to my experience. The determination of hyperchlorhydria is not so significant as may be supposed, in case of doubt in the diagnosis of ulcer in comparison to gastralgia. For hyperacidity is often associated with gastralgic pains without the gastric mucous membrane being necessarily at the same time affected by an ulcer; nor is

[1] Non-malignant hypertrophies of the muscularis occurs occasionally in ulcer, the same as tumour-like, circumscribed perigastritic exudations; but they are only rarely to be considered differentio-diagnostically.

the time of the occurrence of the pains determining, as in both cases the attacks of pain set in principally after the ingestion of irritating food. Of especial importance for the differentiation of gastralgia and ulcer is the suspension of the pain by external pressure in gastralgia, while palpation usually increases the pain in ulcer.

But the last-named characteristic is of little value; I, at least, would not like to make a positive diagnosis on the strength of it. It would be rather more advisable to employ the electric current *during the process of digestion* as a means of examination. If the pain disappears entirely upon application of the same (especially of the anode), it would speak in favour of gastralgia; if it persists, either affection, gastralgia or ulcus, may be present. Only the positive finding—i. e., the temporary *suspension* of the pain by the electric current—is of some significance diagnostically. *Intercostal neuralgias* sometimes also present some diagnostic difficulties, according to my experience, if the painful area is situated in the epigastrium and if dyspeptic disturbances happen to accompany it, or if a filling of the stomach causes pain. Diagnostic doubt can usually be easily removed if the abdominal walls are raised in a strong fold and the latter, without exerting pressure below, is indented in various places and examined as to an eventual painfulness. It is also easy, as a rule, to demonstrate painful areas in the course of a certain intercostal space.

Gastric Ulcer and Cholelithiasis.—Finally, it has often happened to me that I was doubtful in the differential diagnosis between *gall-stone colic* and gastric ulcer, and that the situation did not become clear until after the passage of gall-stones. We should be guided principally by the result of the acid determination of the vomitus (which, in case of hyperacidity, almost surely speaks in favour of ulcer), but, above all, by the distention of the gall-bladder and by the jaundice, and also by the *swelling and painfulness of the liver, especially upon palpation of the border adjacent to the gall-bladder*, which decides in favour of cholelithiasis. As to the remainder, I refer to what has been stated in the diagnosis of cholelithiasis.

It has been positively determined that other ulcers also occur in the gastric wall besides the usual peptic ones, viz., *syphilitic* and *tuberculous*. But their diagnosis is impossible because their symptoms are the same as observed in peptic ulcer. Besides, tuberculous individuals and such as are debilitated by syphilis are very much predisposed, owing to the weakening of their constitution, to become affected by a simple gastric ulcer; and robust persons afflicted with syphilis, on the other hand, are as little protected as others occasionally to acquire a simple gastric ulcer. Those who do not on principle object to diagnoses *ex juvantibus*, may, as much as they please, draw an inference upon the syphilitic nature of the gastric ulcer in question from a strikingly remarkable favourable success of a specific cure.

Still one word as to the diagnosis of the *seat* of the ulcer in the given case. It can be diagnosticated in the rarest cases only. The strict localization of the pain upon a certain small area, its regular occurrence when the patient assumes a certain posture, may give rise to the suspicion that the painful area corresponds to the seat of the ulcer. But it is best never to attempt such diagnoses. The only facts to be considered would be if gastrectasis develops besides the symptoms of ulcer, which speaks directly in favour of the seat of the ulcer being in the pylorus, and, further, if, upon the passing of solid, cold and hot food into the stomach—i. e., during the last act of deglutition, pain occurs regularly under the xiphoid process. Cardiac ulcer may be diagnosticated in the latter instance, and we should

take our therapeutic measures accordingly, especially prescribe only cool, fluid, or pappy diet, etc. But, above all, no diagnostic sounding should be done in such a case, because, according to my experience, an abundant hæmorrhage is very apt to be brought about by the passing point of the tube, which is bound to touch the ulcer, thus producing a desperate situation to both physician and patient.

CANCER OF THE STOMACH—CARCINOMA VENTRICULI

Carcinoma of the stomach is in a certain relation to gastric ulcer in so far as it may be produced by the latter. I have seen several too remarkable cases to doubt the inherent connection between both diseases. One of the most striking is the following case, which proves how impossible it is sometimes diagnostically to separate carcinoma from ulcer:

Case of Cancer of the Stomach simulating Ulcer.—Officer N., about thirty-two years old, while traveling acquired a severe intestinal hæmorrhage with discharge of large quantities of pitch-black stools; the appearance of the otherwise robust man was totally anæmic. As it seemed that, according to history and symptomatology, the typical picture of gastric ulcer presented itself, he was ordered a rest cure with strictest observation of diet. A remarkable improvement followed, so that the patient, after three weeks, could partake of beefsteaks, etc., with great appetite and without pain, gaining about 3 pounds in weight per week. Suddenly, during apparently complete recovery, symptoms of perforation occurred; chills, severe painfulness and drum-like distention of the abdomen, with collapse. Death occurred after two days. If ever symptoms pointed to simple gastric ulcer, it was the case in this instance: youthful age, absence of dyspepsia, of vomiting, and of any indication of tumour, discharge of large quantities of blood in the stools, gain in weight of several pounds during the cure, and sudden perforation during apparently complete recovery. The autopsy (Hauser) revealed an ulcer the size of a two-mark piece in the pylorus, with flat, carcinomatously infiltrated borders and a small perforation.

However, the diagnosis of gastric carcinoma, if we are certain that the stomach is the affected organ, does not present any difficulties in the majority of cases.

Diagnostically Useful Symptoms of Gastric Cancer.—It is true, at the onset cancer of the stomach takes its course in the picture of chronic gastric catarrh (loss of appetite, eructation, coated tongue, pressure in the epigastrium), and cannot be differentiated from the latter. But gradually the diffuse painfulness becomes more prominent, it may possibly become localized in the area of the carcinomatous tumour, and may, although rarely, assume a paroxysmal character; pain is almost never absent during the entire course of the disease. Vomiting, almost constant, and especially well marked if the seat of the carcinoma is in the pylorus, brings out remnants of food, mucus, epithelia, and fungi, but, above all, the ominous *coffee-ground-like masses.*

Characteristic Vomiting.—As little as it is permissible at once to conclude from these masses upon the presence of carcinoma of the stomach, just as little should this symptom ever be considered insignificant. For, although it is possible that the blood extravasated into the stomach may assume a chocolate-like appearance in other affections also (see p. 274), it occurs incomparably less often in these than in cancer of the stomach.

The vomiting of coffee-ground-like masses is, and always will be, very suspicious in every instance. Bright blood is vomited but rarely, only when larger vessels become eroded by the carcinomatous ulcer, and if the blood is not retained in the stomach for some time. Owing to the deficient digestion and to disintegration of tissue albumin, caused by the carcinoma as such, the symptoms of *cancer cachexia* occur then in the course of the affection, viz., a yellowish pale complexion, emaciation, and slight œdema. The latter symptom rarely assumes larger dimensions, but if it does, it is apt to simulate another disease, especially nephritis.

Tumour.—But more important than all the above-mentioned symptoms is the occurrence of a *tumour* in the gastric region, which most frequently—in 50 to 60 per cent of the cases—is located in the pylorus, rarer—in about 10 per cent of the cases—in the cardia. The tumour may be seen in some cases, but usually it can only be felt; sometimes it does not become accessible to palpation until after lavage of the stomach. It is advisable in most cases to make the determining examination with the patient in chloroform narcosis. The tumour is hard, its surface mostly uneven, sharply to be defined. It is usually immovable, but quite an extraordinary movability is observed in some instances. It happened in one of my own cases that an apple-sized tumour could be displaced in the abdomen at will, to the left as far as below the left costal arch. Such a movability in cancer of the stomach seemed impossible to me, but an exploratory incision revealed a carcinoma of the pylorus which was successfully removed. Respiration usually exerts no influence upon the position of the tumour; but, on the other hand, I cannot admit, according to a vast experience, that a gastric tumour, contrary to other abdominal tumours, especially those of the liver and spleen, should not move downward on inspiration. *In fact, displacements of tumours of the stomach occur, even without the prevalence of coalescences of the tumour with the diaphragm, the liver, and the spleen.* Percussion does not give undoubted results either; the percussion sound is dull tympanitic, but the same sound is found in tumours which are located in the left lobe of the liver towards the border, and these are the ones which are to be considered in the differential diagnosis.

Absence of Free Hydrochloric Acid in the Stomach Contents.—An examination of the stomach contents will show, in by far the majority of cases, an *absence of free hydrochloric acid.* But this is not pathognomonic of carcinoma of the stomach, as was expected immediately after the discovery of von den Velden in 1879. For, on the one hand, the secretion of hydrochloric acid is reduced to a minimum in several other diseases of the stomach also, and eventually no free HCl can be demonstrated as in intense gastric catarrhs, in corrosions of the stomach, in atrophy of the mucous membrane, in amyloid degeneration of the gastric mucous membrane, and in nervous affections of the stomach; on the other hand, there are, which is an established fact to-day, cases of gastric carcinoma in which not only free hydrochloric acid can be demonstrated in the stomach, but even hyperchlorhydria. The question whether hydrochloric-acid reaction is absent, depends essentially upon the extension of the carcinomatous

infiltration and glandular atrophy; upon the simultaneous development of catarrh and atrophy of the mucous membrane connected with it; and finally, upon the progressive cachexia, all of which factors are followed by a decreased secretion of acid. Besides, lactic acid, butyric acid, acetic acid, in fact those chemical products of disintegration (referred to in chronic gastric catarrh) of the ingesta, which were retained in the stomach beyond the normal time, are demonstrable in the washed-out stomach contents or in the vomitus.

Lactic Acid in the Stomach Contents.—The demonstration of *abundant quantities of lactic acid* in the gastric contents is of particular importance for the diagnosis of carcinoma. The production of lactic acid of fermentation is connected in general (see p. 256) with the reduction of hydrochloric acid, with motor insufficiency and deficiency of absorption in the stomach, with factors, therefore, which, as is well known, prevail particularly in cancer of the stomach. Thus it is not astonishing that we observe in this condition a remarkably intense development of lactic acid, and *it is advisable, therefore, in cases in which no lactic-acid fermentation is demonstrable in the stomach, to be very careful with the diagnosis of a carcinoma.* Rare exceptions to this rule occur—i. e., cases in which the formation of lactic acid fails to take place in spite of absence or marked decrease of free hydrochloric acid and of stagnation of the stomach contents; besides, it should not be forgotten that large quantities of lactic acid are sometimes found in the stomach contents also in other affections of the stomach in which the secretion of gastric juice and the motor power of the stomach have become insufficient, as in atrophy of the mucous membrane with atony (see p. 272). But still, it will be best to adhere to the above diagnostic rule. Besides the reduction of hydrochloric-acid secretion and the occurrence of abundant lactic acid there occurs also, as may be expected, a deficiency of pepsin and lab-ferment in the stomach.

In keeping with the stagnation of the stomach contents usually associated with development of carcinoma, we furthermore find in almost all cases, upon lavage of the stomach seven hours after a test meal and upon lavage before breakfast, more or less abundant quantities of undigested food in the washed-out fluid. It is true, there are exceptions to this rule if the simultaneous gastric catarrh is slightly developed in carcinoma, or if the muscular force interferes compensatorily, causing the stomach contents to be passed into the intestine within the normal time, because the cancer is not located in the neighbourhood of the pylorus in these cases. In such an instance it may happen that the hydrochloric-acid reaction is absent and yet the duration of digestion is normal, as I was recently able to determine shortly before the death of patients at various times, in a most remarkable manner in a patient afflicted with carcinoma. Although, therefore, the importance of the absence of the hydrochloric-acid reaction and of the symptoms associated with it is limited in various directions, this much is certain, *that the presence of free hydrochloric acid in the stomach contents speaks in case of doubt, although not with absolute certainty, yet with the greatest probability against the presence of gastric cancer.*

The presence of carcinoma of the stomach can be demonstrated with

absolute certainty from an examination of the washed-out fluid or of the vomitus, if particles of the tumour are accidentally brought out. Unfortunately, this happens only in the rarest instances.

Little can be concluded from the symptoms of *perforations* which occur in various directions, except that if a pneumothorax or a pneumoperitonæum complicate the gastric affection, thus proving that an *air-containing* organ is perforated, or if—a rare occurrence—the perforation takes place to the external skin, rendering the carcinomatous proliferation visible externally. More important for the diagnosis in cases in which so far the diagnosis as to the character of the gastric affection was subject to doubt, is the demonstration of *metastasis in the liver*, which is most frequently (in about one third of the cases) affected by metastases in cancer of the stomach.

Differential Diagnosis as to the Origin of the Tumour.—It should not be believed that the above statements comprise everything that concerns the diagnosis of gastric carcinoma. Even the most expert diagnostician will always ask himself upon palpating the tumour in the gastric region whether it may not belong to another abdominal organ than the stomach. For, even in the almost complete presence of the above-named symptoms—cachexia, dyspepsia, coffee-ground vomit, gastrectasis, absence of free hydrochloric acid, besides the occurrence of large quantities of lactic acid in the stomach contents and palpable tumour—a simple chronic catarrh of the stomach may be present, *and the palpable tumour may not affect the stomach*. Under no circumstances can we do entirely without a differential diagnosis in this respect, and it is this part of the diagnosis of gastric carcinoma particularly which usually requires more care and deliberation than the demonstration of the other diagnostic points.

The first measure I usually employ to recognise whether the tumour belongs to the stomach, is (after evacuation, which should be as complete as possible, of the intestine, the same as in all examinations of abdominal tumours) the distention of the stomach with gas or the alternate filling and emptying of the stomach with fluid. The distention of the stomach by means of a Seidlitz powder will displace the boundaries of the stomach, and with it the tumour; then it is usually easy, inasmuch as the position and shape of the stomach become more distinctly visible by the distention, to recognise that the tumour belongs to the stomach. If the artificial distention causes the tumour to become inaccessible to palpation, to return after the escape of the gas, it points to the seat of the tumour being in the posterior wall or in the lesser curvature of the stomach (Riegel). Other points of support for the diagnosis of the tumour belonging to the stomach in a given case are obtained if the stomach is filled with water and emptied again by means of the tube, and if, then, the position of the tumour is compared with the boundaries of dulness of the filled and empty stomach. If the tumour remains within the boundaries of the artificially produced dulness, and if the latter disappears above and below the tumour after emptying the stomach, the diagnosis of gastric tumour may be made. If the tumour is situated in such a manner that this procedure does not accomplish anything—i. e., if the tumour is adjacent to liver, spleen, or transverse colon, the more frequent occurrence, the border of the liver

should be carefully followed by palpation and the contours of the latter in comparison to the tumour in question be determined.

Tumour of the Liver.—It is sometimes possible partly to grasp the upper point of the tumour, and thus to separate it from the liver by palpation as a tumour of the stomach. In other cases we may be able to demonstrate that the tumour is in part situated within the boundaries of the borders of the liver. Then it is a question either of a gastric tumour which has spread to the liver by continuity, or of a tumour of the liver that passes the boundaries of the border of this organ at a circumscribed area. The first of these two possibilities is always the more probable one, as it may be presumed that a neoplasm of the liver which passes the border of the liver downward, also diffuses laterally and causes the liver to appear enlarged and tuberous in its entire extent, whereas this is not the case in a gastric tumour which spreads to the liver, at least not at the onset. Minkowski has reported another means of recognising coalescence of a gastric tumour with the liver.

Respiratory Fixability of Gastric Tumours.—If a tumour of the stomach is held at the height of inspiration, the tumour might be prevented from ascending upon the following expiration, whereas this cannot be accomplished if the tumour is firmly grown together with the liver or belongs to the latter.

Carcinoma of the Gall-Bladder.—The latter rule applies also to *carcinomata of the gall-bladder*. According to their location they may be confused principally with carcinoma of the pylorus, but they are distinguished from the latter by the fact that (as they usually do not become associated as secondary cancers with gastric carcinoma) the signs of dyspepsia and the effects of cancer of the stomach are not present at all, which fact may be utilized for the diagnosis at least when, upon repeated examinations, no decrease of hydrochloric acid can be demonstrated, and if a secondary gastrectasis is absent which does not fail to appear in carcinoma of the pylorus. If carcinoma of the pylorus is not coalescent with the surrounding parts, it is distinguished, contrary to carcinoma of the gall-bladder, by its "expiratory fixability" and by its movability towards the side and particularly downward. It is true that exceptions to this rule in regard to relative difficulty of locomotion of gall-bladder carcinoma occur sometimes. Thus I was recently able to displace a gall-bladder which was filled with gall-stones and pus, beyond the median line, and erroneously, as later laparotomy taught me, I had in this case excluded tumour of the gall-bladder for this very reason.

Tumour of the Spleen.—It is less easy to confuse a carcinoma of the fundus of the stomach with *tumour of the spleen*. The possibility of spanning the upper border of the tumour in the hypochondrium—i. e., below the left costal arch—and the determination of normal percussion borders of the spleen, protect from wrong diagnoses, especially if an examination of the stomach contents for acidity shows a reduction of the latter. In fact, this chemico-diagnostic expedient is determining in all abdominal tumours which are still to be discussed, and which are to be considered in the differential diagnosis.

Carcinoma of the Pancreas.—Confusion with *carcinoma of the pancreas* is very apt to occur. The deep location and the immovability of the tumour, the supervention of symptoms of portal-vein stasis (see p. 223).

and the complication with intense jaundice speak in favour of carcinoma of the pancreas in comparison with pyloric carcinoma. It is possible with marked emaciation, especially of the abdominal walls, to palpate the healthy head of the pancreas, and, as happened to me once years ago, to confuse it with carcinoma of the pylorus.

Tumours of the Lymph Glands.—It is not very rare, according to my experience, to palpate at the vertebral column, *near the descending aorta, enlarged lymph glands* which may give the impression of gastric neoplasms, especially if the patient also complains of dyspeptic symptoms. Many years of observation of several cases having convinced me of the mostly harmless character of such glandular tumours, I do not attach as much importance any more to small, smooth nodes situated near the aorta as I did formerly. Such tumours disappear after artificial distention of the stomach.

Aneurysms.—It has also been determined that carcinoma of the stomach may enter into such close local relations with the aorta that eventually an *aneurysm* may be simulated, as general pulsation, systolic murmurs, changes of the crural pulse, etc., may occur in such a condition. A wrong diagnosis is best avoided by observation of the consistence of the tumour and, furthermore, of the fact that pulsation communicated to the gastric tumour from the aorta takes place principally only in *one* direction, viz., anteriorly, whereas pulsations of an aneurysm occur in all directions, especially also markedly transversely. [Expansile pulsation.]

Carcinoma of the Intestine—Carcinoma of the Transverse Colon.—Tumours of the *transverse colon* generally sink, because this portion of the intestine is supplied with a mesentery of its own, and therefore possesses a greater movability, and we usually obtain differentio-diagnostic information by experimental filling of stomach and colon with water or gas. Besides, the symptoms of intestinal stenosis caused by tumours of the colon, especially the distention of the ascending colon with fæces and gas behind the carcinomatous area of the transverse colon, supply important points of support which speak against gastric cancer. If an intestinal carcinoma becomes coalescent with the wall of the stomach, it will impair the movability of the intestinal tumour; it is also possible that a perforation of the intestine into the stomach occurs, and that the vomit or the washed-out gastric fluid contain fæcal masses, or, if a valve formation or a very small perforation prevent the entrance of fæces into the stomach, they may at least smell of fæces.

Duodenal Carcinoma.—It is scarcely ever possible to confuse a carcinoma in another portion of the intestine with gastric carcinoma, except *duodenal carcinoma* which, eventually, *cannot* at all be differentiated from carcinoma of the pylorus. It is true we might assume that the determination of free acid in the stomach contents will permit of a positive decision, as there is no reason for a deviation of the gastric-acid secretion from the normal in duodenal carcinoma. But in several cases (Riegel, Ewald) free hydrochloric acid has been *absent* in the gastric contents, either on account of a reflux of bile into the stomach caused by stenosis of the lumen of the intestine, or on account of a simultaneous extensive atrophic degeneration of the mucous membrane of the stomach. The eventual presence of jaundice is no certain symptom of a duodenal affection; the vomiting of coffee-ground-like masses and secondary gastrectasis are common to both diseases.

Tumours of the Omentum.—The greatest difficulties, finally, are presented (at least to me) by the differentiation of tumours of the omentum, respectively of the peritonæum, from gastric carcinoma. Of course, it is presumed in such a case that symptoms of disturbed gastric digestion are associated with the tumour and that the dimensions of the latter are not very large, passing beyond the boundaries of the stomach. The best protection against a wrong diagnosis is the immovability of peritoneal tumours on inspiration; they show at most an apparent locomotion, whereas genuine respiratory locomotion may eventually be determined in carcinoma of the stomach. Furthermore, ascites can be demonstrated as a result of peritoneal tumours, although not in every case; the nodes are usually not as circumscribed as gastric tumours, and of a secondary nature, so that primary neoplasms can be determined in other places. It is obvious that the above-mentioned factors are only relatively

certain characteristics; it is very gratifying, therefore, that the filling of the stomach with water or gas and the chemical examination of the stomach contents now furnish us means which, at least in most cases, aid us to overcome the difficulties of the differential diagnosis.

Nature of the Tumour.—After the stomach has been recognised in such a manner, with certainty or with probability, to be the organ in which the tumour is located, it is still a question whether the tumour actually is of a carcinomatous nature. The probability is in favour of carcinoma to begin with, as tumours of a different character which affect the stomach or the gastric region are very rare in comparison to carcinomatous tumours. Thus *benign hypertrophy of the muscularis at the pylorus* which, in my experience, is much rarer than is generally supposed. It is not possible to differentiate a tumour of this character from a smooth, small carcinoma of the pylorus. Common to both are consecutive gastrectasis and chronic gastritis; the result of the chemical examination, therefore, is often not determining; not even coffee-ground vomit, which may also occur in benign hypertrophy of the pylorus muscle if the blood, which originates from an unhealed ulcer, is detained for some time in a dilated, chronically inflamed stomach, and is decomposed. Only the *course* of the affection decides in such a case, above all the absence of carcinomatous cachexia, although enormous emaciation may sometimes also be seen in such a "benign" tumour due to gastrectasis, etc. In other cases, however, it is possible at once to make the diagnosis, if the tumour is very tuberous, if metastases in the liver can be determined, etc.

Abscesses of the Abdominal Wall.—It occurs comparatively seldom that an *abscess of the abdominal wall* is a source of error; it may happen if it takes a chronic course, if it has not yet become soft and if it is limited exactly to the gastric region. The marked bulging of the skin without a corresponding extension of the tumour to within, the ease with which the tumour may be grasped from the abdominal walls, the immovability of the skin which is usually coalescent with the tumour, as a rule secure the diagnosis of abscess of the abdominal wall, especially if *fever* is present at the same time, which, according to my experience (contrary to that of others), occurs only very exceptionally in gastric cancer unless special complications prevail. But, nevertheless, the differential diagnosis may eventually become very difficult if, for instance, a gastric ulcer perforates towards the abdominal wall and the pus gradually forces its way out, as I have seen in one of my cases, in which only the gradual pointing and softening of the tumour rendered a correct diagnosis feasible.

Gastric Sarcoma, Fibroids, etc.—Other tumours—fibroids, sarcomata, myomata, lymphadenomata, etc.—which occasionally occur in the gastric wall, offer only a pathologico-anatomical, not a clinical interest. They cannot be diagnosticated, not even when the conditions are very much in favour of a bold diagnosis varying from the usual diagnosis of carcinoma, thus in general sarcomatosis which also extends to the skin. In such a case I actually found also a sarcoma in the stomach; in another case, however, besides the sarcomatous tumours of the skin, a genuine epithelial carcinoma in the stomach! Nor does the diagnostic art embrace the determination of the carcinoma *ante mortem*, whether a fibrous, medullary, or colloid carcinoma be present in the given case.

But it is the duty of the physician to decide whether the location of the tumour in the stomach has caused certain subsequent symptoms depending upon the same—i. e., the consequences are to be determined especially which are due to formation of carcinoma at the entrance or at the exit of the stomach. The former have already been described in the discussion of stenosis of the œsophagus, respectively cardia, while the result of stenosis of the pylorus—gastrectasis—will be enlarged upon in the following chapter.

I wish to conclude the diagnosis of cancer of the stomach with a practical hint. Much as everything else in the diagnosis of gastric carcinoma is of minor importance to the diagnostician who wants to form a positive opinion, in comparison to the palpable tumour, practice teaches us that the tumour cannot, during the entire course of the disease, be palpated at all in a small number of cases (about 20 per cent), and in a great number it cannot be felt at the beginning at least. To exclude carcinoma because no tumour is felt would not do; in such cases it becomes necessary rather to assume the possibility of gastric cancer, *and this becomes very probable if the gastric affection in question occurs in an individual who has had a good stomach for fifty or sixty years, of which no care was taken, and which was able to digest everything.* If such a person becomes afflicted with a gastric affection, any other possibility is *a priori* less probable than the development of carcinoma of the stomach. An often repeated examination of the stomach contents for acid will very soon prove negative, a dietetic cure will be unsuccessful, cachexia will develop in a disproportionately marked and rapid manner, and thus the presumption of the pernicious character of the gastric affection will become more and more confirmed long before a tumour can be felt.

DILATATION OF THE STOMACH—GASTRECTASIS

Contrary to the gastric affections discussed so far, the *physical* examination is predominant in the diagnosis of gastrectasis. With a certain amount of experience on the part of the physician, gastrectasis is that disease of the stomach which is easiest to diagnosticate.

The symptoms are: dyspeptic manifestations, loss of appetite, eructations, *habitual vomiting of enormous masses* of food, which in part were partaken of a long time previous. The vomit contains all kinds of fungi: schizomycetes, sarcinæ, yeast fungi, etc.

Disturbances of Digestion.—*The chemical examination* of the vomit or of the washed-out stomach contents gives varying results, according to the cause of the formation of the ectasis: sometimes decreased acid secretion, in fact complete absence of the reaction of free hydrochloric acid, at other times normal or excessive quantities of acid. At the same time we find in the gastric contents the products of *processes of fermentation*, such as occur upon long-lasting stagnation of the stomach contents—lactic acid, butyric acid, acetic acid, and various gases.

Retention of the Stomach Contents.—The latter are either products of carbohydrate fermentation during which carbonic-acid gas and hydrogen develop and the gastric gases, if large amounts of hydrogen are present, may burn with a bluish or yellowish colour, or products of albumin putrefaction. The latter may cause the formation of hydrogen, and especially sulphuretted hydrogen. Both kinds of fermentation may be expected principally in stomach contents deficient in hydrochloric acid, but they also occur if it contains considerable amounts of HCl. As gas fermentation is never found in normal motor energy of the stomach, its demonstration may be considered as a sure proof of motor insufficiency.

The consequence of the latter is, furthermore, *that the ingesta remain too long in the stomach,* as is proved also by the experiment with a test meal, the stomach is mostly not emptied even overnight. In fact, sometimes we find after some time more fluid in the stomach than was introduced. This fact is comprehensible since the well-known experiments of von Mering, inasmuch as water is not only not absorbed by the stomach, upon impediment of its passage from the stomach, therefore remains in the same, but an eventual absorption of such substances as are resorbable in the stomach (sugar, dextrine, alcohol, peptone) is also associated with a secretion of water *into* the stomach. However, the resorptive activity of the wall of the stomach is generally decreased in cases of gastrectasis.

This disturbance of resorption and of the mechanical impairment of the passage of the ingesta into the intestine is also *connected,* besides, with retention of food in the stomach, *with the torpor of the stools, with scanty urine, dryness of the skin and desiccation of the nervous and muscular tissues,* to which latter condition it may be possible that the (although very rare, I have only seen one case) spasms (tetany) may be referred which have been observed in the course of gastrectasis. But they may also, as has become probable from recent experiences, be the symptoms of an autointoxication originating in the stomach. The deficient utilization of the food introduced into the stomach will, finally, cause gradually increasing marasmus, enormous emaciation, cold extremities, etc. Other morbid symptoms observed in gastrectasis—slowing of the pulse, dyspnœa, or even asthma and disturbance of the activity of the intestine—are too inconstant and, regarding their cause, too little depending upon gastrectasis to be of value in the diagnosis.

The most important part of the diagnosis of gastrectasis is the *physical examination of the dilated stomach* which we are now about to describe.

Inspection.—*Inspection* shows a *bulging of the abdominal walls in the height of the umbilicus and below, which downward shows the contours of the greater curvature.* If a marked downward displacement of the stomach exists, with a more vertical position of the longitudinal axis of the organ, the contours of the lesser curvature are also distinctly visible below the xiphoid process. Sometimes we see, similarly as in pronounced ileus, powerful peristaltic (rarely also antiperistaltic—i. e., those that progress from right to left) movements in the gastric region, which evidently are intended to overcome the obstacle.

Palpation.—It is sometimes possible to demarcate by *palpation* the boundaries of the enlarged organ from the other abdominal viscera, owing to its uniform elastic resistance. But this requires great practice, and is of little diagnostic significance. It is more important that palpation constantly causes the observation of a *splashing sound.* It is true the latter may be produced, even without gastrectasis, upon palpating the abdominal walls in the region of the stomach (and colon). However, the succussion sound which arises in the stomach will then be less constant and intense than in gastrectasis. It is also of importance to determine the *locality* of its origin. In gastrectasis the succussion sound is found still *below* the umbilicus and, in tracing from above downward, it can be

determined by delicate, careful palpation that it extends exactly to the contours of the greater curvature and ceases here, thus at the same time furnishing a means to determine the size of the stomach. Upon introduction of the stomach-tube we are surprised how far this tube goes down without stopping, and that lavage produces comparatively more fluid than was introduced with the first funnel.

Palpation of the Point of the Tube.—I have stated previously that, in dilatation of the stomach, the point of a hard tube is felt far down in the abdomen. For a number of years I have *not* employed this manœuvre any more in the diagnosis of gastrectasis, using now soft-rubber tubes exclusively; *it is true, the point of the elastic rubber tube is also sometimes distinctly felt through the abdominal walls,* but only when the latter are thin and very soft. Fortunately, we possess other very sure methods for the determination of gastrectasis, so that it is not necessary to go back to the employment of the hard tube in the interest of diagnosis. The most certain demonstration of gastrectasis is based upon the results of percussion.

Percussion.—*Percussion* of the gastric region serves to find the size of the stomach without a previous special filling of the organ. I percuss, to attain that object, with the patient in the erect posture, slightly outside of the left parasternal line, from the costal arch downward, until dulness appears. The latter, caused by the level of the fluid contained in the stomach, then disappears and is replaced by tympanitic sound if the patient assumes the recumbent posture. As the stomach, especially when dilated, almost always contains fluid, it is very rarely that this method of examination proves futile, and the result thus obtained may, at least, serve as a first guide. If the area of dulness (contrary to the condition in healthy individuals in whom the lower border of this dulness is about 3 to 7 cm. above the umbilicus) is found at the height of the umbilicus or more or less below this point, a dilatation of the stomach becomes *a priori* probable. Of course, a transverse colon that is filled with gas and fluid masses will give the same results on percussion. But the diagnostic doubts caused by this fact will disappear at once *if the stomach-tube is used to determine the dulness produced by the fluid in the stomach.* After the tube has been brought into the stomach, fluid is alternately introduced and withdrawn through the tube, and we are thus enabled to ascertain the size of the stomach with absolute certainty. Upon introduction of the fluid dulness appears, the upper and lower boundaries of which, changing with the quantity of the fluid introduced, can be determined; upon withdrawal of the fluid the dulness is replaced by a tympanitic sound. If the dulness of the fluid is below the umbilicus, gastrectasis is *sure* to be present. If we intend to demarcate from the colon sound the dulness or tympanitic resonance which is artificially produced at will in the stomach, it is necessary first to remove, by a purge, the fæces which are in the colon and which may eventually cause dulness of the sound, or to drive gas into the colon from the rectum, so as to cause the sound of the colon to be surely tympanitic, but this procedure usually gives unreliable results and is not at all necessary. Another means of demonstrating gastrectasis is the *artificial distention of the stomach with carbonic-acid gas or air.* This method does not give as reliable results regarding the boundaries of the stomach as does the

above-described combined percussion method, because, in my experience, it often fails, and furnishes at least doubtful results. But, on the other hand, it offers the advantage that it is the surest means of demonstrating the exact position of the upper curvature and the shape of the stomach, and it becomes possible thus to differentiate a dilated stomach from a vertically placed organ or from gastroptosis (see below).

Auscultation.—Auscultation does not yield any practical results, at any rate, in comparison to the results of the last-mentioned diagnostic measures, only very unreliable points of support for the diagnosis of gastrectasis. We hear, upon placing the ear upon the abdominal wall, the fluid which the patient drinks splash way down, and, upon marked development of carbonic acid, bubbling murmurs, etc.

A determination of the *capacity of the stomach* can be made in such a manner that we either note, in the above-described percussion method, the quantity of water which can be introduced into the stomach (a healthy stomach will hold about 1.5 litre), or that gas is blown into the previously emptied stomach until the patient has a sensation of tension. It is necessary that the gas which is blown in or withdrawn should be measurable by a certain contrivance.

Differential Diagnosis between Mechanical and Dynamic Gastrectasis.—After we have succeeded in the above manner in making the diagnosis of gastrectasis, it may still be specially the question to determine whether a coarsely *mechanical* occlusion of the pylorus by tumours, cicatrices, etc., be present, or whether a dilatation has developed in spite of the absence of such mechanical obstructions. The former condition is favoured by the strength of the visible peristaltic or antiperistaltic movements of the stomach, violent, lasting vomiting, and absence of bile in the washed-out fluid. Besides, it is advisable, if we are to decide differentio-diagnostically between the two above-named forms of gastrectasis, always to inquire carefully into the ætiological conditions of the given case and to consider them in the diagnosis.

The diagnostic characteristics mentioned so far refer to the diagnosis of *dilatation of the stomach which has become permanent to gastrectasis in its strictest sense.* The latter can, accordingly, be diagnosticated with ease and certainty, and it is really not worth while to enter upon the possibility of a confusion of gastrectasis with ascites, ovarian cysts, hydronephrosis, etc., as errors in this respect should not occur in cases in which a searching, careful examination is at all possible (compare chapter on Ascites). But it is more difficult to diagnosticate the first stages of a development of gastrectasis—the temporary distention of the organ.

Insufficiency of the Stomach—Gastric Atony.—According to the principles which apply to the origin of gastrectasis, the extensive explanation of which is out of place here, permanent gastrectasis (apart from those cases of very acute, sometimes even fatal gastrectasis which were recently observed in several instances) sets in quite *gradually*, when the disproportion between stomach contents and removal of the same has become *constant*, when the obstacle to the evacuation can no longer be *compensated* by increased muscular action and activity of resorption. It is obvious that these compensatory factors usually do not relax suddenly, but gradually—i. e., that periods exist during which relaxation occurs only upon greater demands upon the organ, while when the demands are less, the available energy suffices. In such cases we may speak of a *relative insufficiency of the stomach*, and we may surmise the same during examination of the patient if, after more abundant meals, the motor power of the stomach is not sufficient to remove the contents within the regular time,

and the lower boundary of the organ is lower than in healthy individuals, therefore at the height of the umbilicus, or below, whereas a dilatation of the stomach fails to appear with lesser demands upon the action of the stomach. If motor insufficiency is due to a congenital or acquired weakness of the musculature, we may also speak especially of an *atony* of the stomach (in contradistinction to cases in which a relative motor insufficiency becomes manifest with hypertrophic musculature of the stomach). Motor insufficiency may be associated with symptoms of gastric catarrh, or even often excessive acid secretion, which, again, may lead to spasm of the pylorus and may impede the removal of food from the stomach; this may eventually be followed by a dilatation of the stomach. *The abnormally marked dilatation of the gastric wall upon a certain degree of loading*, in comparison to the dilatation of the gastric wall in healthy individuals upon the same degree of loading, can at any time be demonstrated also by the fact that a certain amount of fluid is introduced into the empty stomach by means of the tube, and its action upon the dilatation of the wall of the stomach is ascertained by percussory determination of the lower and upper boundary of the fluid.

In such a manner it becomes feasible also to differentiate from gastrectasis an anomalous condition of the stomach which is usually confounded with it, or which is not diagnosticated because it does not cause any disturbances, viz., *megalogastria*. This condition, the "simply large stomach," may be congenital or acquired; it is distinguished from gastrectasis in the strictest sense essentially by the fact that it is, in that case, the question of a stomach *with abnormally large capacity but normal function*. Upon the introduction of fluid, whether large or small quantities are used in the diagnostic experiment, the lower boundary will always be low, whereas the examination of the motor power of the stomach shows normal conditions. *Megalogastria* differs from gastroptosis—i. e., from a descent of the stomach (see below)—by the fact that in the latter not only the lower, but also the upper boundary has descended uniformly, without the size of the organ exceeding normal proportions.

CHANGES OF FORM AND POSITION OF THE STOMACH

Malformations of the Shape of the Stomach—Hour-Glass Stomach.—*Of changes of form and size of the stomach*, besides those already mentioned, only the following are rarely and insignificantly considered diagnostically: *Malformations of the shape of the stomach* which are brought about by *tumours, adhesions* to the neighbourhood or in consequence of the formation of cicatrices in connection with ulcerations, etc. The latter may cause actual constrictions of the stomach, so that its lumen is divided into several sacs separated by the cicatricial constriction (*hour-glass formation*, etc.). These malformations are recognised best by inflation of the stomach with gas. It may also occur during lavage of the stomach that one sac, being separated from the other and communicating with the latter only by a more or less narrow opening, is emptied alone. If the washed-out water runs out clear, it may suddenly, especially upon a change of posture of the patient, become very cloudy, the adjacent sac emptying its contents into that one which is in connection with the tube.

Displacements of the stomach, also, are more interesting, than diagnostically practical, occurrences; thus the displacement of the stomach into the thoracic cavity in total or partial congenital defect or in rupture of the diaphragm, the distortion of the organ by *adhesions*, deposition of the stomach into large umbilical or scrotal herniæ, etc. The displacements of the organ caused thereby usually escape being diagnosticated until more marked disturbances of digestion supervene. If they occur, the state of affairs can easily be cleared by lavage undertaken for that purpose if percussion is carried out simultaneously—which procedure I advise in every case. Upon the flowing in of the water the expected dulness will not be present at the normal area; but further investigation will reveal, at other places of the abdomen, dull areas which become clear again after removal of the water. The diagnosis of displacement is complemented by palpation of the point of the tube (which can be distinctly felt at the place at which dulness appeared during lavage), but, above all, also by inflation of the stomach with gas. So-called *gastroptosis* is also demonstrated in this manner.

GASTROPTOSIS

This displacement of the stomach, which was first described by Glénard, may be accompanied with relaxation of the ligaments of the organ; the same as is also frequently observed, as is well known, in the intestine and in the various intestinal glands. If the relaxation of position caused thereby affects several abdominal organs simultaneously, we may speak of *general enteroptosis*. There can be no doubt, according to the observations which have recently been made by many, especially French [and American], physicians, that such cases of gastroptosis and enteroptosis actually occur. But it appears to me to be certain, especially in consideration of the so far meagre autopsy results, that simple gastroptosis, not associated with gastrectasis, does not at all occur as frequently as the discoverer of the affection and others believe. The most common cause of gastroptosis, which is incomparatively more frequent in women, is the unsuitable mode of dress (lacing and tight fastening of the skirts); the origin of the affection is also favoured by relaxation of the abdominal muscles, etc. Gastroptosis is often associated with dilatation of the stomach—i. e., a stomach dilated for some reason or other is, according to my experience, found almost always displaced *in toto*, so that the *greater and lesser curvatures* appear displaced downward. In most cases of this kind gastrectasis is the primary affection; sometimes it may become secondarily associated with gastroptosis, inasmuch as the latter may cause an obstructed passage of the food into the intestine and thus gradually lead to dilatation of the stomach. It is obvious that this descent of the stomach may be combined with all kinds of disagreeable sensations in the abdomen and with general nervous manifestations—disturbances of appetite, constipation, etc. One special form of downward displacement of the stomach may be mentioned in particular:

VERTICAL POSITION OF THE STOMACH

This condition is either congenital or acquired, especially by tight lacing, thereby forcing the pylorus downward and to the left. This causes the pyloric part of the greater curvature to advance downward below the height of the umbilicus, and thus gastrectasis may be simulated. It is true, it should be emphasized that such a vertical position of the stomach predisposes to the development of gastrectasis, and therefore is usually found in association with it, to which facts Kussmaul drew special attention. It is possible to *diagnosticate* gastroptosis in general, as well as the vertical position of the stomach in particular, only if we succeed, by inflating the stomach with gas, to render its contours accessible to inspection and percussion; upon the introduction of the water through the tube, the lower portion of the dulness thus resulting can be demonstrated as a comparatively *narrow* sac which, in the usual form of vertical position of the stomach, is situated *exclusively in the left half of the abdomen.*

NEUROSES OF THE STOMACH

Preliminary Remarks.—The domain of neuroses of the stomach has gained considerably in range during the last decade. If I compare what I gave in my text-book of over two decades ago as the foundation for the conception of neuroses, and especially the method which I then established for the critical examination of gastric diseases which could be ascribed to altered function of the nerves:

1. Increase or decrease of sensibility.
2. Increase or decrease of contractility.
3. Increase or decrease of intensity of secretion.

Pathological pictures have at present been found for all these dis-

turbances of function of the gastric nerves, whereas at that time I was forced to admit that we must forego a special demarcation of the clinical pictures of the various neuroses, in consideration of the then prevailing state of our knowledge. As it is obvious in a newly investigated branch of pathology, much of what has been found requires further confirmation, and the question is very much open to discussion whether it is practical and answers a clinical requirement to divide the nervous disturbances of the stomach into many separate pictures, as has recently become customary. However, the above scheme indicates the method how to investigate neuroses of the stomach; only, we must bear in mind that a disturbance of the function of the nerves in a certain direction does *not* usually *remain an isolated one*, but becomes manifest *in combination* with disturbances of another character. *We include* in the category *of neuroses of the stomach all those affections of the same in which the disturbances are essentially restricted to the nervous apparatus of the stomach*—i. e., *affections which refer especially to functional disturbances of the stomach, and in which nothing can be found anatomically that is contradictory to the assumption of an exclusive affection of the nervous system.*

Little being known until then except neuralgia of the stomach (gastralgia) and becoming convinced in the course of time that numerous affections of the stomach did not fit into those morbid pictures which are based upon the well-known anatomical changes, but, instead, should be conceived as nervous disturbances of the activity of the stomach, I was anxious to differentiate them, as they usually present themselves to the practitioner, diagnostically from the bulk of diseases of the stomach, and I selected for these morbid pictures the name "*nervous dyspepsia.*" It is my intention to discuss first this disease which, in my experience, is the most frequently occurring neurosis of the stomach.

I regret that the naming of the disease has caused so many discussions up to this day—why, I was never quite able to comprehend. I believe one principal reason for dissatisfaction with the name was due to the word "dyspepsia."

Determination of the Conception of Nervous Dyspepsia.—*I understand the word to mean difficult digestion, as implied by the name; but this embraces not only a disturbance of the chemistry of the stomach.* The symptoms of dyspepsia as they present themselves at the sick-bed becoming fruitful sources of complaints of the patient, are mostly of a *nervous* character, thus the alterations of appetite and taste, nausea, vomiting, increased salivation, mental depression, sensations of anxiety, headache, vertigo, the sensation of pressure in the gastric region, etc. The nerve tracts which transmit these symptoms *may become irritated by anatomical changes of the stomach and the chemistry altered thereby*, and they actually do so quite often. (In such cases they are just *symptoms* of gastritis, etc., the same as cardialgia is a symptom of ulcer.) However, the nerve tracts may also become pathologically affected with *anatomically normal* conditions of the organ, so soon as the respective nerve tracts are more irritable, either because, for some reason or other, a local irritability exists, and a general nervousness is gradually inaugurated from the nerves of the stomach, or that, *vice versa*—decidedly the more frequent occurrence—the latter (general nervousness) forms the principal object, and irritability of the gastric nerves only represents a prominent partial symptom of the same. I considered it necessary to premise this, as I trust, clear definition of my conception of "nervous dyspepsia," for which condition a better name cannot be found, in my opinion, before entering upon the discussion of the diagnosis of nervous dyspepsia.

NERVOUS DYSPEPSIA

Symptoms.—It is well known that the process of digestion causes, even in healthy individuals, an irritation of the nervous system; confused conditions of the head, fatigue, slight malaise, sensations of pressure and fulness are present, at least indicatively, in every person after meals. If these disagreeable nervous symptoms, which physiologically accompany the process of digestion, occur with unusual intensity and are supervened by further symptoms of dyspepsia—alterations of appetite and taste of a bizarre character, eructations, pyrosis, nausea, salivation, headache, vertigo, diffuse sensibility to pressure in the gastric region, etc., while the digestion usually takes its course in the regular time and with the correct final result—the presence of a *nervous dyspepsia* may be thought of. The probability that these symptoms of dyspepsia, of difficulty of digestion, are of a *nervous* character, sometimes becomes enhanced by certain secondary symptoms, for instance, that the patients, more stimulated by external impressions (during fascinating professional activity, in congenial society, etc.), do not feel their disagreeable sensations; that the pressure in the epigastrium is not constant; that eructations occur unusually often and without regard to surroundings; that vomiting, however, is generally rare, etc. At other times again, the patients are greatly tormented by their morbid symptoms; they concentrate their thoughts upon the affection, especially upon overexertion in business, do not sleep well, etc. It is characteristic that these factors, which refer to the nervous system, cause the morbid symptoms to become more prominent than errors of diet. Furthermore, other regions of the nervous system will occasionally show disturbances, which, however, compared to the dyspeptic symptoms, are of a minor or of an inconstant character. The same as most patients with gastric affections, individuals suffering from nervous dyspepsia are also troubled with constipation.

The diagnosis, or rather the presumption that this form of dyspepsia is of a nervous character, receives a firm support only by the *examination of the stomach with the stomach-tube.*

Results of Investigation as to the Time of Digestion.—*The digestive experiment with the test-meal proves the stomach to be empty after seven hours.*

Although it has been emphasized by several authors that this is *not always* correct if the dyspepsia is of a pronouncedly nervous character, I must insist that this simple experiment should be resorted to in every instance. The occurrence of exceptions—i. e., that the washed-out fluid may sometimes contain some undigested remnants of food—is just as likely to be possible as an occasional consummation of digestion within the normal time by an organ with anatomically palpable affections of the stomach and retardation of the time of digestion. But to reject, for such a reason, the digestion test as to the time of stomach digestion, is, according to my experience, to deprive ourselves of the most important objective, and at that practically most convenient criterion of the nervous character of the dyspepsia. Rare exceptions do not disprove rules which are valid in by far the majority of cases.

However, we must not restrict ourselves to this test of the digestive activity of the stomach, which should always at first be resorted to, but *a*

chemical examination of the stomach contents should follow under all circumstances.

This will give varying results: *Normal* acidity, but also *excessive* production of acid, of great *reduction* of the acid secretion, or almost total inacidity, according to the prevalence of irritative or depressive conditions of the secretory nerves. There exist examples of all these forms of nervous dyspepsia. I submit case histories of each of these three types:

Case I. Nervous Dyspepsia with Normal Secretion of Acid.—A *savant*, thirty-six years of age, formerly quite healthy; since his twentieth year he has been suffering from gastric troubles, the same as now—pyrosis, sensation of pressure and inflation in the gastric region, bad taste in the mouth, eructations of gas, which can be heard at a great distance and which continually occur without restraint during conversation. When the stomach becomes empty, or is entirely so, stomach pains set in, occasionally vertigo, headache, especially pressure in the scalp, etc., constipation, with a dejected mental condition.

Examination of the stomach shows no dilatation, no tumour, no pain on palpation, nor can the gastric region be designated as distended.

Repeated tests with the tube as to time of digestion, both after test-meals and after very opulent meals, always show the stomach to be *empty*. *Determination of acid*: 0.15 per cent HCl.

Case II. Nervous Dyspepsia with Excessive Production of Acid.—A banker, thirty-four years old, suffered from gastric troubles for two years, commencing with painful pressure in the pit of the stomach. The pressure commenced almost regularly at 11 A. M. and at 4.30 P. M., and was independent of the posture of the patient; *it subsided when the patient took a few drops of cold water, and it occurred immediately after mental emotions.* Besides, *loss of appetite*, bad taste in the mouth, nausea, sometimes vomiting during the daytime, as well as at night. Frequent headache, palpitation of the heart, much eructation of gas, obstipation.

Examination of the stomach shows *normal boundaries* of the same, *but painful upon pressure.*

A test-meal was completely digested after seven hours. Determination of acid shows 0.39 per cent HCl.

Case III. Nervous Dyspepsia with Subacidity respectively Anacidity.—Merchant, fifty-two years old, suffering from stomach troubles for seven years; his affection consisted in formication and burning in the gastric region. Bitter, salty taste in the mouth, much pyrosis, frequent eructations of gas (sometimes after food), and disagreeable sensations of taste (sulphur taste); no painful stomach pressure; *good appetite*; severe headache; no palpitation of the heart; flatulence and borborygmus; consistence of the stools changing between constipation and diarrhœa.

Examination of the stomach shows: Normal boundaries of the stomach, no sensibility of the stomach to pressure. Test-meal as well as more opulent meals always thoroughly digested after seven hours. *Acidity*: 0.06 per cent HCl.

Normal Time of Digestion with Normal, Increased, or Decreased Production of Acid.—*The termination of the time of digestion is normal*, although not without exceptions, yet in the greatest majority of cases, the same as in the above case histories. This is obviously as it should be in cases with normal secretion of juice, and it becomes especially prominent here that, in spite of undisturbed chemism of the stomach, the process of digestion causes trouble only because the nervous system, which is in a state of great irritability, reacts more markedly—i. e., *pathologically*—to the same. But even in cases of *excessive formation of acid* the duration of gastric digestion which takes place under difficulties, is not longer than normal, often accelerated, it is true, but rarely delayed (delayed possibly

when hyperchlorhydria causes a temporary spasm of the pylorus, thus favouring a longer retention of the food). But in the same manner it is possible that a nervous dyspepsia with *subacidity or inacidity* may give a normal result upon examination of the stomach with the tube regarding the condition of the motor activity. We should surmise that deficient acid production would lead to retarded digestion. However, experience teaches that this theoretical presumption is usually incorrect in nervous dyspepsia with deficient production of acid. I am in possession of numerous case histories of marked nervous dyspepsia with great reduction of acid production, in which the stomach, nevertheless, finished the expulsion of the food within the regular time. The wall of the organ being anatomically intact in nervous dyspepsia, it is not astonishing that compensatory factors, especially increased muscular activity, may effectually interfere in such cases of nervous subacidity.

It is a main rule in the diagnosis of nervous dyspepsia not to make it until the other gastric affections which are associated with increase or decrease of acid secretions (ulcer, carcinoma, atrophy of the mucous membrane, etc.), have been excluded. If, after mature deliberation, we reach the conclusion that the affection actually is a nervous dyspepsia, this diagnosis should be supplemented by: "with hyperchlorhydria or with subacidity," leaving it to the diagnostician eventually to reverse the acid alterations, if he considers them the main object, and the nervous-dyspeptic symptoms as essentially depending upon them, and also the name of such pathologically nervous condition, and to speak of hyperchlorhydria with nervous dyspepsia. The choice of a name, it seems to me, is of minor importance in such a case—*denominatio fiat a potiori!*

Ætiological Diagnosis of Nervous Dyspepsia.—The diagnosis of this condition is materially supplemented by an observation of the ætiology, which forms a most important factor in every single instance, more so because our therapy usually takes its direction accordingly. At first we must determine whether the nervous dyspepsia is a more independent affection in the given case or a partial symptom of a general nervousness, of a "neurasthenia." Furthermore, the *urine* should be examined, as, in my opinion, dyspeptic symptoms of contracted kidneys are mostly of a *nervous* character, the expression of insidious uræmic intoxications of an inferior degree; also the *spleen*, as I have surely observed that nervous dyspepsia may mask malaria. In other cases, again, chlorosis or hysteria is present. In such cases in which an infectious or constitutional disease exists, nervous dyspepsia is the expression of the effect of those diseases upon the nervous system in general. But this symptomatic nervous dyspepsia may eventually be so prominent, it may have assumed such clinical independence that (although based upon those affections) it must be diagnosticated as such. Gastric catarrhs and gastric ulcers seem to be other sources of nervous dyspepsia, in so far as they leave, *after recovery from them*, a long-lasting condition of great weakness and irritability of the gastric nerves; in other cases, again, the irritation of the gastric nerves radiates from the genitalia, thus in women with uterine diseases, etc. The period of development of the sexual sense may also, as I have observed in several very remarkable cases, cause as the principal symptom a high-

graded nervous dyspepsia which, after puberty has been reached, heals spontaneously after having resisted all cures until then.

Differentiation of Nervous Dyspepsia from other Gastric Neuroses.—As to the question into which category of our scheme, as given in the introduction to the neuroses, nervous dyspepsia is to be classified, there can be no doubt that it is principally a *sensory neurosis of the stomach.* However, the restriction of the symptoms of nervous dyspepsia to the sensory sphere (as in other neuroses) is *not absolutely strict*; this is seen, on the one hand, from the fluctuations of gastric juice secretion, from the secretory sphere therefore, and, on the other hand, from the eructations, the peristaltic unrest, the retching—i. e., from motor-irritative symptoms of various kinds; therefore, we have to do with a *"combined" neurosis of the stomach. It is determining for the diagnosis that the nervous manifestations refer to the process of digestion and never become prominent in the pathological picture as excessively developed in one direction.*

If the latter is the case, other morbid types will be produced, viz., upon excessive irritation of the sensory nerves, especially *gastralgia*; upon irritation of the secretory function, *gastrosuccorrhœa* and others; upon greater irritation of the motor function, *spasm of the cardia*, of the *pylorus*, or of the *entire stomach*, etc., the latter usually occurring in association with gastralgia. This irritation of the nervous system affecting *simultaneously* the sensory and motor spheres, is still more pronounced in *nervous vomiting.*

Our knowledge regarding neuroses of the stomach which are based upon *a lessening of the sensory, motor, and secretory activity of the gastric nerves*, is rather inadequate for the time being, so that a strict diagnosis of the same cannot be thought of as yet.

To begin with the first group, we shall discuss the various

SENSORY NEUROSES

This chapter is generally considered to embrace, as types of the *irritative* condition of the gastric nerves, *hyperæsthesia of the mucous membrane of the stomach, gastralgia*, and *hyperorexia*; as types of *depressive* conduct, *anorexia* and *acoria*. Clinically the longest- and best-known of the sensory neuroses is

GASTRALGIA, GASTRODYNIA, SPASM OF THE STOMACH

Gastralgia.—The pathological picture of this affection is very characteristic: *In paroxysms* there occur violent stirring, boring, *spasmodic pains in the pit of the stomach* radiating from the xiphoid process to the back, which may increase to the highest degree of painful sensation, associated with a feeling of annihilation, with attacks of fainting and symptoms of collapse. Their onset is without premonition, sudden, or they are ushered in by pressure in the epigastrium, toothache, salivation, globus hystericus, etc.; at times they are accompanied with globus hystericus, bulimia, strangury, and vomiting. Often, during the height of the attack, the patients press their fists into the epigastrium, or they may put

their stomach firmly against some solid object. The attack disappears, after having lasted for minutes or hours, sometimes with a gradual diminution of the pains, at other times with eructation, vomiting, etc. An abundant, bright urine (*urina spastica*) is occasionally voided after the attack; during the interval between two attacks the patient feels perfectly well in pure neuralgias. The frequency of the attacks varies considerably, sometimes several attacks occur in one day, at other times months will pass by before a recurrence takes place. The diagnosis, therefore, of pronounced gastralgia is easy; but, nevertheless, confusion with other abdominal affections which are accompanied with paroxysms of pain, is possible. A differential diagnosis by exclusion, therefore, is the principal object in the diagnosis of gastralgia.

Differential Diagnosis.—Intercostal neuralgias, which are located in the *lower* intercostal nerves, may simulate gastralgia, because the pains in such intercostal neuralgias are restricted to the epigastrium and, *vice versa*, in gastralgia the pains radiate to the intercostal nerves. If, now, upon proper examination, the pain is found to be concentrated upon the abdominal walls and the painful areas in an intercostal space, the diagnosis is clear, especially if an exploratory electrization of the respective intercostal space moderates or removes the pain in the epigastrium.

Gastric Ulcer.—The differentiation of gastralgia from *gastric ulcer* is more difficult, if cardialgias prevail among the symptoms of the latter. In favour of gastralgia are, in this instance, the long duration of the interval between two paroxysms, marked independence of the attacks of pain from the ingestion of food (so that often food extremely difficult to digest can be partaken of without disturbance, whereas, it is true, at other times hot or cold dishes, sharp-edged ingesta, etc., may cause a gastralgic attack). In favour of gastralgia are also the simultaneous presence of tabes, hysteria, and other nervous affections which may give rise to gastralgia, the disappearance of the pain upon external pressure or upon application of the electric current during the process of digestion.

Gall-Stone Colic.—It is very easy to confuse gastralgias with *gall-stone colics*, especially as the latter may become associated with a sympathetic gastralgia. Painfulness and swelling of the liver, jaundice, dilatation of the gall-bladder are decidedly in favour of cholelithiasis; however, the last-named symptoms are more often absent than present in gall-stone colic.

Intestinal Colic.—A confusion with *intestinal colic* is not very apt to occur. The facts that the pains in intestinal colic may really have their seat in the epigastrium, but usually change their location, are accompanied with local distention of the intestines and often suddenly cease with discharge of fæces or flatus, protect from a wrong diagnosis.

Ætiological Diagnosis.—We should, under no circumstances, be satisfied with the diagnosis of gastralgia until the above-named pathological conditions have been excluded and at least an attempt has been made also to determine the *cause of gastralgia* in the given case. The irritations are abnormal which affect the nerves of the stomach either peripherally (tumours compressing the pneumogastric nerve), or centrally (in this respect the *crises gastriques* in tabes may be mentioned which some-

times occur as an initial symptom); furthermore, abnormal conditions of nutrition and reaction of the nerves of the stomach which may serve as a foundation for gastralgia, thus in hysteria, neurasthenia, chlorosis, arthritis, etc., or, possibly, infectious diseases (malaria, chronic articular rheumatism). Finally, it happens in a number of gastralgias that the affection is brought about by "reflex" irritation or radiation from another diseased organ, viz., nose, uterus, etc.

HYPERÆSTHESIA OF THE MUCOUS MEMBRANE OF THE STOMACH

This affection, caused by a morbidly increased irritability of the sensory gastric nerves, manifests itself by pressure and fulness in the epigastrium, burning and shooting pains in the stomach, eructations, bulimia, nausea, and vomiting. I count these cases, unless they represent mild degrees of gastralgia, among the *nervous dyspepsias*, especially as the sensibility of the stomach becomes most prominent particularly during the process of digestion, and because, in this case as in the other, besides hyperæsthesia, alterations of secretion and motor irritative symptoms, such as vomiting, are also superadded; other nervous and hysterical symptoms combine with the morbid picture as secondary symptoms, the same as is the case in nervous dyspepsia.

ANOMALIES OF THE SENSATIONS OF HUNGER AND SATIATION

Anomalies of the sensations of hunger and satiation, hyperorexia (bulimia), anorexia, and acoria (loss of satiation) are also to be considered as disturbances in the sensory sphere.

Bulimia (*insatiable hunger*), suddenly occurring both on an empty as on a full stomach, may give rise to fainting, sensation of anxiety, headache, palpitation of the heart, etc., unless the desire for food is at once satisfied. This condition can scarcely be confused with anything else, at most with polyphagia, but it is easily differentiated from the latter in so far as in the latter the sensation of hunger occurs only between meals after the ingesta have been more or less completely digested. Of course, to diagnosticate bulimia as an independent neurosis, it must be excluded that the insatiable hunger is, in the given case, only the symptom of an anatomically demonstrable, gastric disease and as such becomes predominating.

Anorexia, Acoria.—As *depressive* forms of neuroses of the sensory apparatus of the stomach, we shall briefly mention *nervous anorexia* and *acoria*.

Anorexia nervosa, designating a reduction or complete cessation of the appetite, is brought about in a strictly nervous manner without any organic change of the stomach. The diagnosis of nervous anorexia should be made only after careful consideration whether every anatomical change of the stomach which may cause a reduction of appetite can be excluded, and with proper regard of the remaining conditions of the patient in the given case. The demonstration of a synchronous chlorosis, hysteria, or psychosis serves especially to facilitate the diagnosis.

Acoria, the loss of the sensation of satiation of the appetite, manifests itself in so far as the patients, even after an abundant meal, have no longer the sensation as though enough food has been partaken of. The hunger in its pure forms is, at the same time, by no means increased, and thus it is easily possible to distinguish acoria from hyperorexia and polyphagia, with which it is otherwise very apt to be confused, (because an abnormal amount of food is partaken of in all these morbid conditions). Acoria is, according to the assumption of Fleischer, probably only the expression of a simple *anæsthesia* of the *sensory nerves of the stomach*, which causes the sensation of fulness, which, after plentiful ingestion of food, gives the impression of satiation, and does not become perceptible in this case. If this conception of the character of acoria is correct, and I have no doubt it is, then patients with acoria should not, as Fleischer correctly emphasizes, have the sensation of gastric pressure upon marked artificial extension of the stomach with gas or fluids, as is the case in healthy individuals.

NEUROSES OF SECRETION

As previously stated, the secretion of the gastric juice depends directly upon the nervous system. It is therefore *a priori* probable that alterations of the secretion of gastric juice occur as independent neuroses. Clinical observation teaches, indeed, that cases of disturbances of gastric-juice secretion exist which can only be explained as *neuroses* of secretion, and we see them occur either as an increase or as decrease of the intensity of secretion. Therefore, we distinguish at present two forms of neuroses of secretion: nervous *hypersecretion* (*gastrosuccorrhœa*) and nervous *hypogastrica or achylia gastrica.*

NERVOUS HYPERCHLORHYDRIA; NERVOUS GASTROSUCCORRHŒA (SUPERSECRETION OF GASTRIC JUICE)

We are indebted principally to Reichmann, Rossbach, Sahli, and Riegel for the knowledge of these anomalies of secretion. *According to the occurrence of an increase of secretion in consequence of digestive irritations, thus causing the secretion of an excessively abundant*—i. e., *too abundant in comparison to the irritation, especially excessively acid—juice, or according to the occurrence, in other cases, of a secretion of quantities of juice which are abnormally abundant even outside of the time of digestion*—i. e., *without digestive irritation on an empty stomach*[1]*—we speak of "hyperchlorhydria" on the one hand, and of "supersecretion" on the other.* I do not consider these designations as well selected, as it is not certain whether in hyperchlorhydria exclusively *only* hydrochloric acid is secreted, or whether, besides, ferments may not also be secreted in increased quantities, owing to the abnormally marked activity of the gland cells. However, it is not my intention to discredit the long-adopted name of *hyperchlorhydria*—the excessive secretion of hydrochloric acid is, undoubtedly, the main object in the respective cases—and I shall therefore adhere to the designation hyperchlorhydria in the following; whereas I shall not use the name "supersecretion" to avoid misunderstandings, but I shall only speak of gastrosuccorrhœa or excessive secretion of gastric juice.

NERVOUS HYPERCHLORHYDRIA

We have repeatedly observed that *hyperchlorhydria*—i. e., *excessive secretion of gastric juice, especially hydrochloric acid, following digestive irritations*—is found in various affections of the stomach, especially in gastric ulcer. This symptomatic hyperchlorhydria is distinguished from the *nervous* form in so far as, in the latter, all organic affection of the stomach is absent and that the entire pathological picture is in favour of the nervous character of the disease. Of course, the most important diagnostic factor is the demonstration of an excessive secretion of acid subsequent upon a test-meal or test-breakfast.

In other respects the affection manifests itself in disturbances which occur a short while after a meal, and which consist in pressure and fulness in the epigastrium (sometimes in actual spasmodic pains), in acid eructa-

[1] Moderate amounts of gastric juice containing HCl are often also found, as was first demonstrated by Schreiber, in the stomach of healthy individuals; however, in such cases it is only a question of small quantities of gastric juice, whereas in pathological succorrhœa often 400 and more cubic centimetres are found in the empty stomach.

tions, pyrosis, and bulimia. The vomiting of intensely acid masses is rarely brought about, upon the occurrence of which the disturbances vanish. The latter are therefore absent during the time when no food is to be digested, and that usually at night. The gastric region is diffusedly sensitive to pressure during the paroxysms of pain, but not so at other times. The cause of these disturbances is the secretion of excessively abundant quantities of hydrochloric acid which, at first, is in combination and only later, when it remains uncombined in larger quantities—i. e., "free"—causes pathological symptoms. Thus it is comprehensible that the latter never occur at once, but only sometime after meals, that, sometimes, "difficult" meals are digested without disturbances, and that the pains at the onset of their occurrence can often be aborted by the administration of milk, etc., or of sodium bicarbonate, which substances cause the free hydrochloric acid to combine. The appetite is usually good, thirst is increased during the time in which an excessive amount of HCl may be supposed to be present in the stomach, and the acidity of urine is decreased during this period. The diagnostic examination of the time of digestion shows, almost without exception, a rapid course of digestion, that of the stomach contents an acidity of 70 to 80 and more; the albuminous substances appeared to be well digested, amylaceous substances, if introduced in larger quantities, poorly, because the action of ptyalin is impeded by the free hydrochloric acid.

To diagnosticate hyperchlorhydria in the given case as a *neurosis of secretion*, it is necessary first of all to be able to exclude every anatomically demonstrable gastric affection as a cause of excessive acid secretion. Furthermore, attention should be paid to the connection of hyperchlorhydria with mental emotions, mental overexertions, hysteria, etc., and, above all, it should be noted whether frequent changes in the condition of the patient occur in the nervous form of hyperchlorhydria—i. e., whether the patient often remains without any disturbance for days and weeks, eventually again to become ill in the above-described manner, due to psychical effects. This paroxysm-like occurrence is also characteristic in the intermittent form of gastrosuccorrhœa which we shall discuss in the following chapter.

NERVOUS GASTROSUCCORRHŒA

Gastrosuccorrhœa is differentiated from hyperchlorhydria essentially by the fact that, in the former affection, the gastric mucous membrane secretes large quantities of gastric juice, even without digestive irritation, the stomach, therefore, contains abundant quantities of gastric juice even when empty. The affection occurs in two forms, as *intermittent* and as *continuous gastrosuccorrhœa.*

INTERMITTENT GASTROSUCCORRHŒA, PERIODICAL EXCESSIVE SECRETION OF GASTRIC JUICE

In this condition it is a question of a *paroxysmally* occurring disturbance of the secretion, with intervening, longer pauses, during which the patients, at least usually, are perfectly well. The paroxysm commences with pyrosis and spasmodic pains in the stomach, and terminates in re-

peated vomiting of an abundant, greenish-yellow fluid which contains hydrochloric acid (often hypernormal quantities) and ferments with epithelia and some bile. During the attack the appetite is lacking entirely, the pulse is small, the patient is very liable to fall into collapse, urine is scanty, of a neutral or often alkaline reaction. An inconstant secondary symptom is headache which, however, becomes very prominent in some cases (such cases were once designated "gastroxynsis" by Rossbach). The intensity and duration of the attack vary considerably; sometimes it lasts only a short while, at other times it persists for days.

There is scarcely a difference of opinion as to the *nervous* character of the intermittent form of gastrosuccorrhœa. We note the occurrence of the morbid condition principally in irritable individuals, after mental overexertions, severe anger, etc., also in tabes and other diseases of the spinal cord. The diagnosis itself does not present any difficulties if we adhere to the description of the pathological picture, if vomiting occurs long after the ingesta have left the stomach, and if the vomited fluid proves to be gastric juice by the presence of hydrochloric acid and pepsin.

CONTINUOUS GASTROSUCCORRHŒA—CHRONIC EXCESSIVE SECRETION OF GASTRIC JUICE—CONTINUOUS SECRETION OF JUICE

The conception of continuous gastrosuccorrhœa as a neurosis of secretion is justified in a very few cases only. The secretion of gastric juice is more *continuous* in this affection, the symptom-picture is that of chronic dyspepsia, manifesting itself in sensation of pressure in the stomach region, acid eructations, bulimia, pains (especially at night) and vomiting; the disturbances are, in general, like those of hyperchlorhydria. But to this is added that the pains occur not only a few hours *after* meals, but also *before* eating; that vomiting takes place *also when the stomach is empty,* and large quantities of a slightly cloudy fluid are ejected which proves to be gastric juice and which eventually contains gases and undigested amylaceous substances. The thirst is increased in many cases, also the appetite, which is particularly directed to food rich in albumin; the urine is scanty and, in keeping with the profuse secretion of acid gastric juice and with the vomiting of acid masses, deficient in acid. The nutrition of the patient suffers considerably upon long duration of the disease.

We have already stated that gastrectasis may develop from continuous gastrosuccorrhœa. It is especially the merit of Riegel to have drawn attention to the coincidence of continuous secretion of gastric juice and gastrectasis, and to have defined the mutual relations of both morbid conditions. As continuous gastrosuccorrhœa has been observed without dilatation of the stomach and, on the other hand, gastrosuccorrhœa is absent in most cases of gastrectasis, and as, furthermore, cases of continuous secretion of gastric juice are observed, it may be assumed that, in the combination of gastrectasis and continuous gastrosuccorrhœa, the latter is the primary, and gastrectasis the secondary condition. How the latter condition develops as the subsequent condition can probably be explained in the following manner: The acid condition of the gastric juice impedes amylolysis

and to this is eventually superadded, as another factor, a spasm of the pylorus, owing to increased acid irritation, or an anatomically demonstrable stenosis of the pylorus (especially in consequence of an ulcer cicatrix), all being factors which retard the emptying of the stomach. In other cases, in which a stenosis of the pylorus is the primary condition and to which, as has been proved, a continuous gastrosuccorrhœa has associated itself secondarily, it may be presumed, from a hyperchlorhydria which existed originally beside gastrectasis, that a more pronounced condition of irritation of the gastric-juice-secreting glands developed in the course of the affection and, with it, a continuous gastrosuccorrhœa. It is conceivable from the above remarks that cases may exist of gastrectasis with hydrochlorhydria *without* continuous secretion of gastric juice, and also cases with the latter condition.

These variations in the combination of gastrectasis with hyperchlorhydria and continuous gastrosuccorrhœa, as well as the presence of the latter in general, can only be determined by lavage of the stomach; which should be done in quite a distinct order, first defined in principle by Reichmann, the discoverer of gastrosuccorrhœa. At first the stomach is washed out seven hours after a test-meal, and it should be determined whether the stomach still harbours, at this late period of the digestive process, profuse quantities of finely distributed remnants of amylaceous substances, but no fluid containing any, or at most but very few, isolated remnants of meat and gases, also whether this fluid shows great acidity, especially abundant hydrochloric acid. The first lavage is to be followed by a second one of the *empty* stomach, to determine whether a very marked motor insufficiency is present or not—i. e., whether the stomach still contains remnants of food from the previous day or whether it is empty. In the former case we must continue the procedure in such a manner that the stomach of the patient is washed clean at night, and this continued until the washed-out water is clear and has a neutral reaction, and care is to be taken that no water or as little as possible remains in the stomach at the end of the lavage. The following morning (until which time the patient should not have partaken of anything) the stomach contents should be expressed. If, then, more or less profuse quantities of the above-described fluid with the properties of the gastric juice, especially with distinct reaction to free hydrochloric acid, are found, the diagnosis of continuous gastrosuccorrhœa may now be made with certainty.

DEPRESSIVE CONDITION OF THE SECRETORY NERVES OF THE STOMACH—NERVOUS SUBACIDITY—ACHYLIA GASTRICA NERVOSA

The decrease or suspension of secretion of gastric juice is a symptom which occurs in many affections of the stomach, and which is caused by certain organic diseases of the stomach (carcinoma, gastritis, atrophy of the mucous membrane). The same as in hyperchlorhydria, we observe also in subacidity, respectively achylia gastrica, cases in which this depression of the function of the gastric-juice-secreting glands is not a symptom of certain gastric diseases, but a purely nervous affection. The symptom-complex

of achylia has been previously (p. 271) described, and the differentiation of the nervous variety from that caused by atrophy of the mucous membrane has been fully discussed. I only repeat briefly that the hydrochloric acid in the washed-out gastric juice has been found markedly reduced or is entirely absent, that, in spite of the want of the gastric-juice secretion, the motor power of the stomach may interfere compensatorily and the stomach contents may be passed into the intestine within the regular time, whereas, if the latter is not the case, severe disturbances of digestion, abnormal fermentations, etc., may develop. In favour of the diagnosis of *nervous* achylia are: The absence of the anamnetic demonstration of an affection of the stomach which is acknowledged to lead to atrophy of the mucous membrane (severe gastritis, carcinoma, etc., see above p. 272), and, in a positive sense, that exquisitely hysterical and neurasthenic affections manifest themselves in the patient, besides achylia. To the nervous character of achylia would point, above all, if a certain change exists in the production of gastric juice—i. e., if, upon repeated examination of the gastric function, at one time no digestive juice, at another time hydrochloric acids and ferments can be demonstrated in the stomach contents, and if the reduction of gastric-juice production coincides regularly with psychical emotions, etc., or if an affection of the central nervous system (as in one of my cases, tabes) causes paroxysmal vomiting, and this, occurring one hour after breakfast, does not show any trace of free hydrochloric acid.

MOTOR NEUROSES

These neuroses of the stomach are of less importance in a clinico-practical respect.

IRRITATIVE CONDITIONS OF THE GASTRIC MOTOR NERVES

Very rare and very little known is the excessive motor action which affects the entire stomach—viz.:

HYPERKINESIS, PERISTALTIC UNREST OF THE STOMACH

It is doubtful whether a *primary* nervous increase of the motor action of the stomach occurs. In by far the majority of cases in which the diagnostic sounding of the stomach demonstrates a too rapid expulsion of the stomach contents, it is a question of a hyperchlorhydria or, as we have just seen, of an achylia, both of which may be followed by a hyperkinetic action of the stomach. Much rather a primary neurosis of motility should be thought of in those cases which, under the designation "peristaltic unrest of the stomach" were first described by Kussmaul. In this condition extremely lively, rapidly repeated muscle contractures occur in the stomach which are particularly intense after meals, but which may also take place *when the stomach is empty*. The latter circumstance distinguishes these peristaltic movements, which are due to an abnormal irritability of the motor nerves of the stomach, also from the well-known peristaltic movements which can, in stenosis of the pylorus, so often be observed as powerful

bulgings and retractions of the stomach or as undulating motions progressing from left to right, and which are the expression of muscular contractures the object of which is to overcome the mechanical obstacle (see p. 288). A peristaltic unrest occurring without stenosis of the pylorus as a neurosis of motility, has not been noted as yet in a normally located stomach, but it has occurred in a displaced or dilated stomach with thin abdominal walls. Otherwise, to make the diagnosis, we must rely on the statements of the patients that they suffer from a troublesome sensation of unrest, of swaying to and fro, rumbling, etc., in the stomach. If the peristaltic unrest is to be explained as the expression of a primary, independent neurosis of motility, it is necessary that stenosis of the pylorus and hyperchlorhydria can be excluded as causes of these increased peristaltic movements, as well as a *peristaltic unrest of the intestine.* They cannot be confused, upon closer observation, with peristaltic unrest of the stomach, if the boundaries of the stomach are noted, and if we determine whether the peristaltic movements are limited strictly to the stomach or are noticeable beyond the boundaries of this organ. This is best accomplished by artificial inflation of the stomach, which also serves to decide whether this peristalsis does not occur in *abnormally* situated coils of the small intestine, which may be able to simulate a peristaltic unrest of the stomach if, with a displaced stomach, they are situated above the stomach and are very much in motion. The diagnosis of the purely nervous form of peristaltic unrest is supported by the demonstration that the patients affected are otherwise *nervous,* and that mental emotion, sexual excesses, etc., preceded the onset of the peristaltic unrest in the given case.

Much more frequent and much more certain of diagnosis are those spasmodic conditions which do not affect the stomach as a whole, but individual portions of the same, especially cardia and pylorus.

CARDIAC SPASM

Spasm of the cardia is distinguished by the fact that suddenly a spasmodic, constricting sensation occurs in the patient, behind the xiphoid process. Usually it is produced when a morsel of food passes the cardia, possibly also when the stomach-tube, on being introduced, reaches the region of the cardia. We feel, in the latter case, how the tube is suddenly grasped, and can only be freed and withdrawn after application of force with stretching of the tube. If the spasm lasts for some time, it may occur that the food, which becomes lodged or accumulates above the nervous stenosis, is forced out. The mere swallowing of air may cause a spasm of the cardia. If this occurs after large quantities of air have been swallowed, and if this condition becomes associated with a spasm of the pylorus, the air cannot escape either upward or downward, and now a condition of lasting inflation of the stomach develops, *pneumatosis* of the stomach, which may be followed by upward displacement of the diaphragm, præcordial anxiety, and a dyspeptic asthma. Spasm of the cardia may be caused by ulcer, by carcinoma of the cardia, by hyperæsthesia of the mucous membrane of the cardia, by profuse accumulation of gas in the stomach

upon stagnation of the food, or by hyperchlorhydria. In such cases it is a symptom of the respective pathological conditions; but spasm of the cardia may also occur as an independent motor neurosis. It is to be thought of if the above-named morbid conditions, the diagnosis of which has been fully discussed, can be excluded, and if it is a question, in the given case, of an extremely nervous individual. The facts, that a thick tube passes the narrow area without obstruction, whereas at other times a thin tube becomes lodged; that, after cocainization of the region of the cardia, sounding and swallowing of food is easily accomplished; or that, as I succeeded in doing in a case of chronic spasm of the cardia, during the systematic use of morphine and belladonna shortly before meals, the obstacle to deglutition caused by spasm of the cardia disappears regularly. If these points may be utilized in the diagnosis of spasm of the cardia, it may be classed as an independent motor neurosis.

SPASM OF THE PYLORUS

As often as spasmodic contraction of the pylorus occurs as a subsequent symptom of hyperchlorhydria, of ulcer, of carcinoma, of tugging cicatrices, etc., as rarely is it observed in a stomach that is fully intact anatomically. In the latter case it is brought about by food which is spicy, too cold or too hot, by accumulation of gas, etc., and eventually it may be diagnosticated by the delayed occurrence of salicylic acid in the urine in Ewald's salol test, or by the late appearance of iodine in the saliva in Fleischer's iodoform test (0.1 iodoform in gelatine capsule).

NERVOUS ERUCTATION AND VOMITING

Nervous Eructation.—This includes cases in which eructations occur in a nervous manner, and in which the belching is usually rapid and noisy. This manifestation is found principally in hysterical individuals and is probably caused by swallowing of air, followed by an immediate rising of the latter. Psychical emotions and other nervous factors may be the immediate cause of the occurrence of the eructations which usually take place in paroxysms of varying duration, and which may, temporarily or often permanently, be removed by suggestion, by distracting the attention, by keeping the mouth open, etc. The observation of the fact that the belching takes place without any restraint, with a certain ostentation, has often helped me to make a correct diagnosis.

Nervous Vomiting.—*Nervous* vomiting is understood to be vomiting which is not due to anatomical changes of the wall of the stomach, but to direct or indirect (reflex) irritation of the nerve tracts which cause the process of vomiting. A morbid irritability of those nerve tracts is usually to be presupposed.

Nervous vomiting occurs most frequently in *hysterical* and neurasthenic individuals, sometimes in the course of a fright, of anger, etc., also in various kinds of *cerebral and meningeal affections, spinal-cord diseases*, partly with anatomically demonstrable medullary changes (I have quite recently observed a case of tabes dorsalis in which the vomiting occurred periodically, was never accompanied with gastralgia, and always lasted several days), in affections of the kidneys, uterus, liver and, above all, of the peritonæum.

The diagnosis of vomiting, as being nervous vomiting, is based principally upon the possibility of excluding with certainty gastric affections which are usually associated with vomiting, and the subsequent demonstration of one of the above-mentioned causes of nervous vomiting. To a certain extent characteristic of this malady are: The frequency and ease with which the vomiting takes place mostly even without nausea, the contrast between the good appetite and the often irrepressible vomiting, etc., that it occurs entirely independent of quantity and quality of the ingested food and only at certain times, during which a more pronounced irritability of the nervous system is present.

MOTOR NEUROSES OF A DEPRESSIVE CHARACTER

A *genuine nervous atony of the stomach in toto*, independent of other affections of the stomach, is decidedly rare; some cases of gastric atony in hysterical persons may be explainable as nervous atonies of the stomach, also those gastric atonies which have been observed after cholelithiases, and motor insufficiencies which take place after overloading the stomach, which, in certain individuals, take place from childhood on and easier than in other individuals (*"congenital" predisposition to gastric atony, "weak stomach"*). Regarding the signification of paresis of the musculature of the stomach with reference to the origin of gastrectasis, I refer to what has been stated on p. 290. The points of support which are to be considered diagnostically in the assumption of an atony of the stomach have also been discussed at the same time.

INSUFFICIENCY OR INCONTINENCE OF THE PYLORUS

While the pylorus becomes insufficient owing to anatomical changes in some cases of gastric ulcer or carcinoma, etc., there are undoubtedly also instances of incontinence of the pylorus in which this condition occurs in the course of tabes dorsalis or hysteria, and is then explained as a nervous form of insufficiency of the pylorus. The diagnosis of this condition may, as was first taught by Ebstein, be made certain as follows: A large quantity of Seidlitz powder is introduced into the stomach, and then we must observe whether the developing carbonic-acid gas, as usually, distends the stomach and is retained here for several minutes, or whether it escapes at once through the insufficient pylorus without causing an inflation of the stomach. The diagnosis of incontinence of the pylorus is by no means certain in all cases and is, for the present, also without any clinical importance worth mentioning. Of more practical significance is

INSUFFICIENCY OF THE CARDIA

If the cardia is insufficient, the gases of the stomach escape more readily than is normally the case; this may explain many cases, previously described, of nervous eructations in hysterical persons. If not only gas, but also stomach contents escape through the insufficient cardia we speak of a *regurgitation*. If, then, the stomach contents which have ascended into the mouth, are not expectorated, but at once, after having been masticated once more, are swallowed again, we speak of *rumination* (*merycism*).

The diagnosis of this special form of insufficiency of the cardia is so

simple and so easy that a confusion with other morbid conditions leading to a similar picture, is scarcely possible. At most, it may be that the forcing up of previously swallowed food in stenosis or diverticulum of the œsophagus simulates a regurgitation; but it differs from the latter in so far as hydrochloric acid is constantly absent in the expectorated morsels of food which come from the œsophagus. The consideration of the disturbances of deglutition which are present and the result of the examination with the stomach-tube remove all diagnostic doubts.

That rumination represents a motor *neurosis* of the stomach, is undoubtedly proved by the ætiology of the affection, inasmuch as it owes its origin to the occurrence of pronounced psychical emotions, heredity, imitative instinct, hysteria, epilepsy, etc.

DISEASES OF THE INTESTINES

ACUTE INFLAMMATORY PROCESSES OF THE INTESTINES

ACUTE DIFFUSE (SIMPLE) ENTERITIS, ACUTE INTESTINAL CATARRH

Symptoms of Value in the Diagnosis of Enteritis.—The symptom which dominates the diagnosis is *diarrhœa*. The *stools* are thin, mushy, or watery in consistence and of a pale yellow or greenish colour, rarely blood-tinged, but as a rule containing mucus. If repeated evacuations of the bowels follow one another rapidly, the fæces lose their colour, obviously because the biliary constituents are less in proportion to the quantity of the excrement; the fæces now assume a rice-water character. The odour is then no longer fæcal but stale, sour; occasionally the diarrhœic stools have a mouldy odour and are foamy. In the microscopical examination there are found, besides undigested food, few round cells, many epithelial cells, which are still partly well preserved, partly greatly changed (enlarged with granular protoplasm, at times with a distinct, at other times with an indistinct, nucleus). Besides this there are found micro-organisms of various kinds: cocci, bacilli (among others the bacterium coli communis in great numbers) the saccharomyces cerevisiæ (yeast fungi), etc. None of these organisms is therefore of any clinical significance, as they are also found in the normal fæces in enormous quantities (over fifty millions per day). Neither can the crystals of triple phosphates, cholesterin, calcium salts, etc., which are also noted in the fæces claim any diagnostic importance.

Besides the most important symptom, diarrhœa, there are also present, in acute intestinal catarrh, *colic*, gurgling in the abdomen (borborygmi), frequent movements containing, as a rule, nothing but moist flatus, and slight alterations of the appetite. Usually the *abdomen is inflated* due to the accumulation of gas; on palpation the fluid contents of the intestinal coils show themselves by the sensation of fluctuation. As a result of the marked loss of water by way of the bowel, *great thirst* and *oliguria* may occur. The sparse urine may contain *albumin* as a result of the lessened blood pressure in the glomeruli (due to irritation of the splanchnic nerve). Also the

signs of reflex weakening of the cardiac activity, and of collapse occasionally result, especially if the intestinal catarrh occurs in aged individuals or in children.

Fever.—*Fever* is rarely present, it may, however, in my experience, attain great height (103.5° F.). Under such circumstances enteritis of an infectious nature should be thought of; the diagnosis may be between enteritis and enteric fever in such cases, although the regular temperature curve and the continued height of the fever, the relative slowing of the pulse and the enlargement of the spleen, even in the first few days before the eruption appears, are evidences of enteric fever and remove all shadow of doubt. Enlargement of the spleen in acute intestinal catarrh, according to my experience—rare exceptions might occur—is never noted.

Cholera Nostras.—The symptoms of *cholera nostras and acute intestinal catarrh of very young* children are somewhat modified, partly by the acuteness and intensity of the process and partly by the reaction of the infantile organism. The diagnosis of both affections is, however, easy. In cholera nostras not only the bowel, but the stomach is materially implicated in the process. For, besides diarrhœa, vomiting is also an essential symptom; as the fæces, so also is the vomited material of a purely watery consistence, after the food that has been last taken is vomited. Corresponding to the frequently enormous losses of fluid substance, unquenchable thirst, with oliguria and albuminuria, yes, even nephritis, occur. Turgescence of the skin disappears more and more, the pale or bluish face becomes emaciated, and general collapse makes its appearance; the pulse becomes small and even cannot be felt, the skin is icy and the voice inaudible, simulating the appearance of Asiatic cholera, all the more as painful muscular cramps, especially in the region of the calves, occur. A differentiation of the severe variety of cholera nostras from epidemic Asiatic cholera was simply impossible until recently. Since the discovery of the *comma bacillus* by R. Koch this differential diagnosis has obtained the required certainty (see description of comma bacillus under Asiatic Cholera.)

Acute Infantile Gastro-Enteritis.—The clinical picture of *acute infantile intestinal catarrh* is only differentiated from acute enteritis and cholera nostras of adults by unimportant details. The reaction of the fæces is mostly acid, the colour often greenish, the biliary colouring matter more or less unchanged and easily detected by the Gmelin reaction. Collapse is more intense, the extremities are cool, the fontanelles are depressed, anæmia of the brain shows itself in the greater pulse frequency, dilated, sluggish pupils, delirium, dyspnœa, convulsions, even rigidity of the muscles of the back of the neck—in short, the clinical picture of an "hydrocephaloid" is seen.

Diagnosis of Hydrocephaloid.—A mistake in the diagnosis of the affection is hardly possible; at most the symptoms just enumerated might simulate a meningitis. The differential diagnosis will be discussed later on (see Meningitis); only this much will be mentioned here, that the preceding diarrhœa, the sunken condition of the fontanelles, and the absence of partial paralysis are very much in favour of hydrocephaloid, as well as the absent ophthalmoscopical findings (in contrast to the frequent positive findings which are characteristic of the eye ground in tubercular meningitis).

Special Varieties of Acute Enteritis.—If the inflammation does not affect the greater portion of the bowel, but is *localized and limited to certain areas*, other phenomena arise, the diagnosis of which requires special elucidation. We will proceed from above downward:

Acute Catarrh of the Duodenum.—A catarrhal process limited to the *duodenum* can only be diagnosticated from the symptoms just enumerated

with the addition of catarrhal jaundice. This is not even a constant condition, and under such circumstances the *diagnosis* is *impossible.*

If in cases of acute catarrh of the stomach conspicuous tenderness in the right hypochondrium is added, it is well to think of the spread of the catarrhal process from the stomach to the duodenum, and the urine should be tested for bile pigment; only, however, when the symptoms of jaundice make their appearance are we justified in making a diagnosis of duodenal catarrh.

Acute Catarrh of the Jejunum and Ileum.—An acute catarrh of the jejunum and ileum not accompanied with a catarrhal process of the large intestine, which by the way is quite rare, gives rise to no distinct phenomena. As the most important symptom of the intestinal catarrh, the *diarrhœas, is absent here,* on account of the fact that normally the chyme passes through the small intestine very rapidly—in a few hours. Whether this is due to the process being confined to the small intestine or not, is irrelevant so long as the fæcal mass has sufficient time, as in the normal state, to mass itself in the large intestine, which undoubtedly is the case, provided the catarrhal process is confined to the small intestine. It is therefore not astonishing that, in catarrhal processes limited to the jejunum and ileum, there is no increase in the number of stools from the normal; however, *the composition of the fœcal mass is different* from the normal condition.

Composition of the Dejecta.—*The masses of mucus which have become detached in the inflamed small intestine are intimately commingled with the thickened fœcal mass in the large intestine;* even if macroscopically no mucus can be detected, *microscopically* there may be seen, in the compact and putty-like mass, small mucous particles.

Nothnagel was the first to call attention to this condition. Unfortunately it is of little practical moment in a differentio-diagnostic respect, as the consistence of the fæces does not at all point to an irritative disease of the bowel and does not call for an examination. Only when colicky pains and borborygmi are present without diarrhœa, and the nutrition of the patient appears to suffer to a marked extent, will we be cautious enough to make a microscopical examination of the fæces and to look for mucus in the compact stool.

Catarrh of the Small Intestine and the Upper Portion of the Colon.—The same is true of cases in which *the small intestine and the upper portion of the colon are implicated in a catarrhal process.* Here also the mucous particles are intimately admixed with the fæcal mass, but active diarrhœa is not a symptom.

Combination of Catarrh of the Small Intestine and the Colon in its Entire Course.—More certain diagnostic points are obtained *if the colon is affected in toto with the small intestine by the catarrhal process, or if the colon shows an increase at least in the peristalsis,* so that the rapid downward movement of the contents of the small intestine through the large intestine enables us to note the character of the pathological dejecta due to the catarrh of the small intestine. The intimate admixture of the fæcal masses quoted above is no longer of value in this supposedly thin consistence of the excrement; on the other hand, the examination of the stool

for *unchanged bile pigments* and the *condition of the admixed particles of food in the fæces* convey to us valuable diagnostic hints.

Bile Pigment Reaction.—In the normal bowel, bile pigment can only be detected downward as far as Bauhin's valve (ileo-cæcal valve) by the Gmelin reaction; from this point onward the bile is altered. If there should be found a distinct reaction for bile pigment in the diarrhœic stool, it is a proof that a more decided peristalsis affected the large intestine, or that this was also the seat of a catarrhal inflammation. However, the reaction with the filtrate of the fluid dejecta is rarely distinct; it is rather better marked in the diarrhœic stools of nurslings, but, nevertheless, I have convinced myself that it also occurs in the stools of adults.

Nothnagel found that the *bile pigment adheres especially to the mucous particles*, and if the reaction is attempted with them, it will result positively, whereas it but rarely occurs if the watery constituents are used for the test. Bile pigment is also found attached to the epithelial and round cells of the excrement, and especially to the so-called yellow mucous granules, mucous particles as large as poppy seeds, to which we shall refer again (see p. 317).

Faulty Digestion of Various Foods.—For the same reason that unchanged bile pigment is found in catarrh of the small intestine, with simultaneous catarrh or increased peristalsis of the large intestine, *undigested, well-preserved particles of food* are found (*"lientery"*), that is, upon microscopic examination *many* muscle fibres, well-preserved starch granules, large quantities of fat masses, even without the partaking of nourishment rich in fat by the patient.

I coincide fully with Nothnagel, who maintains that the finding of fat in large amounts in the stools has no practical value for the localization of the intestinal affection, as disturbances of the biliary and the pancreatic secretions may prevent the resorption of fat, and the finding of muscle fibres and of starch granules requires limitation in the diagnosis of catarrh of the small intestine; that is to say, it is only of value provided that other factors which are influential in preventing the digestion of these products of nutrition (fever, catarrh of the stomach, etc.) are absent in the individual case, and, on the other hand, certain symptoms point directly to the presence of a catarrh of the intestine.

If the acute catarrh is limited to the *large intestine*, the clinical picture will vary accordingly as the upper or lower portions of the same are affected separately. According to the seat of the affection, we may differentiate: typhlitis with inflammation of the vermiform process, colitis and proctitis.

INFLAMMATION PROCEEDING FROM THE LARGE INTESTINE IN THE RIGHT ILIAC FOSSA—TYPHLITIS, SKOLIKOIDITIS (NOTHNAGEL), "APPENDICITIS," PERITYPHLITIS

It is well known that inflammatory processes are very frequent in the right iliac fossa. It was formerly believed that they originated in the cæcum and were the results of an inflammatory catarrhal process due to stagnating fæcal masses (*typhlitis stercoralis*) and from thence the inflammatory process would spread with or without the formation of pressure ulcers from the cæcum to the adjacent peritonæum (*perityphlitis*). This view has recently been entirely abandoned, as post mortem examination, and especially the early operations which were undertaken to subdue

these inflammatory conditions, have proved conclusively that their origin is to be found in the cæcum only in the rarest instances, that, moreover, more than 90 per cent of the cases originate in the *vermiform appendix*. It is the rule, that a circumscribed peritonitis (*perityphlitis*) is superadded secondarily, and this represents the most important factor in the condition. As, however, this extremely important disease in practice is almost exclusively an inflammation of the vermiform process, the appendix of the cæcum, the disease and its consequences will not be described under the affections of the peritonæum but among the inflammations of the bowel.

We will first describe the symptom-complex of the ordinary form of perityphlitis that arises from an inflammation, respectively perforation, of the vermiform process and which runs its course with a more or less well-pronounced tumour formation, then discuss the diagnosis and further on see in how far it is possible in the individual case to diagnosticate separately an isolated inflammation of the vermiform appendix, the simultaneous inflammation of the retrocæcal connective tissue ("*perityphlitis*") and circumscribed or diffuse secondary peritonitis individually.

Symptoms of Perityphlitis.—Generally in the best of health, or following indigestion, "taking of cold" or the like, preceded by any ordinary trifling indisposition, the affected person is attacked by *pain* in the right iliac fossa. It is rarer that another circumscribed area of the abdomen, for example the gastric region or the entire abdomen, should be affected, and only later that the pain should localize itself to the cæcal region, a fact that undoubtedly occurs, according to my experience, but does not appear to me to be explainable in a satisfactory manner. The *pain is increased by walking*, frequently, also, upon urination, upon coughing, and, above all, by percussion and pressure in the affected area. In my experience, the boundaries of the peritoneal inflammation may be very accurately determined by *light* percussion, by asking the patient to denote whether the individual light percussion-blow is painful or not, and this method of percussion, aimed at defining the tender area, if practised several times daily, is an excellent method of diagnosticating the progress of the inflammation and of reaching an indication for the performance of laparotomy. As the pain in the inflammatory area is due to peritoneal irritation, it is usually accompanied (in at least over one half of the cases) *with vomiting, constipation*, and occasionally with the symptoms of a mild *collapse*. Fever occurs at the onset, which, however, is irregular and not at all of a characteristic type. Especially is there no parallel between the height of the fever and the severity of the process. Every physician of experience will have seen cases, that were operated upon, which showed gangrene and foul-smelling pus in which the temperature was normal or at least subfebrile. Nevertheless, this is the exception; as a rule, there is decided fever, generally of 103° F. and above this. The fever also has some slight influence on the prognosis. Absent or slight fever does not necessarily indicate a mild course, as we have seen; but, on the other hand, a high temperature of 103° F. and over, if it continues for a week or more, almost invariably shows a severe course. High temperature with marked remissions (with chills) is in favour of septic infection.

Tumour—Fæcal Vomiting.—More important than the symptoms above enumerated is resistance or *tumour formation* in the right iliac fossa.

Occasionally this may be noted at first sight, and may be confirmed by percussion showing dulness; but inspection and percussion give uncertain results—palpation proves with exactness that a more diffuse or a sharply defined border of a tumour may be noted in the cæcal region or its immediate vicinity. The mass is immovable, its surface smooth, only later in the course of the affection, when abscess formation occurs, does it become soft and fluctuating. The tumefaction is the result of the inflammatory infiltration of the walls of the cæcum and of the appendix, of the peritoneal exudate, of an inflammatory serous infiltration of the belly wall, and, finally, of masses of fæces that may be present in the cæcum. A symptom which in part is due to the tumour formation which, however, is rare, is *fæcal vomiting*. The cause of this may be an occlusion of the bowel, due to compression of a coil of intestine as a result of the perityphlitic mass, or it may result from an obstruction (nipping off) of the bowel by peritoneal adhesions. But even without mechanical hindrances, the clinical picture of volvulus may appear in perityphlitis due to reflex paralysis of the bowel.

Course of Perityphlitis.—The *course* of perityphlitis varies greatly in the individual case, and the prognosis is frequently exceedingly uncertain on this account. In by far the greatest majority of the cases (statistics vary about 90 per cent) *spontaneous recovery* occurs. The fever subsides, and *resorption* of the exudate takes place, be it due to the fact that the latter has been serofibrinous or that it did not contain large quantities of virulent pus. That resorption of the pus may occur, can no longer be doubted at this late day. In any case, a callous induration occurs; in some of the cases a pocket of pus may remain in the connective-tissue mass which may be the source of a relapse. A second variety of the course of the disease is that the pus may *rupture* into the bowel, into the bladder, rarely into the vagina, through the skin, etc., with sudden subsidence of the fever and spontaneous recovery. Entirely different, however, is the condition if the pus ruptures into the peritoneal cavity. An acute, severe, diffuse peritonitis of a septic character with a fatal outcome suddenly develops. But even without perforation, the purulent perityphlitic inflammation may gradually encroach upon the peritonæum and give rise, partly to a diffuse peritonitis, partly to sacculated pus foci, which latter at least are capable of a partial cure.

Differential Diagnosis.—*The diagnosis of perityphlitis* in the majority of cases, after what has been said, is easy. The decision is more difficult if we are not content with a simple diagnosis of perityphlitis but attempt to designate how far the retrocæcal connective tissue is implicated or whether the process is confined to the appendix and its serous coat.

Inflammation of the Vermiform Process.—The limits of the pain and the tumour in the region of the appendix are in favour of the isolated inflammation of the vermiform process. Both symptoms have very questionable value. The pain may radiate, or, as has been mentioned above, may be found in the epigastrium or in the abdomen, and the tumour mass is only then in favour of appendicitis if it occupies the area in which the appendix is found according to experience. This point is between the anteror superior spinous process of the ilium and the umbilicus about 6 centimetres

from the former (McBurney's point) and corresponds to the origin of the appendix from the cæcum. This position is nothing less than constant, as the appendix is sometimes found in the true pelvis, occasionally behind the cæcum and even in other regions of the abdomen. Occasionally the appendix is seen pointing upward and running along the course of the ascending colon, as in a case of my own; it has even been found in the left and in the right hypochondrium, therefore beside the liver and beside the spleen. Naturally, under such circumstances a diagnosis cannot be made. Of greater importance than the position of the appendix is the *shape* of the tumour; if the inflammation be localized to the appendix, there will be found a cylindrical mass having the thickness of the little finger, and painful to the touch. All other symptoms that have been noted in appendicitis, such as vomiting, constipation, fever, etc., convey no differential points in separating this affection from perityphlitis in general.

Paratyphlitis.—It is quite usual that the retrocæcal connective tissue should be affected by the inflammatory process; on the other hand, according to the most recent investigation, it is very questionable whether a *primary* paratyphlitis ever precedes the perityphlitic process—a mere theoretical question, as the cæcum is almost entirely enveloped by peritonæum and, on the other hand, it is infrequent that the appendix should be found intraperitoneally. The inflammation of the retroperitoneal connective tissue will therefore, in case it should develop primarily, naturally combine with a perityphlitis; the diagnosis of a paratyphlitis is therefore of very little value. The deep position of the swelling, radiation of the pain into the right lower extremity, paræsthesia and weakness in the leg, pain on flexing the right thigh, point to a participation of the retrocæcal cellular tissue and allow of the assumption that this part is particularly affected.

Differentiation of other Tumours of the Ileo-Cæcal Region.—Mistaking the perityphlitic mass for resistance of the muscles or other tumour in the ileo-cæcal region, such as *tumour of the kidney, "incarcerated" wandering kidney, descending abscess in caries of the vertebræ, carcinoma of the cæcum,* and others, provided no complication is present, does not occur so long as the entire clinical picture of perityphlitis is kept in mind and a careful physical examination is made. The tumour should never be considered alone in the diagnosis. The same is true of possible mistakes in the case of *inflammation of the hip-joint,* which is only possible if the symptoms of retrocæcal connective-tissue inflammation, such as pain in the hip and the painful position of the thigh, are present and the other signs of perityphlitis are entirely ignored. However, errors as to the nature of the mass cannot be avoided if the appendix should occupy an abnormal position in the abdomen. In those cases in which fæcal vomiting instead of ordinary vomiting occurs, the diagnosis may waver between perityphlitis and *occlusion of the bowel.* The details of the differential diagnosis will be explained later on (see p. 329); only so much shall be mentioned here, that *intussusception* is most frequently ileo-cæcal and also sets in with (colicky) pains and vomiting, but, as a rule, the mass has a different shape —a thick, smooth, cylindrical tumour is found on palpation, and these symptoms are accompanied with a bloody, mucous diarrhœa.

COLITIS, PROCTITIS

Acute Colitis.—*Acute colitis* is characterized by thin stools, provided the inflammation affects not only the upper portion of the colon. More or less mucous masses are contained in these stools; however, in contrast to those cases in which there is a simultaneous catarrh of the small intestine, they *do not contain decomposed bile pigment.* Bile-stained epithelial and round cells as well as quantities of muscle fibres are also absent in these diarrhœic stools. Besides the condition of the stools in acute colitis, there are to be considered in the diagnosis: *Tenderness along the course of the colon* and a *gurgling or splashing sound* corresponding to this area, which is audible and may even be detected by palpation, due to fluid and gas being present in the large intestine.

Proctitis.—If the inflammation affects the rectum, *tenesmus* occurs, bearing down, with preceding colicky pains in the left lower abdominal region. The sphincter ani is spastically contracted; the evacuations from the bladder are irregular and spasmodic. The act of defecation is very painful, and all but small quantities of bloody mucus are evacuated; with this the rectal wall may protrude in the form of a deep red mucous membrane tumour. If fæces are evacuated at all, they are covered by a layer of blood and mucus. The diagnosis of proctitis in the presence of these symptoms is not difficult.

The direct examination of the rectum by means of the finger or speculum, which in the chronic variety of proctitis determines the diagnosis, is unnecessary in the acute variety, and in more marked grades of the affection it is even impossible, as the morbid contraction of the sphincter and the intense pains render the digital examination impossible, and even if it be accomplished nothing but a swollen and hot mucous membrane will be detected.

Phlegmons and Diphtheritic Enteritis.—Besides the ordinary form of acute inflammation of the bowel, there also occur *phlegmonous and diphtheritic inflammations.* The former has but a pathologico-anatomical interest, the latter may be diagnosticated under some circumstances. It is true that in some cases no symptoms are present, in others, however, similar to infectious dysentery, mucous and purulent masses, partially in the shape of white clots, further necrotic mucous-membrane particles and blood are evacuated. The fæces are passed accompanied with marked tenesmus and severe colic; but these subjective phenomena are not prominent in the non-dysenteric variety of diphtheria of the bowel, as the disease usually occurs in the course of constitutional affections (carcinoma, Bright's disease, diabetes, etc.), and of the infectious diseases (typhus, sæpsis, puerperal fever), and the severity of the underlying affection entirely masks the subordinate bowel affection.

[*Sprue* or *psilosis* is a form of inflammation of the digestive tract, chronic as a rule, occurring in unacclimated persons residing in tropical climates. It is characterized by "a peculiar, inflamed, superficially ulcerated, exceedingly sensitive condition of the mucous membrane of the tongue and mouth; great wasting and anæmia; pale, copious, and often loose, frequent, and frothy fermenting stools; very generally by more or less diarrhœa, and also by a marked tendency to relapse" (Munson). Post mortem, ulcerations of the colon have been noted. It is considered by some authors a variety of dysentery.]

CHRONIC INTESTINAL CATARRH

Constipation.—Whereas in acute intestinal catarrh diarrhœa is the most frequent form of defecation, in chronic catarrh *constipation* is the rule. In some of the cases, a permanent sluggish condition of the bowels is noted, in others constipation alternates with diarrhœa—in fact, irregularity of the fæcal evacuation is the most prominent symptom of the disease. Occasionally even continuous diarrhœic stools are evacuated.

The reason for the *constipation* is a weakened condition of the muscular coat of the bowel, due to the chronic inflammation (analogous to the languid condition of the muscular structure of other organs in chronic inflammations) and of the inactivity of the nerve supply of the bowel, especially the automatic inactivity of the nervous apparatus of the gut (Nothnagel). If constipation alternates with diarrhœa, accompanied with colic, according to my view, this may be explained, that on account of the predominant constipation and a more marked foul decomposition of the contents of the gut, an intense irritation of the nervous apparatus of the bowel occurs which produces the increased peristalsis. As a proof of the correctness of this observation, the treatment of such patients with non-irritating laxatives gives the best therapeutic results. In other cases the thin evacuations are due to more pronounced irritation of the inflamed mucous membrane by dietary errors, etc.

Abnormal Constituents of the Fæces of Value in the Diagnosis.—More reliable information than the consistence of the stools in the diagnosis of chronic intestinal catarrh, and points to localize the affection to a particular part of the bowel, will be obtained by an *examination of the fæces for abnormal constituents.* The most important of these is *mucus*. It is almost never absent in chronic catarrh, and will be particularly noted if the fæces have been observed for some time; its commingling with the fæces, in fact, determines the diagnosis. *If pure mucous masses* are evacuated this points to a catarrh of the *rectum; enveloping of the scybala in mucus* is noted in the *rectum high up, extending into the large intestine as high as the transverse colon.*

It is self-evident that these are only general rules, that therefore, per example, if there should be an inflammation higher up, in case the colon contains but little fæces in its lower portion, the mucus may also be evacuated in an almost pure condition, and *vice versa*, in spite of a catarrhal condition of the colon and rectum, mucus may be entirely absent if in the passing of the fæces it has not been grasped, etc.

If there be noted macroscopically mucus in the mass and upon microscopic examination mucous particles are intimately mixed with the compact or pulpy stool, it indicates, as already mentioned, that there is a participation of the upper portion of the large intestine *and of the small intestine* in the catarrhal process.

"Sago-Pearl" Particles in the Stool.—Occasionally there are found in the dejecta "frog-spawn-like" swollen, glassy particles resembling "sago pearls," which are apparently composed of mucus. In some of the cases this is, in fact, true. I have been able to convince myself of this, by microscopical examination, frequently; besides, Kitagawa has proved the mucous character of the particles in my laboratory by chemical examination. More

frequently, it is true, these sago-like structures are of *vegetable* origin. At times both kinds of sago-like structures are found together in one and the same stool. More important for the diagnosis are the "*yellow mucous granules*" first described by Nothnagel.

Yellow Mucous Granules.—They are the size of poppy seeds, yellow or yellowish-brown in colour, and these soft particles when they are present in the stool in great numbers give it a yellowish-brown speckled appearance. They owe their colour to unaltered bile pigment, and thus their origin from the small intestine is established. This is also proved by the bile-stained mucus which is evacuated in shreds. It is presupposed that peristalsis is increased in the large intestine so that the onward movement of the bile-stained mucus is more rapid, and a transformation of the bile pigment cannot take place. For this reason the yellow discoloration of the stool in *chronic* catarrh, even when the small intestine is implicated, is of *rare* occurrence; this is also true of the yellow-stained cylindrical epithelium. On the other hand, they undergo a special alteration in chronic catarrh which must be mentioned; they become "*cloddy*" (Verschollung) —that is, the epithelium is small, homogeneous, without nucleus, and spindle-shaped.

Cloddy Epithelium.—This "cloddy" epithelium is especially found in the mucus that envelops the compact fæcal masses, and was also first described by Nothnagel; they have very likely undergone this metamorphosis by a sort of drying process. *Round cells* are rarely met with in large quantities, and then only when there is a *proctitis* present, in which case there is also an evacuation of pus with the fæces.

Membranous Enteritis.—In individual cases of *catarrh of the large intestine* (occasionally at the onset) separation of large, coherent, white, shred-like masses occurs. From time to time, often daily, large quantities of these, *at times membranous, at times solid cylindrical masses,* are passed (*"mucous colic," colica s. enteritis membranacea*). According to my experience, it is certain that these structures may arise *during the course of a chronic enteritis*, the simultaneous presence of round cells and (partly cloddy) epithelial cells in the membrane favouring this view.

As regards their *chemical* composition, this is apparently not always the same. Sometimes they consist principally of mucin, in other instances there are but traces of mucin present in the chemical examination and the membranes then consist, as I have proved in my laboratory, almost entirely of an *albuminoid* substance.

Nervous Enteropathy with Formation of Membranes.—As certain as is the occurrence of membrane formation during the course of disease of the intestine, it cannot be denied, as every physician who has frequently seen the affection, will admit, that there is a connection between this disease and *nervous affections*. Nearly all the patients observed by me were hysterical; all medication aimed at improving the catarrh of the bowel, even when continued for months, proved futile. After the great number of cases that I have observed, I cannot withhold the conviction that in these cases of disease of the bowel, accompanied by the formation of membrane, in the majority of cases, at least, this is a *neurosis of secretion*. The

masses of secretion that have become separated very likely collect for some time in a portion of the bowel that has become inactive or *spasmodically contracted* (obstinate constipation accompanies this condition) in the longitudinal folds of the upper surface of the wall of the intestine and form into round strands, then under marked peristaltic action of the bowel, with colicky pains, they are evacuated, a view which in its fundamental principles was enunciated by Marchand, and, according to my opinion, is correct.

Mistaking these membranes for vegetable formations (asparagus stems, etc.), coagula of milk and others, may occur upon a superficial examination of the dejecta. A closer, especially a microscopic, examination of these questionable structures will soon clear up any possible mistake.

Secondary Symptoms of Chronic Enteritis.—Besides the altered condition of the fæces, which is of value in the diagnosis of chronic intestinal catarrh, the other symptoms are but of slight importance, partly because they are not constant and partly because they are ambiguous.

Among these are flatulence, borborygmi, the deficient passage of flatus, the slight pains in the abdomen, most frequently in the region of the transverse colon, eventually increased by palpation, further, palpitation of the heart, the secondary dyspepsia and the psychical depression, which are almost never absent in chronic enteritis. The *general nutrition* also suffers in the course of long-continued catarrh of the bowel. This is especially marked in *chronic catarrh of the small intestine* and in the *chronic catarrh of the intestine of children in the first years of life* ("*pedatrophia*"). Marked *fatty stools* are even noted in this condition. The children, who have emaciated almost to the bone, are, as a rule, either rhachitic or tubercular [the cœliac affection of Gee]. In long-continued infantile, intestinal catarrh tubular atrophy of the glands and wasting of the muscles occur. In adults there is frequently also *atrophy of the mucous membrane of the intestine* as a result of acute and chronic catarrh of the intestines.

INTESTINAL ATROPHY (ATROPHIA INTESTINALIS)

Atrophy of the Mucous Membrane of the Bowel.—The atrophic change principally affects the glands, which may completely disappear; but the other parts of the intestinal wall also take part in the process, although to a lesser degree, especially the muscularis; the submucosa and the follicles are, however, rarely implicated. The small and the large intestines may be the seat of the affection, the cæcum and the adjacent portions of the ileum and the ascending colon are especially predisposed.

According to Nothnagel, who has made a special study of the condition, *a clinical diagnosis of atrophy of the bowel* cannot be made, when there is a circumscribed atrophy of the cæcum and the lower portions of the ileum, as there are no symptoms. The condition, however, is different when large areas of the mucous membrane of the large and small intestines are implicated in the atrophy. In this event a probable diagnosis, at least, is possible.

Atrophy of the Mucous Membrane of the Large Intestine.—First, as regards the *atrophy of the mucosa of the large intestine*, this is shown in the disturbance of function of the latter, by its faulty resorption of water, and the absence of mucus formation. The stools are then *soft*, *pulpy*, and *without mucus*; but a diagnosis cannot yet be made from this condition, as this composition of the fæces can only then be expected, when the *entire* large intestine is affected by the atrophy, and under some circumstances the bowel may show similar fæcal masses when in entirely normal condition, due simply to an increase of peristalsis. On the other hand, mucus may be present in the dejecta, in spite of atrophy, provided the parts adjacent to the atrophy have become affected by the catarrhal process.

Atrophy of the Mucous Membrane of the Small Intestine.—Atrophy of the mucous membrane of the small intestine is especially characterized by insufficient resorption of the constituents of the food; individuals thus affected show a severe type of cachexia (in children this condition is spoken of as phthisis or tabes mesenterica). To attribute these conditions absolutely to intestinal atrophy is not allowable, it is only highly *probable*, if the symptoms of a previous catarrh of the small intestine are no longer present and the cachexia which nevertheless exists cannot be explained in any other manner. But these factors which allow of a probable diagnosis will rarely be found to coexist. In my opinion, intestinal atrophy, for the time being at least, is based more upon an anatomical than upon a diagnostico-clinical foundation.

ULCERS OF THE INTESTINES

The diagnosis of ulcer of the intestine can rarely be made with precision. *In the majority of cases the ulcers run their course without symptoms*; at least, the phenomena which are supposed to be characteristic of them: diarrhœa, pain, blood and mucus in the fæces, etc., upon critical analysis prove that their diagnostic value is insufficient and too uncertain to establish a positive diagnosis.

For a long time certain symptoms, such as diarrhœa, with sago-pearl-like mucous particles, were supposed to be pathognomonic of ulcer of the bowel, because in the cases of phthisis in which this diagnosis was made, the autopsy apparently confirmed this view; but these ulcers would also have been found post mortem even if no diarrhœa had been present during life, that is, the diagnosis would have been just as probable that intestinal ulcers were present, as the latter are almost invariably present in long-continued tuberculosis.

Diagnostic Symptoms.—If we examine the symptoms without prejudice and separately, which have been noted to occur, in regard to their diagnostic significance, we will come to the conclusion, not very pleasing no doubt, that an absolutely certain diagnosis can hardly be arrived at from the symptoms which are usually present in ulcer of the bowel.

Diarrhœa.—First, as regards *diarrhœa*, my own observations and the opinions of others have shown that wide-spread ulcers of the bowel may exist without giving rise to diarrhœa.

It is certainly correct that extensive ulcerative processes give rise to diarrhœa, in that, as a result of the ulcerative process, the nerves which have been laid bare, and which give rise to greater peristalsis in this manner, prevent the resorption of fluids. However, this does not hold good after what has been said in catarrh of the intestines, especially not for ulcers of the small intestine and the upper portion of the large intestine. It is different with ulcers of the lower half of the colon and the rectum; here, necessarily ulceration must produce diarrhœa. In fact, this is occasionally true, but only in some of the cases; in other instances it is even absent, as ulcers which run a chronic course, as in other portions of the body, do not react to weak impulses, and the concomitant catarrh of the bowel which is also favourable for the development of diarrhœa, is so chronic or so slight that an obtunded condition of the nervous apparatus of the bowel does not react to this stimulus.

Composition of the Stools.—Somewhat more important for the diagnosis of ulcer of the intestine is the *composition of the stools*. Quite properly, stress has been laid, in this respect, upon the mixture of blood, pus, and particles of tissue.

Blood in the Fæces.—Blood may occur in the dejecta as a result of all kinds of pathological processes, and I wish only to enumerate the most important besides ulcer: In trauma, in conditions of hyperæmia, in hæmorrhoids, in enteritis, in neoplasms, in embolism of the mesenteric artery, in purpura, and in all general diseases which give rise to hæmorrhage.

It is most easy to mistake the bleeding which arises from the dilated veins of the mucous membrane of the rectum without the occurrence of perceptible hæmorrhoids; but in this case an examination of the rectum by means of the finger or speculum will soon give the diagnosis the proper direction. A simple non-toxic, especially chronic, enteritis rarely gives rise to bloody stools, so that the inclusion of simple inflammation of the bowel into the differentio-diagnostic circle is usually more of theoretical than practical utility. More or less, this is also true of the above-quoted causes of enterorrhagia, as, on the one hand, they are so well characterized by other symptoms besides blood in the stool, that they are not likely to be confounded with ulcer of the intestines, and, on the other hand, ulcers arise under very pronounced ætiological conditions: enteric fever, tuberculosis, etc.

If there should therefore be present in the individual case a recognised factor which frequently admittedly gives rise to ulceration of the bowel, the appearance of enterorrhagia would be a very important diagnostic indication. The absence of an admixture of blood from the fæces, however, proves nothing against the presence of ulcer, as even with the most extended ulceration of the intestine blood may be absent.

Pus in the Fæces.—*Pus may only be expected in the dejecta when the ulcers have their location in the large intestine, especially in its lower portion.* As the ulceration, however, most frequently takes place in the small intestine, and the pus which is deposited is speedily washed away, the value of finding pus in the stool is of less practical importance—that is, *the absence of pus from the fæces does not permit us to exclude ulcerative processes in the bowel.* Exceptionally, there may appear in the stool small particles of pus, even when the ulceration is exclusively limited to the small intestine. On the other hand, pus in the fæces loses something in its positive aspect as a symptom of ulcer of the bowel, as this condition occurs in other affections of the bowel, especially in cancer of the intestine. Nevertheless, the appearance of pus in the fæces is a symptom of importance in ulcer of the bowel, as it is of relatively frequent occurrence.

Mucus in the Stool.—The appearance of *mucus* in the stool, on the contrary, is of no diagnostic importance; only the swollen sago-pearl-like mucous particles are regarded by many as a sign of follicular ulceration. However, in those instances in which these particles are actually composed of mucus and are not, as is so frequently the case, of vegetable origin, it is very questionable whether they are the mucous output of the follicular ulcers, as these minute coherent mucous masses are also found in the dejecta in simple catarrh of the intestine, but, in cases that terminate fatally, they have been missed at the point of the ulcer, at the autopsy (Kitagawa).

Tissue Shreds.—*Tissue shreds* are decidedly more characteristic of ulcer than is pus in the stool. Unfortunately, they are of rare occurrence, so that, if we except the cases of dysenteric ulceration, we are hardly ever in a position to make use of these pathological constituents of the stool in the diagnosis of ulcer of the bowel.

Other Symptoms of Ulcer—Pain.—All other signs which have been supposed to occur in ulcer of the bowel are comparatively *worthless*, such as *pain* in the abdomen, *fever*, and others. *Pain* may be entirely absent; it is only of diagnostic value when it is *circumscribed*, always localized to the same region, and is increased upon pressure. Under these conditions, pain is of importance in the diagnosis, provided symptoms and history are in favour of the existence of an ulcer. *The pain, however, dare never, even if it be localized strictly, be the starting-point for the diagnosis.* I have seen a case in which pain was distinctly circumscribed to the left flexure of the colon, terminate fatally, and upon post mortem examination the most minute pathologico-anatomical research failed to reveal the cause of the pain or the reason of the fatal termination. These statements are, however, not meant to imply that ulcers of the bowel, as such, are not capable of giving rise to pain. In some individual varieties of ulcer the pain even has a typical character, especially due to the seat of the ulcer; *ulcers of the duodenum* give rise to *cardialgia*, those of the rectum to painful *tenesmus*.

Complicated by Peritonitis.—If the ulceration invades deeply and the peritonæum is irritated, there are added to the mild symptoms, or to the ulcer which up till then may have run a latent course, a *circumscribed peritonitis*. The pains now become much more severe, and slight dulness (due to the exudate and the formation of abscess) may arise in the particular area, with nausea, etc., or a general *peritonitis* may arise, usually with perforation of the bowel and the entrance of air into the peritoneal cavity. If ulcers are located in portions of the gut that have no serous coat, such as the posterior wall of the inferior, descending, and transverse parts of the duodenum, or of the ascending and descending portions of the colon, they may rupture without giving rise to peritonitis; then purulent infiltration of the cellular structure in the renal region, etc., occurs, and descending abscesses in the inguinal and other regions may form.

Duodenal Ulcer.—Some few ulcers of the bowel show a clinical picture which varies from the conditions just described, and in the interest of diagnosis they must now be discussed. *Duodenal ulcer*, analogous to ulcer of the stomach, may run a latent course; usually, however, it gives rise to dyspeptic phenomena, pressure or severe pain in the epigastrium, which is situated to the right of the pylorus and is increased by the taking of food and upon pressure. With this, there is vomiting, especially if a consecutive dilatation of the stomach develops. Eventually, even hæmatemesis occurs. As a rule, however, the blood is passed in the form of thin black stools; these bleedings may terminate fatally, and perforation with peritonitis may also be the cause of the fatal catastrophe. *Duodenal ulcer can never with certainty be differentiated from gastric ulcer.* Certainly the complication with jaundice, and, further, the fact that dyspeptic symptoms are absent, as a rule, that pains arise later than in gastric ulcer after the partaking of food, that vomiting does not relieve the pain, make the diagnosis of duodenal ulcer certain; however, these are all theoretically constructed assumptions and not substantiated by clinical facts.

Perhaps the fact that in ulcer of the stomach, in the majority of the cases at least, there is an increase of free hydrochloric acid, may be of value in the differential diagnosis, as *in a doubtful case the absence of superacidity of the gastric juice would point to the diagnosis of duodenal ulcer.* A case of duodenal ulcer lately observed by me (on which a post mortem was held) was in favour of this assumption (0.16 per cent acidity); naturally *one* case proves nothing. Besides, in gastric ulcer low amounts of total acidity occur, and the theory that duodenal as well as gastric ulcer is due to the peptic strength of the acid, respectively superacid, condition of the gastric juice, is *à priori* opposed to such a differentio-diagnostic discernment between the two diseases.

We may suspect a duodenal ulcer instead of a gastric ulcer *if the blood is only passed downward*, if vomiting and pronounced cardialgia occur rarely, if the pains are steadily limited to the right hypochondrium, and do not occur immediately upon the entrance of food into the stomach, although this latter condition is by no means constant in ulcer of the stomach.

Ulcers of the Vermiform Process.—*Ulcers of the vermiform appendix* form, as a rule, an accompanying condition of perityphlitis, which has been described on p. 312. We have there also explicitly discussed the diagnosis of inflammations limited to the appendix. We shall here only discuss whether a differential diagnosis between gangrenous or perforative appendicitis and simple catarrhal inflammation of the appendix is possible. I believe that a probable diagnosis, but not an absolutely certain one, is possible, as with catarrhal appendicitis superficial loss of substance (erosion ulcer) occurs as a secondary manifestation. The milder development of the appendicitis in all of its symptoms (pain in the ileo-cæcal region, the cylindrical shape of the tumour, as thick as the little finger in the region of the vermiform appendix, etc., see p. 313), are in favour of the catarrhal, inflammatory form, whereas the fulminant course, the vomiting, the high fever with chills are especially in favour of the gangrenous variety. Whether with these symptoms a perforation of the appendix occurs, or without this—i. e., with a relatively intact wall of the organ—the peritonæum surrounding the appendix is implicated in the inflammatory process, *cannot be diagnosticated* in the individual case, and it is not even of importance for the further course of the affection.

From what has been said it has been sufficiently shown *how difficult it is to make a diagnosis from the objective phenomena of ulcer of the intestine*. The diagnosis gains in certainty, if the ætiological side of the ulcer formation is considered. It should therefore become a rule, that under no circumstances should we content ourselves in determining the probability of an ulcer of the intestine from the morbid phenomena on the part of the bowel, but always to look for the cause of the ulceration in the special case. Only then are we upon a less uncertain ground as regards the diagnosis. From the ætiological standpoint the following varieties of ulcer may be differentiated: catarrhal ulcers, pressure ulcers, peptic and infectious ulcers (typhoid, tuberculous, dysenteric, syphilitic, anthrax ulcers).

Ætiologic Diagnosis.—Primarily in the ætiological differential diagnosis we must separate the peptic and a part of the infectious ulcers, which generally is not difficult.

Peptic Ulcer.—The prototype of peptic ulcer, the *duodenal ulcer*, has a symptomatology varying so much from the clinical picture of the other ulcers of the intestine, that if a diagnosis be at all possible, it can hardly be mistaken for any other variety of ulcer. The ætiological fact that after *burns of the skin*, in the digestive tract—from the stomach to the lower portion of the intestines—ulcers may form, and that the ulcer formation is incomparably more frequent in the duodenum than in the rest of the intestinal tract should be considered in the diagnosis of a probable ulcer of the duodenum. The development of ulcer after skin burns is, according

to experience, a very rapid one, in from two to fourteen days after a burn, the clinical evidences of a duodenal ulcer may be noted. Another variety of ulcer of the bowel which I also count among the peptic is the *embolic* ulcer —that is, an ulcer that arises from an embolus of arterial twigs due to necrosis and ulceration of small areas of intestinal wall; but this extremely rarely falls into the path of diagnostic possibility. The condition may be surmised, if in the course of an endocarditis or of a septicopyæmia widespread embolic processes are noted, and the symptoms of an ulcer of the intestine suddenly arise, or following an enterorrhagia an embolism may be found in other organs (spleen, kidney).

Thrombotic Neuritis and Amyloid Ulcers.—In connection with embolic intestinal ulcers a brief mention of ulcers of the bowel occurring in the course of *multiple neuritis* will be made, as they also represent a changed condition of the smaller arteries due to thrombosis and also of "*amyloid ulcers.*" These ulcers are rarer in the amyloid mucous membrane of the entire intestinal tract, their origin, in my opinion, is due to an amyloid degeneration of the walls of the smaller intestinal arteries and is the result of a diminished circulation.

Infectious Ulcers.—Among the *infectious* ulcers, the *typhoid* and *anthrax* intestinal ulcers are never the object of special diagnosis.

This is not even the case with *internal anthrax with localization of the anthrax toxine to the mucous membrane of the digestive tract*, as the phenomena of a general infection dominate the clinical picture and entirely subjugate the symptoms of the intestinal ulcer. If there be found, besides bloody stools, suggilations of the mucous membrane of the mouth, as I have seen in several cases of anthrax, especially in a case of purely internal anthrax, the diagnosis of intestinal anthrax is probable; it only attains certainty provided the blood contains the characteristic anthrax bacilli.

Tubercular Ulcers.—In the other forms of infectious ulcers, such as the *tubercular and the syphilitic*, the bowel affection may be so isolated or so prominent that they may become the subject of special diagnosis; in the dysenteric ulcer this is almost always the case. As regards the *tubercular ulcer* of the intestine, in by far the majority of all cases it is a part phenomenon of general tuberculosis, especially of a secondary localization of the infection, after it has spread from the lungs. But cases of *primary tuberculosis* of the intestine also occur, especially in children, so that the diagnosis of tubercular ulcer of the intestine even gains in importance. A diagnosis of tubercular ulcer of the intestines depends upon: continued high fever, emaciation and marked loss of strength, hereditary predisposition, and, as the most important factor, the examination of the stools resulting positively in the finding of many tubercle bacilli. With this the likely diagnosis becomes positive, especially in cases of rare *primary tuberculosis* occurring without preceding pulmonary affection. This occurs principally from the use of meat and milk of tubercular cows. If pulmonary tuberculosis is synchronously present, as is usually the case, the finding of tubercle bacilli in the fæces is not final, as they may have been swallowed with the sputa, and in this manner have found their way into

the intestine, without having given rise to an ulceration. If tubercle bacilli are found regularly and in large quantities, the last-mentioned eventuality is the less likely. In children who are the subject of tuberculosis of the bowel, a variety of so-called *tabes mesenterica*, infiltrated tubercular mesenteric glands may occasionally be felt through the belly wall; this is of importance in the diagnosis. If cicatrization of the tubercular serpiginous intestinal ulcer occur upon a large scale, kinking of the bowel and stenosis with their consequences may arise.

Dysenteric Ulcers.—In addition to the general symptoms of intestinal ulceration there occur in *dysenteric ulcers*: tenesmus, frequent fæcal passages, and above all the characteristic composition of the dejecta which are fæcal at the onset, becoming later in the course of the dysentery more and more mucoid in character or muco-purulent, meat-water-like or hæmorrhagic, and frequently contain the oft-mentioned sago particles; in the later stages, the mucoid secretion becomes rich in leucocytes, that is, it becomes pure pus. Small shreds of tissue are also noted in the stool in simple dysentery; large necrotic shreds that have been separated from the intestinal wall are found in the dejecta in the gangrenous variety of dysentery in which the stool takes on a brown-black colour due to the decomposition of hæmoglobin and has a cadaveric odour. A *chemical* examination of the fæces shows, besides mucin, very great amounts of albumin, the microscope reveals: leucocytes, red blood corpuscles, intestinal epithelium, eventually cast off tissue, from the intestinal wall, and numerous bacteria, also amœbæ. The recognition during the time of an epidemic presents no difficulty. If no epidemic is present, the individual cases of dysentery cannot be differentiated from (not dysenteric) diphtheritic enteritis which shows the same condition of the fæces. Should the dysentery become chronic, then for months there will be bloody, purulent stools, and the condition may now be confounded with syphilitic ulcer of the bowel.

Syphilitic Ulcers.—Syphilis of the bowel only has a clinical interest when it affects the rectum; the very rare syphilitic ulcer of the colon and ileum cannot be diagnosticated as luetic, but in the rectum (and even there rarely) they present several characteristics which confirm their origin as syphilitic. These ulcerations originate partly from primary affections and papules which ulcerate, and partly from gummatous new formations which first arise in the submucosa and which disintegrate and form sinuses; the ulcers then undermine the mucous membrane. By shrinkage, these later develop stenoses of the bowel, which are somewhat characteristic of syphilitic ulcers and may be felt by the finger. They may, however, develop, if a chancre affects the rectal mucous membrane. Besides these primary affections, broad condylomata occur at the anus. I have also seen pointy condylomata. With this there is a marked purulent hæmorrhagic flow and tenesmus, prolapse of the rectal mucous membrane, etc. It is obvious that the diagnosis also depends upon the other symptoms of syphilitic infection, besides the ulcers and the stenosis of the bowel. These syphilitic strictures and ulcers are most common in women; recently a part of these changes have also been attributed to gonorrhœa and its consequences.

Follicular Ulcers.—The diagnosis of *catarrhal ulcers*, to which I also count the *follicular ulcer*, is much more difficult, as they are due to a swelling and necrobiosis of the solitary follicles arising in the course of catarrh; their seat is especially the colon, much more rarely the small intestine. They are characterized, in general, by the symptoms that have been described previously—i. e., by the blood, the mucus, and the pus in the stools—and the probable diagnosis of follicular ulcer is permitted, if during the course of a long-continued, non-healing catarrh of the large intestine, blood and pus are seen in the dejecta. With this the sago-pearl-like particles are also noted, which in this case are actually, at least partially, composed of mucus.

Pressure and Other Rare Ulcer Forms.—*Pressure and traumatic* ulcers, finally, may only be surmised, and then only when the symptoms of ulcer have followed the swallowing of indigestible substances: fruit seeds, pins, pieces of bone, and others, or a long-preceding constipation with the retention of fæcal masses (stercoraceous ulcers). In *leucæmia*, especially in the acute variety, intestinal ulcers have been observed as a result of lymphatic infiltration of the intestinal wall and secondary necrosis. In the course of *scurvy* ulcers are occasionally noted (as a result of hæmorrhages of the intestinal wall), in *gout and uræmia* (probably due to carbonate of ammonia), and as the result of the action of various *poisons*, especially mercury (*toxic intestinal ulcers*), they are seen.

CANCER OF THE INTESTINE

Aside from carcinoma of the rectum, the diagnosis of cancer of the intestines is always difficult. The symptoms which the disease presents are by no means so distinct, and further, the various tumours of the abdomen may simulate cancer of the bowel and thus give rise to mistakes in diagnosis.

Cachexia and pain, which belong to carcinoma of the bowel as well as to every carcinoma, are naturally of no value in the special diagnosis of intestinal cancer. The proper direction is given to the diagnosis only by the changes in the composition of the fæces and the tumour which may be felt in the abdomen.

As *cancer of the rectum*, in comparison with other cancers of the intestines, gives rise to an entirely different pathological picture, it will be practical to discuss rectal cancer separately. We shall concern ourselves then only at this time with the consideration of cancer of the bowel exclusive of rectal cancer.

Stenosis of the Bowel.—The result of the majority of annularly developing carcinomata of the bowel is the collection of fæces and gases above the narrowed area—i. e., the picture of stenosis of the bowel. I shall consider the detailed description of the diagnosis in a chapter later on. The consequence of the stenosis produced by the carcinoma is *obstinate constipation*; this is rarely absent, or the long-continued irritation may occasionally lead to diarrhœa, which may go on for months, as I have seen in the case of a cæcal carcinoma.

Ribbon-Shaped Fæcal Masses.—The form of the fæcal mass is sometimes characterized by ribbon-shaped masses or resembling the dung of sheep, as only small and flat pressed scybalous masses are able to pass the narrow opening. This condition of the fæces is also noted in other affections and

only then has diagnostic significance if normally formed fæcal discharges are not passed in the interval. Besides this change in the shape of the fæces there is noted (aside from the admixture of mucus, etc., from a complicating catarrh of the intestine) a change in the composition of the fæces, which is much more characteristic of cancer—namely, the appearance of *putrefactive foul-smelling dejecta, which contain blood and pus, in rare cases even desquamated portions of the malignant mass.* In three cases of carcinoma of the colon observed by me, several masses the size of a hazelnut were evacuated.

Symptoms Dependent upon the Seat of the Carcinoma.—It is evident that, according to the seat of the cancer which produces stricture, the consequent phenomena must vary in keeping with the portion of the bowel affected. A *duodenal carcinoma* will naturally produce symptoms resembling cancer of the pylorus—i. e., cardialgia, vomiting, dyspepsia, gastrectasis, complete retraction of the abdomen on acccount of absence of any fæcal formation. Similar symptoms may also be caused by carcinoma of the jejunum, i. e., frequent vomiting of bile, dyspeptic manifestations, dilatation of the duodenum and stomach. In contrast to this, *the lower the seat of the cancer is found in the intestine,* the more will the abdomen above be found to be inflated by gas and fæces, and the more unchanged and unaltered will the just-described abnormal constituents of the dejecta: blood, pus, putrid material, etc., be seen.

Perforation of the Carcinomatous Bowel.—The symptoms resulting from the perforation of a carcinomatous bowel vary greatly; under some conditions, however, they serve to localize the position of the carcinoma, as, for instance, in a communication of the carcinomatous transverse colon with the stomach, under which condition fæcal vomiting occurs which may consist of pure fæces or material having a decided fæcal odour. In a perforation of the gut into the bladder, fæcal masses mixed with urine and intestinal gases are passed, etc.

Tumour.—The most important element in the diagnosis of cancer of the intestine is the possibility of palpating a *tumour.* If this most objective sign is absent the diagnosis is impossible; because cases which show cachexia, dull or colic-like pains, appearance of putrefactive material, with blood and pus in the dejecta, with marked irregularity of the stools, supply insufficient data to establish *a positive diagnosis,* even if the constituents of the stool point to malignant disease. The diagnosis only becomes probable after a tumour has been demonstrated in the abdomen.

This tumour is hard, somewhat uneven, round or slightly oval in outline, and may pulsate if it be situated over one of the larger abdominal vessels, and upon percussion gives a dull tympanitic note. With the establishment of a tumour, the difficulty begins, in deciding which organ of the abdomen is the seat of the tumour. To prevent the erroneous conception at the onset that the appearance of stenosis of the bowel, besides a palpable tumour, justifies a diagnosis of carcinoma, I need but emphasize that abdominal tumours of the most varied description may, if they compress the gut by their growth, produce the symptoms of enterostenosis. Therefore, there is nothing else to be done upon demonstrating a tumour than by means of differential diagnosis to exclude other abdominal tumours, in establishing the diagnosis of carcinoma of the gut with certainty.

Differential Diagnosis between Tumours of the Gut and Other Abdominal Tumours.—As the larger portion of the bowels are capable of great locomotion on account of the long mesentery, and the intestinal coils by becoming filled with gases and fæces become approximate and thus form layers one upon the other, *a frequent change in the position of the tumour and the distinctness with which it may be felt, as well as its decided tendency to displacement, are in a measure characteristic in the examination of cancer of the bowel.* This is particularly true of the superior horizontal portion of the duodenum, of the small intestines, of the transverse colon, and of the sigmoid flexure, whereas the remaining portions of the intestines are only partly covered by peritonæum and adhere to the neighbouring cellular tissue during the growth of the tumour, and in this fashion their movability becomes decidedly more limited. *Cancer of the bowel should therefore always be suspected in an abdominal tumour that is characterized by marked movability,* if the other symptoms, especially the condition of the dejecta, favour this view *and other movable tumours of the abdomen can be excluded.*

Cancer of the Pylorus.—The differential diagnosis from carcinoma of the pylorus must first be considered in this respect. The differentiation from *duodenal carcinoma* may be very difficult, in some cases, for which I can vouch, it may be impossible. Jaundice and the presence of the HCl reaction, as a rule, favour cancer of the duodenum; the diagnosis becomes positive, provided the cancer is situated in the lower portion of the duodenum, and with the consequent gastrectasis, profuse vomiting of *bile and active pancreatic* juice occurs. A possibility of mistaking carcinoma of the small intestine for a pylorus carcinoma is also possible, if attention is bestowed alone upon the movability of the tumour. Cancer of the pylorus is sometimes so deeply situated and sinks so low in the abdominal cavity and is at the same time so movable that it may be pushed into almost any region of the abdomen. In case of doubt, the prominence of dyspeptic symptoms, and above all the results obtained by the stomach-tube (the absence of the HCl reaction and the presence of large amounts of lactic acid in the stomach contents), and a dilatation of the stomach with vomiting at regular intervals, are all decidedly in favour of carcinoma of the stomach.

Movable Kidney and Spleen.—Further, there are to be considered in a differentio-diagnostic aspect: *corset lobe* of the liver, whose connection with the liver, however, may be readily determined by palpation, by following the contour of the border of the liver; *movable spleen and kidneys*, but the shape of these organs is so characteristic, as a rule, that mistakes are hardly possible, as on frequent percussion the normal position of these organs would give a tympanitic note if spleen or kidney were displaced.

Fæcal Masses.—The differentiation of cancer of the bowel from *fæcal tumours* is much more difficult. Mistakes are all the more liable to occur, as, besides the cancer, there is, over and around the malignant mass, a fæcal accumulation. If it be a question of old hardened fæces, they have precisely the same consistence as carcinomatous masses; but then even hardened fæcal masses, and this is especially true of more recent ones, may be pressed

flat between the palpating fingers; they also convey to the finger the sensation of a softer, more doughy mass.

In carrying out this manœuvre of palpation, it should never be done forcibly, even after the mass has been positively recognised as fæcal, for it must not be forgotten that a portion may be carcinomatous besides. Usually purgatives will be of assistance in this case, and continued irrigations will soon clear up the diagnosis. Nevertheless, I must emphasize the fact that, according to my experience, even after the action of purgatives and when the passage has become open, *without the disappearance of the tumour*, the tumour nevertheless may be nothing but a simple fæcal mass, as a softening may take place in the central part and the compact particles may remain inclosed in the walls of the bowel. The more frequently the physician has the opportunity of palpating fæcal tumours the more certain his opinion becomes, and I can only give the advice that no opportunity should be neglected to acquire the necessary practice in the palpation of fæcal tumours.

If the tumour in question is not at all or only slightly movable, the decision of the question arises, whether there be a tumour of the lower part of the duodenum, or of the cæcum, of the ascending or descending colon (other parts of the bowel only provided secondary adhesions have taken place with immovable organs), and further, whether or not a fæcal tumour is present in this immovable portion of the intestine, carcinoma of the kidney, tumour of the mesenteric glands, a sacculated peritoneal exudate, or even an ovarian tumour may now be mistaken for a cancer of the bowel.

Tumour of the Kidney.—Tumours of the kidney and mesenteric glands [1] develop *behind* the bowel, and only when they have attained great size may they be felt under the belly wall. In their growth they push aside the intestines in so far as the movability of the bowel permits; the ascending and descending colon will occupy a position over and beside the tumour as a wide band, and upon percussion the colon will give a loud tympanitic note. In my experience the symptoms of stenosis of the bowel are absent even when the renal tumour has attained great development, in contrast to the condition in carcinoma of the intestine in which a narrowing of the lumen is certain to occur as soon as the tumour has reached greater dimensions.

Sacculated Peritoneal Exudates.—The diagnosis is even more difficult between sacculated peritoneal exudates and cancer of the bowel; especially the remains of a perityphlitis may be mistaken for cancer. Should these conditions occur in a case in which the development of the tumour is not known, especially in an aged patient, and in which the peritoneal masses of exudate have become hardened, the *diagnosis may be simply impossible*, unless the presence of blood in the fæces and the growth of the tumour without giving rise to new inflammatory phenomena make the presence of cancer of the bowel likely. Occasionally the diagnosis is aided by the application of poultices; the chronic peritoneal exudates become smaller under their use, whereas the carcinomatous nodes grow rapidly.

Ovarian Tumours.—Small *ovarian tumours* may give rise to errors in

[1] Regarding the diagnosis of special tumours of the abdomen, reference will be found in the special chapters treating of these conditions.

diagnosis; mistakes may be avoided by remembering the round, ball-like tumour of the ovary, it being deep-seated and demonstrating its connection with the genital apparatus.

Determination of the Portion of Bowel Implicated by the Tumour.—As a rule it is not difficult to determine which *particular part of the bowel is implicated by the more or less immovable tumour.* Statistics show that carcinoma of the large intestine (exclusive of carcinoma of the rectum) is very much more frequent, about 8–10 times, than carcinoma of the small intestine; we must then always remember that in making a diagnosis of cancer of the small intestine we are diagnosticating a comparatively rare affection. The localization of the tumour is materially assisted by allowing *water to flow into the bowel or by filling the bowel with gas,* under control of inspection and palpation of the abdomen, in this manner it may be determined how high up we are enabled to fill the intestine.

Chronic Infiltration of the Intestinal Wall.—It is worth mentioning that not rarely *chronic inflammatory infiltration of the wall of the intestine* may give rise to mistakes. This is especially true if it occurs in the region of the sigmoid flexure. A flat, uniform, long-drawn-out increase in resistance directly favours chronic inflammatory infiltrations of the intestinal wall; the more frequently these are felt the better they are appreciated as inflammatory thickening of the wall of the intestine, and in my experience they are by no means rare.

Rectal Carcinoma.—While the diagnosis of cancer of the bowel from the duodenum downward frequently gives rise to great difficulty, the diagnosis of *cancer of the rectum* is fortunately all the easier. A simple digital examination almost always is sufficient to determine its presence with certainty. Very rarely is the cancer situated so high up that it cannot be felt by the finger. Should this, however, occur, bimanual examination or the introduction of the hand during chloroform narcosis will establish the diagnosis. In the earlier stages of the development of the carcinoma small hard nodules are felt in the wall of the rectum, over which the mucous membrane cannot be pushed with ease; later the lumen of the rectum forms a hard, funnel-shaped resisting canal narrowing at the top. In other cases the introduced finger reaches a round plug-like mass, more prominent at the lower part and having great resemblance to the vaginal portion of the uterus. The new growth as a rule is very hard, uneven and ulcerated, so that the examining finger is withdrawn covered by blood and putrefactive material.

Causes leading to the Examination of the Rectum for Carcinoma.—Should the patient complain of pain in the rectum during the act of defecation, of tenesmus, of frequent and incomplete evacuation of the fæces, of occasional presence of blood and mucus without fæcal material, of pains in the lumbar region, and of *hæmorrhoids,* it is the duty of the physician to make a digital examination of the rectum. Hæmorrhoids develop as the result of the difficulty with which the venous blood returns from the wall of the rectum, partly due to the new growth and partly to the engorgement and the pressure of the fæces over the narrowed area. *But rarely are hæmorrhoids absent in toto* in carcinoma of the rectum, even not then if the other symptoms are but ill defined. *For a long time, therefore, I followed the rule that in every case in which hæmorrhoids were present to make a rectal examination for cancer.* In this manner I have been able to discover many carcinomata of the rec-

tum in which the symptoms were latent. In rare cases *sciatica*, strangury, etc., lead to a rectal examination; also a doubtful or well-developed *carcinoma of the liver* should lead to an examination of the rectum. This was taught me recently by a very striking case. Under some circumstances the primary cancer of the rectum is so small *that it gives rise to absolutely no symptoms*, whereas the secondary cancer of the liver attains enormous dimensions, and may so dominate the entire pathological picture that the examination of the rectum is entirely neglected.

Differential Diagnosis in Cancer of the Rectum.—After the tumour of the rectum has been demonstrated, it is our next duty to determine whether it be benign or malignant. On the whole, this is more of a theoretical than a practical question, as in those cases in which a hard tumour is detected in the rectum (occasionally myomata, lipomata, etc., are seen) it is almost exclusively carcinomatous.

Rectal Polypi.—It is not difficult to distinguish *rectal polypi* from carcinoma. The polypi are mostly seen in children, give rise to diarrhœa and muco-hæmorrhagic discharge and during defecation appear externally, so that they may be noted at the anus, or are torn from their pedicles, and passed with the stool. In contrast to carcinoma, they are smooth and soft; if they have ever been felt, they can scarcely be confounded with cancer.

If the carcinoma of the rectum upon digital examination fails to show the characteristics which are common to it, it is advisable in such cases to excise a small portion of it and subject it to microscopic examination, to determine the nature of the tumour. Finally, it must be mentioned that the rectum is comparatively frequently the seat of carcinoma and that the various portions of the intestines from above downward are increasingly affected by cancer, so that the small intestine scarcely shows 5 per cent, the cæcum and colon 15 per cent, and the rectum fully 50 per cent of all cancers of the bowel—a condition that should be of value in the diagnosis.

[Sarcoma of the intestines is of rare occurrence. It appears in early life, but may be seen at all ages. The sarcoma originates from the submucosa or from the deeper layers of the intestine. The symptoms are the same as in carcinoma.]

[While the results obtained by the X-rays are still doubtful, nevertheless resort should be had to this aid in the diagnosis of all obscure cases. Only an expert, however, should handle the apparatus.]

ENTEROSTENOSIS—INTESTINAL STENOSIS—OCCLUSION OF THE BOWEL—ILEUS

But few diseases require so careful a consideration in their diagnosis on the part of the physician as *enterostenosis*. According to his diagnosis, the physician is called upon to decide regarding a dangerous operation, at times the only remedy which will save the patient's life. Thus the neglect of the operation or its unnecessary performance may in either instance prove fatal! Not that the diagnosis of enterostenosis is so difficult—the symptoms are so conclusive and so characteristic that doubts as to the affection are hardly possible. As a rule, the question to decide is *the situation and the nature of*

the intestinal obstruction, and the complications that have arisen, which require or contraindicate an operation.

Diagnosis of Intestinal Obstruction.—*The establishment of an actual obstruction of the bowel* is then the primary requirement of diagnosis. According to the degree of the obstruction, the symptom-complex will vary from a slight difficulty in the discharge of the fæces up to the severe, dangerous picture of complete intestinal obstruction. *Fæcal retention* occurs suddenly, or its onset may be gradual, as infrequent fæcal discharges may precede the catastrophe.

Ribbon Shape and Other Forms of Fæces.—The fæces may have, for some time previously, appeared *flat pressed* or in the shape of small globular particles, and resembling the fæces of sheep, an arrangement of the fæcal mass that in my experience has but slight diagnostic value. Without obstruction of the bowel the fæces may have this shape, as a result, obviously, of an intermittent strong contraction of the bowel, but only continued for short distances, or, in rare instances, as a result of relaxation and contraction of the sphincter ani.

Results of Obstruction of the Bowel.—The obstruction to the peristaltic action of the contents of the intestine causes an accumulation of fæces and gases above the constricted area, resorption can no longer take place, hence they accumulate. The entire abdomen, or only portions, appear *bloated.* If the large intestine is filled with compact fæcal masses, the contours of the coils of the large intestine appear as hard thick pads, which appear raised compared to the rest of the abdominal parts. The contents of these "pads" at times are hard as stone, tumour-like masses, at times the mass gives a more elastic sensation, which may easily be distinguished from a neoplasm, as the extension of these pads is elongated and shows the shape of greater intestinal tracts; naturally the bowel filled by these masses of fæces gives a dull note on percussion. The patient is anxious, complains of great pain in the abdomen; the bowel is seen in "stormy peristaltic" action, in battle with the obstruction, and the fluid and gaseous contents of the intestine are heard distinctly being moved about above the narrowed area, *whereas no flatus passes below.* If the closure is complete, phenomena occur that are even obvious to laymen as exceedingly dangerous (*Miserere*).

Ileus—Miserere—Fæcal Vomiting.—Distention of the abdomen *ad maximum,* collapse, coldness and cyanosis of the extremities, feeble pulse, eructation of foul-smelling gases are manifest, also *vomiting, which is at first greenish, due to bile, later resembling fæces and having a fæcal odour,* as the thin fæcal masses which have collected at the point of occlusion take the direction of least resistance, then ascend, reach the coils of intestine contiguous to the stomach, finally regurgitate into the stomach, and are partly also drawn into the stomach by the act of vomiting.

Urinary Changes.—*The amount of urine passed is diminished,* and this is all the more marked the higher up the occlusion of the intestine occurs. *The excretion of indican* is increased, especially in occlusion of the small intestine, but this is not the case in obstruction of the large intestine.

According to Fleischer, the increase of indican excretion in the urine in obstruction of the small intestine occurs in the following manner: *Indol,* the mother substance of urinary indican, is formed in the large intestine, as is well known, and

is the product of the putrefaction of albuminous substances. As the chyme which passes from the small intestine into the large intestine under normal conditions contains only comparatively little putrefactive albumin substance, the excretion of indican in the urine is also slight and remains so, provided the large intestine is obstructed. The conditions are, however, changed if the obstruction affects the small intestine! Here a chyme rich in albumin stagnates, which is prepared by the pancreatic juice for putrefaction and the production of indol (compare also p. 222) which produces an increased excretion of indican in the urine.

Both changes of the urine, mentioned above regarding the diagnosis of the position of the occlusion of the gut, act only in general according to the rule just given, and it must not be forgotten that a complicating *peritonitis* may also produce an increase of the indican in the urine. The latter may also give rise to *albuminuria,* but this may be a symptom of enterostenosis, as soon as, owing to the shock, a diminution of blood pressure occurs, and with this a lessening of the urinary secretion gives rise to the appearance of albumin in the urine.

Differential Diagnosis.—The picture of stenosis of the bowel, respectively of occlusion of the gut, is so marked that only in the rarest cases can there be a question of doubt. *Acute poisoning* with vomiting and collapse, *gallstone, renal, and intestinal colics,* the pressure phenomena of compressed *movable kidney,* may at first glance resemble obstruction of the bowel; however, these affections have but a few of the phenomena (the collapse, the vomiting, oliguria and the abdominal pains) in common with occlusion of the gut. As soon as the entire symptom-complex is considered *in toto* and is analyzed, it is impossible to make a mistake.

Perityphlitis.—The condition may more easily be confounded with *acute perityphlitis,* as in this affection there is, also, onset with pain, vomiting, constipation and a palpable tumour. The position of the tumour and the accompanying disturbances of sensation in the right leg, the concentration of the pain to the cæcal region and the fever, all serve to guide the diagnosis in the proper direction. We have already called attention to the fact that a complete ileus may occur during the course of perityphlitis.

Peritonitis.—*Peritonitis* also has some points of resemblance to obstruction of the bowel. The collapse, the tympany and painfulness of the abdomen, and the vomiting are common to both conditions; constipation and an increase of the excretion of indican in the urine also occur in both diseases. But the diffuse tenderness of the abdomen, which is increased by the slightest pressure, *the absence of visible peristaltic waves in the course of the intestines,* lateral dulness of the abdomen, the presence of fever from the onset, pain upon urination and the almost regular absence of fæcal vomiting in peritonitis, are all points of importance in differential diagnosis. Naturally, it must not be forgotten that in the course of obstruction of the bowel a secondary peritonitis arises, and then the symptoms of both conditions are present side by side. Of importance in the differential diagnosis between ileus and peritonitis, finally, is the observation of the development of the disease and of the ætiology, as in the case of peritonitis there will be found a preceding gastric or intestinal affection, and male some affection of the genital apparatus, etc.

The physician must never content himself with a diagnosis of enterostenosis. Rather is it his duty to determine *where* the obstruction is located and its *cause*. The therapy of the affection depends in great part upon the decision of these questions.

Diagnosis of the Position of the Stenosis—Method of Examination.—Regarding the *position* of the obstacle to the forward movement of the contents of the bowel, *the hernial ring should always be first examined in order to determine whether there be a strangulation or not*. A neglect of this precaution on the part of the physician is equivalent to criminal negligence! After the hernial rings have been found unobstructed, an examination of the *rectum* is to be made and, in the case of the female, an examination of the *vagina* also. By means of (especially combined) examination *per vaginam*, changes in position and size of the uterus, ovarian tumours etc., may be determined which may give rise to stenosis of the intestine; *from the rectum*, neoplasms of the large intestine as well as tumours which arise in the bladder or prostate or project into the pelvis, may be detected. An invaginated portion of the bowel may under some circumstances be felt in the rectum, which may even appear externally at the anal opening as a prolapse of the bowel. Besides, not only a positive result, but the fact of finding the rectum empty, as far as the finger can reach, is of importance; and, further, by the introduction of a rectal bougie or, better even, by the inflation of the large intestine by means of air or carbonic-acid gas (which is possible up to the ascending colon) or by means of pouring water into the bowel, while simultaneously percussing the lower intestines, it may be noted whether the latter are uniformly and *rapidly* filled with gas or water, as the case may be, and therefore *below* the obstruction. The quantity of water that will flow into the bowel is also of value in the diagnosis, in determining the seat of the stenosis in the large intestine. For example, if not more than half a litre of water can be introduced after repeated trials, this is in favour of an obstruction of the rectum; and, *vice versa*, if several litres can be poured into the intestine and they are retained, it may be assumed that the stricture is above the rectum, probably above the sigmoid flexure. In the majority of cases this method of examination is sufficient; in some cases it may be advisable to explore the rectum with the entire hand, the patient being under the influence of an anæsthetic.

Inflated Coil of Intestine in Front of the Obstruction.—If the seat of the obstruction or occlusion of the intestine cannot be determined by the method just described, the minutest examination of the abdomen is now to be undertaken. Great attention must be paid to *inspection*, the degree of meteorism and possible intense intestinal movements should be observed, *especially as regards their more decided development in certain regions of the abdomen*. It is specially important, as newer investigations have taught us, to observe the *condition of the intestines lying in front of the stenosed area*. The peristalsis must be determined and it must be investigated whether this is always in the same direction, and only reaches a certain point. Furthermore, the coils of intestine in front of the obstruction are to be observed. In cases of *acute strangulation*, a localized immov-

is the product of the putrefaction of albuminous substances. As the chyme which passes from the small intestine into the large intestine under normal conditions contains only comparatively little putrefactive albumin substance, the excretion of indican in the urine is also slight and remains so, provided the large intestine is obstructed. The conditions are, however, changed if the obstruction affects the small intestine! Here a chyme rich in albumin stagnates, which is prepared by the pancreatic juice for putrefaction and the production of indol (compare also p. 222) which produces an increased excretion of indican in the urine.

Both changes of the urine, mentioned above regarding the diagnosis of the position of the occlusion of the gut, act only in general according to the rule just given, and it must not be forgotten that a complicating *peritonitis* may also produce an increase of the indican in the urine. The latter may also give rise to *albuminuria,* but this may be a symptom of enterostenosis, as soon as, owing to the shock, a diminution of blood pressure occurs, and with this a lessening of the urinary secretion gives rise to the appearance of albumin in the urine.

Differential Diagnosis.—The picture of stenosis of the bowel, respectively of occlusion of the gut, is so marked that only in the rarest cases can there be a question of doubt. *Acute poisoning* with vomiting and collapse, *gallstone, renal, and intestinal colics,* the pressure phenomena of compressed *movable kidney,* may at first glance resemble obstruction of the bowel; however, these affections have but a few of the phenomena (the collapse, the vomiting, oliguria and the abdominal pains) in common with occlusion of the gut. As soon as the entire symptom-complex is considered *in toto* and is analyzed, it is impossible to make a mistake.

Perityphlitis.—The condition may more easily be confounded with *acute perityphlitis,* as in this affection there is, also, onset with pain, vomiting, constipation and a palpable tumour. The position of the tumour and the accompanying disturbances of sensation in the right leg, the concentration of the pain to the cæcal region and the fever, all serve to guide the diagnosis in the proper direction. We have already called attention to the fact that a complete ileus may occur during the course of perityphlitis.

Peritonitis.—*Peritonitis* also has some points of resemblance to obstruction of the bowel. The collapse, the tympany and painfulness of the abdomen, and the vomiting are common to both conditions; constipation and an increase of the excretion of indican in the urine also occur in both diseases. But the diffuse tenderness of the abdomen, which is increased by the slightest pressure, *the absence of visible peristaltic waves in the course of the intestines,* lateral dulness of the abdomen, the presence of fever from the onset, pain upon urination and the almost regular absence of fæcal vomiting in peritonitis, are all points of importance in differential diagnosis. Naturally, it must not be forgotten that in the course of obstruction of the bowel a secondary peritonitis arises, and then the symptoms of both conditions are present side by side. Of importance in the differential diagnosis between ileus and peritonitis, finally, is the observation of the development of the disease and of the ætiology, as in the case of peritonitis there will be found a preceding gastric or intestinal affection, and in the female some affection of the genital apparatus, etc.

The physician must never content himself with a diagnosis of enterostenosis. Rather is it his duty to determine *where* the obstruction is located and its *cause*. The therapy of the affection depends in great part upon the decision of these questions.

Diagnosis of the Position of the Stenosis—Method of Examination.—Regarding the *position* of the obstacle to the forward movement of the contents of the bowel, *the hernial ring should always be first examined in order to determine whether there be a strangulation or not*. A neglect of this precaution on the part of the physician is equivalent to criminal negligence! After the hernial rings have been found unobstructed, an examination of the *rectum* is to be made and, in the case of the female, an examination of the *vagina* also. By means of (especially combined) examination *per vaginam*, changes in position and size of the uterus, ovarian tumours etc., may be determined which may give rise to stenosis of the intestine; *from the rectum*, neoplasms of the large intestine as well as tumours which arise in the bladder or prostate or project into the pelvis, may be detected. An invaginated portion of the bowel may under some circumstances be felt in the rectum, which may even appear externally at the anal opening as a prolapse of the bowel. Besides, not only a positive result, but the fact of finding the rectum empty, as far as the finger can reach, is of importance; and, further, by the introduction of a rectal bougie or, better even, by the inflation of the large intestine by means of air or carbonic-acid gas (which is possible up to the ascending colon) or by means of pouring water into the bowel, while simultaneously percussing the lower intestines, it may be noted whether the latter are uniformly and *rapidly* filled with gas or water, as the case may be, and therefore *below* the obstruction. The quantity of water that will flow into the bowel is also of value in the diagnosis, in determining the seat of the stenosis in the large intestine. For example, if not more than half a litre of water can be introduced after repeated trials, this is in favour of an obstruction of the rectum; and, *vice versa*, if several litres can be poured into the intestine and they are retained, it may be assumed that the stricture is above the rectum, probably above the sigmoid flexure. In the majority of cases this method of examination is sufficient; in some cases it may be advisable to explore the rectum with the entire hand, the patient being under the influence of an anæsthetic.

Inflated Coil of Intestine in Front of the Obstruction.—If the seat of the obstruction or occlusion of the intestine cannot be determined by the method just described, the minutest examination of the abdomen is now to be undertaken. Great attention must be paid to *inspection*, the degree of meteorism and possible intense intestinal movements should be observed, *especially as regards their more decided development in certain regions of the abdomen*. It is specially important, as newer investigations have taught us, to observe the *condition of the intestines lying in front of the stenosed area*. The peristalsis must be determined and it must be investigated whether this is always in the same direction, and only reaches a certain point. Furthermore, the coils of intestine in front of the obstruction are to be observed. In cases of *acute strangulation*, a localized immov-

able distention of the intestines without peristalsis occurs, even after a brief period, in the region of the incarcerated intestinal coil (" *fixed immovable, inflated intestinal coil* "), to which v. Wahl was the first to call attention. The inflated intestinal coil is pressed towards the belly wall, and produces a partial asymmetrical bulging. The inflated wall of the intestine, on account of its increased tension upon percussion, does not give the ordinary intestinal note, but a deep non-tympanitic sound. I emphasize the fact, that the inflated, fixed intestinal coil shows no peristalsis. Before complete rest occurs in the inflated intestinal coil which is situated in front of the obstruction, according to Schlange, peristalsis may also be noted in the tympanitic gut (" *inflated, fixed intestinal coil with peristalsis* "). This latter behaviour is also a symptom which occurs in acute intestinal occlusion and shows its situation, but it is not (Naunyn) a certain symptom of the existence of a strangulation, as it also takes place in acute ileus without strangulation. This limited, on the whole slight, peristalsis must not be confused with the very strong, marked peristalsis the direction of which may be definitely determined in that it runs towards the obstruction, as usually occurs in chronic stenosis, especially after the wall of the gut has become hypertrophied.

The results of percussion (dulness, high position of the diaphragm, etc.) and the auscultatory proof of a more or less restricted, uniform location of the borborygmi may be utilized in the diagnosis. The composition of the vomit and of the urine must be considered; it is important to investigate whether the vomit contains the unchanged, or at most the biliary, contents of the stomach, or whether fæcal masses are contained in it, whether the amount of urine is scanty or approaches the normal and whether the amount of indican is slightly or markedly increased in quantity. Finally, an attempt should be made, by the use of a salt solution injected into the rectum, to produce an evacuation, a measure which is indicated also from the therapeutic standpoint; attention ought then be given to the character of the stool, whether it be in great quantity, whether the fæcal masses are formed or not, wherefrom there may be concluded, if the picture of the intestinal stenosis has not been changed, that the obstruction is situated high up in the bowel.

Stenosis of the Colon.—*If meteorism is marked and uniformly distributed* over the abdomen and is even found in the lateral regions, if there are to be felt voluminous fæcal masses that may attain the thickness of an arm and give a dull note on percussion, which are unaltered, as regards their size, by injections of water into the rectum, if there be fæcal vomiting and the amount of indican is not increased in the urine, *an obstruction of the lower portion of the colon* is to be thought of, and this is the more likely if the collapse is not very marked and the amount of urine is not especially decreased, which are all symptoms in obstruction of the colon.

In contrast to this! If the meteorism is slight and localized to the epigastrium, the stomach dilated, whereas the lower and lateral portions of the abdomen appear to be retracted, if the meteorism varies with the vomiting, if retraction of the epigastrium occurs subsequently to the vomiting

and injections produce large quantities of fæces without altering the morbid picture, if the vomit varies between being fæcal and non-fæcal in character (due to the presence of the chyme for a longer or shorter interval above the seat of obstruction), *stenosis of the duodenum or the upper portion of the jejunum* may be assumed. In keeping with the rapid occurrence of this dangerous form of intestinal obstruction affecting the upper bowel, is the high grade of collapse and oliguria; the urine may contain large amounts of indican.

Occlusion of the Ileum and Cæcum.—Between these two extreme clinical pictures is found the symptom-complex which denotes *occlusion of the ileum and cæcum*. Here the middle portions of the abdomen are tympanitic, whereas the parts corresponding to the colon are retracted, until a marked extension of the upper intestines causes the latter to overlie the colon and the meteorism then becomes general and uniform. The vomit is fæcal in character, collapse and oliguria soon follow.

By means of the symptoms and signs just mentioned, the position of the intestinal occlusion may be determined with some degree of certainty at least at the onset of the affection.

Complicating Peritonitis.—Later, as has already been indicated, the characteristic symptoms are obscured. The diagnosis as to locality of the obstruction becomes uncertain, even impossible, when *peritonitis* occurs. The examination becomes very difficult if the meteorism becomes general owing to peritonitis; if peristalsis ceases, the quantity of urine decreases, the amount of indican excreted increases, and collapse assumes more marked dimensions.

Diagnosis of the Nature of the Stenosis causing Obstruction.—After an opinion of the probable seat of the stenosis has been formed, the question must now be decided, *What is the nature of the obstruction?* This question is often answered even with greater difficulty than that as to the position of the obstruction. In many instances nothing more than a probable diagnosis can be made.

Examination of Vagina, Rectum, and Hernial Rings.—The diagnosis is certain if the obstruction can be felt from the vagina or rectum, therefore, if *tumours* are present which originate from the sexual organs, from the pelvis, or from the rectum, or *intussusceptions, accumulations of fæces* in the rectum, which are palpable as hard masses without the finger encountering an obstruction or a narrowed passage before the obstruction is reached; coprostasis of a marked degree occurs in the aged, as a rule. An incarcerated external hernia is easily detected upon close examination.

Examination of the Abdomen.—If nothing can be detected in an examination of vagina, rectum or hernial rings, which may be the cause of the obstruction, the next to be considered is whether the condition of the abdomen can give any clew to the difficulty.

Tumour in the Abdomen.—The proof of a tumour in the abdomen is of great value in the diagnosis. It must not be forgotten that the fæcal mass which gathers around the tumour may give the hand a sensation similar to that of the tumour causing the obstruction. A neoplasm should be suspected if, in the presence of a tumour in the abdomen, cachexia exists, and irregularities concerning the evacuation of the fæces have preceded the

occlusion of the gut and if the peripheral lymph glands are enlarged. The diagnosis becomes certain if, as occurs in rare cases, small particles of the neoplasm are passed with the fæces.[1] The composition of the fæces is always to be observed, as pieces of an *enterolith* are also occasionally passed in the fæces, as well as *gall-stones*. In case these conditions are found, they might be looked upon as the cause of the obstruction. Obstruction due to gall-stones is likely, provided the symptoms of gall-stone colic have preceded the ileus and jaundice has been an element in the case. In such instances a gall-bladder filled with stones or a hard mass in the bowel may be felt through the abdominal wall or from the rectum, and periods of obstruction to the passage of the fæces may alternate with periods in which the fæces pass.

Intussusception.—Of the tumours which may be felt through the abdominal wall, *intussusceptions* are the ones which most frequently allow of a correct diagnosis, not only on account of the peculiarity of the shape of the tumour, but also on account of some symptoms which are absent in other varieties of intestinal occlusion.

Intussusceptions, which are most frequent in infancy and are ileo-cæcal, as a rule, i. e., invaginations of the ileum and cæcum into the colon, begin suddenly or follow an overloading of the bowels with indigestible foods. There are, *marked colic*, vomiting, collapse, and diarrhœa, the stools being *hæmorrhagic-purulent*, as the intussusception causes venous engorgement; with this there is commonly tenesmus. The invagination as such does not present itself from the onset, as a rule, but only in the course of the ileus as a palpable sausage-like,[2] somewhat coarse, tumour (usually in the region of the sigmoid flexure or of the cæcum). This is at one time more, at other times less, plainly to be felt, especially distinctly and hard when the tumour was palpated for some time, or possibly when violent, spontaneous, colicky pains preceded the examination (Liebermeister); it may migrate downward in the course of the disease and, finally, be felt in the rectum with the finger. Sometimes the invaginated portion of intestine becomes gangrenous and is discharged with the fæces; the anus is patulous. Of importance for the differential diagnosis are, above all, *the cylindrical tumour, the change in consistence and position of the same, and the muco-hæmorrhagic diarrhœa*, as the latter is always rarer in other forms of enterostenosis, at most appearing after incarcerations, in ileus caused by foreign bodies, and in torsion of the axis.

Torsion of the Axis, Formation of Knots and Invagination.—The latter, *torsion of the axis*, also the *formation of knots* and *invagination*, are most difficult to diagnosticate. They are to be thought of if the causes mentioned so far of intestinal occlusion can be excluded in the diagnosis, and if, furthermore, certain points in the pathological picture allow the

[1] Regarding the details of the diagnosis of cancer of the bowel, reference should be had to the chapter treating of this condition.

[2] If a tumour of the bowel causes the intussusception, the form of the swelling may be different from the typical sausage shape; *vice versa*, in simple inflammation of the intestinal wall an elongated tumour may be felt, cylindrical in shape, which may resemble that of intussusception.

assumption, at least, of one or the other form of those intestinal obstructions.

Common to all of them is the *acute course of the ileus with severe collapse, etc., and the fact that the health of the patient was not materially disturbed previously.* As to the objective symptoms, attention may again be called to Wahl's symptom, "*the fixed, motionless, inflated intestinal coil.*" Otherwise, there can be no question of an even approximately certain differential diagnosis between invagination. formation of knots, and torsion of the axis (most frequently around the mesenteric axis at the sigmoid flexure).

Pseudoligaments as Causes of Invagination—Diaphragmatic Herniæ.—*Pseudoligaments*, and with them an anatomical basis for invagination, which is created by them in the abdomen, may be thought of in individuals who have recovered from peritonitides, especially perityphlitis, perimetritis, etc.

Easiest to be diagnosticated of the internal herniæ are *diaphragmatic herniæ*, which are brought about by severe traumatism, although, according to Leichtenstern's compilation, the diagnosis was made correctly in scarcely 2 per cent of the cases. The following symptoms were found in such cases: The affected half of the thorax bulged out, was impeded in respiration, pectoral fremitus was suspended, the respiratory murmur metallic—symptoms which the rare occurrence of diaphragmatic hernia has in common with pneumothorax (see p. 164). However, apart from eventual symptoms of enterostenosis, certain manifestations are directly in favour of hernia: The change of percussion, the sound which, according to the position and fulness of the intestines which are in the thoracic cavity, is either tympanitic or dull, the absence of a regular change of a dull and a high percussion note upon change of posture of the patient, and, finally, the metallic intestinal sounds which depend upon peristalsis and easily betray their origin in the intestines.

Nervous Ileus.—Finally, it may be emphasized that cases of ileus are observed occasionally in which neither laparotomy nor autopsy will show any anatomical changes in the intestines, or even a simple peritonitis. Ileus in such cases is not brought about upon an anatomico-mechanical, but upon a nervous, basis (*"dynamic" paralytic ileus*).

Paralytic Ileus.—It is to be assumed in such cases that certain portions of the intestines become paralyzed, cease to pass the contents of the intestines on, and become inflated by meteorism, thus possibly causing pressure and stenosis of adjacent, filled, descending intestines; this process, and especially the insufficiency of the intestinal muscular activity, may be the cause of the ileus formation. Such a nervous paralytic ileus is to be thought of if ileus symptoms develop in the course of peritonitis or in persons who have suffered from shock in severe operations, or who even have become exhausted by physical and mental overexertion, or are highly nervous and hysterical. This nervous ileus has been observed to occur under such circumstances, but its diagnosis should only be made if none of the above-discussed anatomico-mechanical causes can be found for the occurrence of ileus in the given case.

Spastic Ileus.—Ileus may also, according to my opinion, be brought about (*spastic ileus*) by intestinal spasms, although very rarely, the same as by paralysis. I have recently observed such a case in a hysterical woman. The patient suffered from nervous vomiting, with good stomach digestion and moderate HCl contents of the gastric juice. It was observed one noon that the yellowish-gray vomit, which at other times presented the usual appearance of the stomach contents, had a decidedly fæcal odour. A purge caused repeated stools, but no flatus was observed for two days. Palpation of the abdomen showed a very remarkable condition: The abdomen was not tympanitic, but a hard, cord-like swelling, the size of a little finger, was distinctly felt, which extended over the entire abdomen from the cæcum to the sigmoid flexure and which showed the course and contours of the entire colon and flexures; the rectum did not prove to be contracted upon digital examination. There can scarcely be any doubt of the fact that a spasmodic contraction of the entire large intestine was present in this instance. But, if this is the case, it can-

not be understood why the fæcal vomiting should not be made dependent upon it, because the fæces will take the direction of the least resistance. The contraction of the intestine disappeared after two days; the same condition reappeared about eight or ten days later. But this time the transverse colon was contracted in a cord-like manner, and the patient again vomited masses with an exquisitely fæcal odour on this and the following days.

It is not very easily possible, in my opinion, and not advisable to go further in the differential diagnosis than is about in keeping with the above explanations, because finer-spun "provisional diagnoses" which concern the anatomical cause of intestinal occlusion, are naturally bound to be much oftener wrong than right and, therefore, are of no value.

NERVOUS INTESTINAL AFFECTIONS

The same causes which are active in gastric affections, are also prominent in a very considerable portion of intestinal diseases, that is, they are of a nervous origin, i. e., it is a question, in the various intestinal maladies, of pathological conditions which can be ascribed exclusively to *disturbances of the function of the intestinal nerves,* and in which nothing can be found anatomically that would contradict the assumption of an exclusive affection of the intestinal nervous system. However, we are not yet able to differentiate the various forms of the nervous diseases of the intestines as distinctly as we have recently succeeded in doing with nervous diseases of the stomach. The types to be classified of disturbances of the function of the intestinal nerves would be, according to the presence of an increase or decrease of the reaction of the intestinal nerves, cases of

Increase or decrease of contractility.
Increase or decrease of sensibility.
Increase or decrease of intensity of secretion.

Examples of all three categories can be found in the pathology of the intestines, or indications are found, occasionally at least, that those forms of nervous disturbances of the intestinal activity may occur.

Physiology of the Function of the Intestinal Nerves.—In order better to understand the neuroses of the intestine, it is necessary to give a brief sketch of the influence of the nerves upon intestinal movements. The plexus myentericus, which is situated between the longitudinal and circular muscular layers, is the anatomical centre of movement of the intestine, and as such it essentially guides the intestinal movements. Irritation of the same, and with it peristalsis, is brought about by blood which is poor in oxygen and rich in CO_2, also by direct irritations (which strike the intestinal wall and are thus transmitted to the plexus myentericus), but especially also by the products of decomposition, particularly the acids of the intestinal contents. *If the irritation acts constantly and violently, a state of relaxation, an intestinal parcsis with suspension of peristalsis, occurs.* The paralyzed musculature no longer offers any resistance to the intestinal gases, the intestine will be inflated (meteorism). Of the peripheral nerves, it is the *pneumogastric nerve* which acts upon the intestinal movements in the sense of an increase of peristalsis, but the *splanchnic nerve acts as a nerve of inhibition*—i. e., its irritaton suspends the intestinal movement so long as the blood has not become venous; in the latter case the inhibitory fibres of the splanchnic nerve relax. This nerve is, at the same time, *the sensory* and also *the vasomotor nerve of the intestine,* inasmuch as its irritation causes a stenosis, its severing a distention of the intestinal vessels. There can be no

question that the central nervous system also exerts an influence upon the movements of the intestine, as has been shown by the experimental investigations of Bechterew into the function of the optic thalami, and of J. Pal into the position in the spinal cord of the inhibitory centres for intestinal movements.

MOTOR NEUROSES OF THE INTESTINE

NERVOUS DIARRHŒA

There are pathological conditions of the intestines in which an *increased peristalsis* takes place *upon a purely nervous basis.* The cause of their occurrence should be looked for in the fact that abnormal contents of the intestines exert a mechanical or chemical irritation upon the nervous apparatus of the intestine, without causing an inflammation. Another reason is that the nerves in general, and especially the intestinal nerves, in consequence of neurasthenia or hysteria may be thrown into a more labile state of irritation, so that they are very liable to react upon weak irritations which, otherwise, are not able to call forth a more marked peristalsis. It is characteristic diagnostically of such "*nervous diarrhœas*" that in the former case, in which abnormal irritations affect the nerves of the intestinal wall, the diarrhœa disappears rapidly and abnormal admixtures to the dejections, such as *blood, large quantities of mucus* and others, which point to anatomical changes in the intestinal wall, *are absent.* If, on the other hand, such an increased lability of the nervous reaction can be assumed, diarrhœa can always be easily produced by psychical impressions; sensations of vertigo depending upon the intestine may occur at the same time, or other nervous symptoms may be found in the pathological picture. The fact that diarrhœa in such cases disappears as quickly as its onset was acute, is in favour of an unchanged intestinal mucous membrane.

It must not be forgotten, in judging whether we may conclude upon increased peristalsis from the frequency of the stools in the given case, that the number of regular defecations in the normal individual fluctuates within a wide range—i. e., that some people have normally two, in fact three, movements daily, others again regularly only one every second or third day. Therefore, in individuals of the latter category, with usually very slow irritation of peristalsis, it may be, upon the onset of nervousness, that a defecation which occurs once a day is an abnormal event and the expression of pathological irritability of the intestinal nerves.

Nervous Diarrhœas in Children.—The category of nervous diarrhœas also embraces some cases of *infantile diarrhœas,* especially those which are combined with *eruption of the teeth,* provided no error of diet or some similar fact can be demonstrated as the cause of intestinal catarrh which accidentally accompanies teething in the given case. That diarrhœa, especially, which recurs with every eruption of a tooth, speaks in favour of a nervous diarrhœa, brought about reflexly.

Nervous Diarrhœa in Hysterical and Tabetic Patients.—In adults, it is especially among *hysterical patients* in whom a decided predisposition to nervous diarrhœas exists; they occur in stages of great excitement, to disappear upon the occurrence of more quiet periods. Tabetic patients may also suffer from profuse, uncontrollable diarrhœas, which can unquestionably be explained as "nervous." The following case, which was observed in the Würzburg clinic, may serve as an example:

Case of Nervous Diarrhœa in the Course of Tabes.—The patient was a railroad employé, thirty-four years old, with most pronounced symptoms of tabes dorsalis, with frequent "crises gastriques" (violent spasmodic pains in the gastric region, nausea, vomiting, etc.); he was ordered injections of corrosive sublimate. A vio-

lent gastric crisis occurred on the tenth day of treatment, which lasted not less than ten days, this time—violent gastralgia with good appetite with undisturbed digestion. To this were added, on the eleventh day, *profuse, uncontrollable diarrhœas,* 10 to 40 in twenty-four hours. The fæces were purely watery, *without any trace of mucus.* All kinds of styptics—tannin, opium, naphthaline, etc.—were administered, without success, until the diarrhœas, which considerably debilitated the patient, ceased after a duration of three weeks.

PERISTALTIC UNREST OF THE INTESTINE (TORMINA INTESTINORUM)

This is understood to be an affection of the intestine which is not rare in hysteria and hypochondriasis, in fact, occurring often in nervous individuals in general, manifesting itself in the following manner: A cooing and borborygmus occur in the abdomen, the contents of the intestine are rolled, with a loud noise, from one portion to the other, without an evacuation from the intestine taking place. The patients are greatly molested by this peristaltic unrest, even if they do not suffer from subjective disturbances, such as a sensation of surging to and fro etc., because the loud intestinal sounds are very audible and are noticed by persons in their surroundings; they may also, at times, prevent the patient from sleeping. The intestinal movements may be visible and palpable if the abdominal walls are thin; the small intestine, preferably, is the seat of these "boisterous" peristaltic movements, which in the experiment were described as "rolling movements." No diarrhœa occurs, as the peristaltic unrest is restricted to the small intestine; on the contrary, the stools are mostly obstinately retarded in the affected persons.

ENTEROSPASM

This designates a *spasmodic contraction of the longitudinal and circular musculature of the intestine at the same time,* which occurs in paroxysms, while normally the contraction of the longitudinal and circular muscles occurs alternately, and upon this fact depends the occurrence of peristalsis. The effect of the spasm is the narrowing of the intestinal lumen, which may lead to a disappearance of the same; it may be distributed over the greatest part of the intestine or limited to small portions of the same, thus causing varying clinical pictures. Enterospasm occurs as a symptom in intestinal affections, especially in acute enteritis; furthermore, in meningitis and in some cerebral diseases, as neurosis in the form of lead colic and also in hysteria and neurasthenia. If the contraction of the intestine is more or less diffusely spastic, the abdomen is *retracted in a "scaphoid" manner*; if the enterospasm affects only isolated portions of the intestine, we see retracted areas of the abdomen besides bulged out portions, which latter are caused by accumulated fæces and gas, and by the relaxation of the intestinal portion above the spastic stenosis. The natural consequence of enterospasm is constipation, from which the affected individuals suffer. It may even happen in very rare cases that spastic stenoses of the intestine cause fæcal vomiting (see page 337), as I have seen in a very remarkable case of hysteria in which the entire large intestine could be felt as a hard tube, as thick as a finger (*spastic ileus*). If fæces are evacuated through the intestine after the enterospasm has lasted for some

time, they appear in the form of ribbon-shaped or lead-pencil-shaped masses which are characteristic of spasm when enterostenoses which are based upon anatomical changes can be excluded in the affected patients. Enterospasm is usually associated with intestinal pains (colic), probably in most cases caused by the irritation of the intramuscular sensory nerves.

Spasm of the Sphincter Ani.—The morbid increase of contractility in the *rectum* manifests itself in a picture which corresponds to the special condition of the musculature and innervation of this portion of the intestine, and which deviates materially from that which we have described above.

The fæces which enter the rectum excite the sensory nerves of the rectum, and, with it, a reflex contraction of the sphincters which is transmitted by the ano-spinal centre of the lumbar marrow. But this contraction is retarded by inhibitory fibres which may be innervated from the cerebrum or by the will (the tracts of the inhibitory apparatus, probably, extend from the optic thalami through the pedunculus cerebri and the spinal marrow to the lumbar marrow), so that the fæcal column passes through the anus without effecting a reflex closure of the sphincter. The external sphincter ani is innervated directly from the cerebrum.

Increase of irritability, or irritability of the motor and sensory nerves of the rectum, and also the reduction or cessation of the innervation of the inhibitory apparatus will be followed by *spasmodic closure of the anus*, and we may at least try in a given case of "*proctospasm*" to decide which nerve-tracts participate principally in the occurrence of a spasm of the sphincters. Proctospasm is seen in hæmorrhoids, fissures of the rectum, proctitis, affections of the bladder, of the uterus, etc., or, as a symptom of affections of the central nervous system, in tabes dorsalis and hysteria. The diagnosis does not offer any difficulties, because the spasmodic pains occurring during the attacks are restricted to the anal region, they impede defecation or render it entirely impossible. A digital examination cannot be made, because the finger cannot enter the spasmodically contracted anus.

MOTOR NEUROSES OF A DEPRESSIVE CHARACTER

Nervous Constipation—Intestinal Atony.—More frequently than conditions of increased peristalsis we see a *decrease of peristalsis with obstinate constipation and distention of the abdomen by the intestinal gases (atony of the intestine)*, which depends upon a nervous origin; this affection is most frequently found in hysteria. The fluid masses which fill the balloon-like, distended intestines, may sometimes be moved from one place to another with splashing sounds. The meteorism disappears as rapidly as it came, upon evacuation of these, generally odourless, gases. Obstinate constipation is also found in diseases of the central nervous system, in affections of the spinal cord and of the brain, and particularly in basilar meningitis.

Constipation—Conditions of Engorgement.—*Conditions of engorgement* with accumulations of CO_2 in the blood should, therefore, according to what we stated regarding the reaction of the plexus myentericus upon blood poor in oxygen, be accompanied with increased peristalsis, i. e., with nerv-

ous diarrhœa. The contrary is actually the case. This is easily comprehensible, as the physiological experiment proves, and as has been previously stated, that *permanent* stasis of the blood in the intestinal vessels is followed by a superirritation and, with it, by relaxation of the reaction of the nerves, paresis of the intestines. Chronic constipation in uncompensated cardiac defects, in emphysema, etc., can be explained in such a manner. On the other hand, *a diet which is different from the usual food* may cause nervous constipation in so far as it exerts too small an irritation upon the nervous apparatus of the intestinal wall. We notice that this occurs also upon adherence to too uniform a diet, because this produces a gradual dulling of the irritability of the intestinal nerves. The effect of certain foods and medicines can be explained in a similar manner.

Characteristic of the nervous character of constipation, besides the above-named ætiological factors, is a sudden cessation of constipation and the absence of abnormal admixtures to the stools, especially of mucous masses, which in the long run are never missed upon macroscopic and microscopic examination in chronic intestinal catarrh which is usually accompanied with constipation.

Paralysis of the Sphincter.—A paralysis of the motor *nerves of the rectum* is a frequent occurrence. The patients are not able to retain the fæces, owing to deficient closure of the sphincters. They are bound to yield to the pressure and defecate upon the slightest straining—i. e., coughing, sneezing, laughing, urination, etc. In more marked degrees of paralysis, in which all influence of the will power upon the sphincter ani has been lost, the anus is patulous and fæces are continually voided involuntarily. This is principally the case in diseases of the spinal marrow, but also occurs in disturbances of the activity of the brain.

SENSORY NEUROSES OF THE INTESTINES

Movements of the intestine which take place quietly are not observed normally; the sensations of movement of the ingesta, accompanied with slight tugging in the intestine, etc., which occur in hysteria and hypochondriasis and other nervous affections, might possibly be ascribed to hyperæsthesia of the intestinal nerves. Only a more marked or even spasmodic contraction of the intestinal musculature manifests itself in a more or less painful sensation (*colic*). The splanchnic nerve is acknowledged by physiologists to be the sensory nerve of the intestine. The contraction of the intestinal musculature occurs usually in association with irritation of the sensory fibres, i. e., spasm and colic are simultaneously present, as we noted in the discussion of enterospasm.

ENTERALGIA, MESENTERIC NEURALGIA, "COLICA NERVOSA"

Enteralgia.—A genuine neuralgia is also observed, besides the above-named colicky pains which are caused by tetanic contraction of the intestinal musculature, in the region of the sensory nerves of the intestine, namely enteralgia.

The neuralgic reaction of the sensory nerves of the intestine manifests itself in violent abdominal pains, which are usually of a tearing, pinching, or cutting character, and which are sometimes so intense that the patient is seen bent, with cold

extremities and a small pulse. The abdominal walls are either retracted, if a spasmodic contraction of the muscles of the intestines is jointly present with irritation of the sensory nerves, or they are, on the contrary, distended. The musculature of the abdominal wall also usually participates in the spasm which then manifests itself in hardness and tension of the abdominal walls. As accessory symptoms in the picture of enteralgia we observe palpitation of the heart and sensation of oppression, vomiting, hiccough and desire to urinate; sometimes even general convulsions accompany the neuralgic pain of the intestines. Enteralgia is characterized as neuralgia, partly by the periodic course and the sudden cessation of the pains and partly by its ætiology. It can be determined in the given case that either a very abnormal condition of the irritations which strike the intestinal wall is present, thus affecting neuralgically the sensory nerves, or that an abnormal condition and ability of reaction of the intestinal nerves proper exists, or that both conditions are simultaneously present. Of the former I name helminthes (intestinal worms), foreign bodies (gall-stones, etc.), *cold* of the external skin, and, above all, *lead intoxication*, rarely arthritis, etc. An abnormal condition of the intestinal nerves and their reaction as cause of the colic may be supposed in hysteria, in the colics occurring in spinal-cord diseases, in "reflex colic" due to diseases of the liver, kidneys, uterus, ovaries, etc.

Differential Diagnosis.—The diagnosis of enteralgia is founded upon the marked pathological picture, the ætiological factors and, especially, upon the possibility of excluding pathologico-anatomical changes in the abdomen which also cause among their *symptoms* colicky pains.

Exclusion of Inflammatory and Ulcerative Processes in the Intestines.—In the latter respect it is necessary, above all, to differentiate abdominal pains, accompanied with *inflammatory or ulcerative processes*, from purely nervous colics (enteralgia) which, according to our diagnostic description of these affections, should not be difficult. It is true that the pains in enteralgia are often decreased by pressure upon the abdomen, and usually increased with the inflammatory changes and ulcerative processes in the intestines; but this differentio-diagnostic symptom is not always reliable, both in a positive and in a negative sense.

Peritonitis.—On the other hand, the pain is surely never lessened by pressure upon the abdomen in *peritonitis*, which is sometimes to be considered diagnostically, especially when the colic is associated with meteorism.

Gall-Stone and Kidney-Stone Colic.—A confusion with *gall-stone colic* or *renal stone colic* is prevented by the concentration of the pains upon their points of origin in these affections, as well as by the other symptom-complex of the same, to which we cannot refer on this occasion. A confusion of enteralgia with *rheumatism of the abdominal muscles* can only occur upon a very superficial examination. The painfulness of the abdominal muscles when held between the fingers, the intensity of the pains upon any pressure or movement, and their rapid disappearance upon the administration of antirheumatic remedies characterizes the rheumatic affection of the abdominal muscles so well that the diagnosis can always be made with certainty.

Diagnosis of the Various Forms of Enteralgia.—After the diagnosis of a purely nervous colic has been determined, the question remains which *special form of enteralgia* prevails. If it commences with vomiting and pains in the epigastrium, "*a colica ab ingestis*" is to be thought of, and the vomited masses should be examined

accordingly. We should also look for intestinal worms, especially tapeworm fragments and ova in the stools, etc. *Hysterical colic* can usually be recognised easily by the entire conduct of the patient, by the simultaneous presence of spasms, etc.

Hysteric Hyperæsthesia of the Abdominal Wall.—It occurs often, according to my experience, that in hysteria a *hyperæsthesia of the abdominal wall* is present besides enteralgia, so that the question of an initial peritonitis suggests itself. However, the very facts that the pressure upon folds in the skin is very painful and that deep pressure is not felt more than superficial pressure, protect us from a wrong diagnosis in such cases.

The above statement shows sufficiently the importance of the fact always to inquire into the ætiology of the case when making the diagnosis of enteralgia. To mention an example, think of the consequences if a physician fails to investigate the possibility of lead intoxication in a case which presents a purely nervous colic. Moreover, the morbid picture—the retraction and tension of the abdominal walls, the decrease of diuresis, the hard and slow pulse, the bluish-gray border at the gums (paralyses are usually absent at this stage) is so characteristic that the mistaking of lead colic is always a very grave technical error.

Neuroses of Sensibility of the Rectum.—It is not possible to state which *portion* of the intestine is affected by the colic, but this is more of theoretical than of practical importance. Only the *neuroses of the sensibility of the rectum* present—like the motor neuroses—a characteristic picture which is so different from that of other colics that their special diagnosis is possible. The pain in these cases is localized in the lowest abdominal and sacral regions, is associated with a violent desire to defecate, i. e., with pressing sensations in the rectum. It is a question of a neuralgia of the sympathetic nerve fibres of the plexus hæmorrhoidalis, which are disseminated in the lowest part of the rectum. The affection is found principally in individuals suffering from hæmorrhoids (hæmorrhoidal colic) and in nervous women. Indications of rectal neuralgias are also usually seen in tabes; if we ask these patients regularly, they almost always complain of pressure in the rectum, they have the sensation of "a wedge in the anus," etc. If the pains in the rectum are more violent, they radiate to the perineum and to the genitalia.

Intestinal Vertigo.—It is probably due to an increased irritability of certain nerve tracts, which are in connection with intestinal nerves and which transmit the sensation of vertigo, that some patients with intestinal diseases suffer from *vertigo*. I have treated a patient in whom the passage of the fæcal column caused vertigo, and in whom it was possible to produce the latter sensation artificially by a digital examination of the rectum as well.

SENSORY NEUROSIS OF A DEPRESSIVE CHARACTER

Conditions of Decrease of Sensibility.—The occurrence of a *decrease of sensibility of intestinal nerves* may be *a priori* assumed as certain, but it cannot be diagnosticated because the intestinal nerves do not normally transmit perceptible sensations. Only the sensory nerves of the rectum are an exception to this rule, also; their paralysis can be diagnosticated as such.

The irritation of the sensory rectal nerves by the fæces entering the rectum causes the sensation of the desire to defecate, which is connected with the irritation of the sphincter ani already mentioned. This sensation of the desire to defecate ceases in some patients, especially in those with diseases of the spinal cord, owing

to the *anæsthesia of the rectum*, so that they have no desire to defecate and do not feel the passage of the fæcal column through the anus. In the higher degrees of the disease the reflex closure of the sphincter ceases also; if, then, the voluntary contracture of the external sphincter is suspended, involuntary defecation occurs, the onset of which is not noticed by the patients until the fæcal odour, or the soiling of the legs, or of the bed, etc., calls their attention to it.

NEUROSES OF SECRETION OF THE INTESTINE

It is well known that the neuroses of secretion have attained great importance in the realm of gastric diseases. It is possible that anomalies of intestinal juice secretion, which rest upon a nervous basis, play a similar independent *rôle* in the pathology of the intestines. It may be that certain "nervous diarrhœas," especially the watery diarrhœas of hysterical patients, depend upon such increased intensity of the intestinal juice secretion, and, *vice versa*, nervous constipation might be referred, at least in part, to a stagnation of the secretion of intestinal juice under nervous influence. The future will tell us how far we are entitled to this premise in the above-named condition. It is probable that an action of the nerves exists upon the secretion of intestinal juice, analogous to other secretions which are subject to nervous influence; some physiological experiences are in favour of this assumption—for instance, increase of intestinal juice after injection of pilocarpine, which acts upon secretory nerves in general. The evacuation of the well-known mucous cylinders in hysteria is, in my opinion, not entirely the result of enteritis but, as stated previously, principally due to a nervous anomaly of the large intestine.

HELMINTHIASIS—INTESTINAL WORMS

The positive *diagnosis of helminthiasis*, tapeworms (tænia solium and mediocanellata, bothriocephalus latus) and of the roundworms (ascaris lumbricoides, oxyuris vermicularis, trichocephalus dispar, anchylostomum duodenale) is based exclusively upon the *direct demonstration of the parasites or their ova in the contents of the intestines*, which can be easily and with certainty accomplished by macroscopical and microscopical examination of the stools. The description of the various worms and their ova does not belong within the sphere of our work. Pathological symptoms which are produced by the presence of helminthes are either entirely absent or are so ambiguous that the diagnosis can never be made from them alone. For completeness' sake it may be mentioned, however, that irregularities of defecation, complaints of pressure and pains in the abdomen, sometimes also an actual enteralgia, which cannot otherwise be traced ætiologically, and, above all, general symptoms, such as bulimia, salivation, vertigo, spasms, etc., may give rise to the assumption that a tapeworm is present; this also holds good of ascarides.

Of diagnostic significance is *pernicious anæmia*, which is caused by the presence of ankylostomices in the duodenum and small intestine in such a manner that the worms suck themselves fast to the wall of the intestine and withdraw blood from their host. In analyzing severe, apparently "essential" anæmias, it is necessary, therefore, always to think of ankylostomices as their cause, especially as it has recently been found that the dissemination is much larger than was supposed formerly, when it was considered an endemic affection restricted to the Orient, especially Egypt. After it had become wider known by the cases which occurred in greater frequency among the labourers at the Gotthardt tunnel, it has also been found occasionally in various countries among brickmakers and other workmen.

mostly very small in such cases, unless albuminuria is present, besides pyuria, due to other causes. However, the albumin excreted in the urine upon the albumin test should be considered as dependent upon the admixture of pus with the urine, only when it does not amount to more than one tenth of the volume of the sample of urine. In the latter case *many pus corpuscles* will always be encountered *in the sediment*. However, the most important support for the diagnosis in deciding this question is an examination of the sediment for urinary casts, the occurrence of which in the urine points decidedly to the fact that the excretion of albumin should also be referred to circulatory disturbances or to tissue changes in the *kidneys*. The presence of *scanty* pus cells and casts, on the other hand, does not allow of any diagnostic conclusion of pyuria, because an admixture of leucocytes to the urine is a very common occurrence in inflammations of the kidneys.

It has been determined beyond doubt that other albuminous substances are also excreted in the urine besides serum albumin; but as yet it is not possible to draw *positive* diagnostic conclusions from the occurrence of globulin, peptones, hemialbumose, etc., as to the existence of certain affections of the urinary organs.

DISEASES OF THE KIDNEYS

HYPERÆMIA OF THE KIDNEYS—CONGESTED KIDNEY

The diagnosis of congestion of the kidney and its segregation from the nephritides is usually easy and of eminent practical importance, because, according to the result of the diagnosis in this respect, prognosis and therapy of the individual case will be of an entirely different character. The symptoms of a disturbance of the circulation in the kidneys due to engorgement are very characteristic, and their occurrence, fortunately, is elucidated experimentally so that we are able, not only to explain their causes correctly, but, as a rule, also to diminish the sequence of renal congestion by proper therapeutic measures, taken in accordance with the experience gained by experiment, sometimes even to cause their total disappearance.

Physiology.—If the renal vein is partially ligated, there occurs at once a decrease in the amount of urine, and albumin appears in the scanty renal excretion. Both these facts are explained by the *decreased velocity of the current in the glomeruli* and the reduction depending thereupon of the function of the glomerular epithelia. Besides, the conditions in which engorgement of the renal veins occurs are, almost without exception, associated with a weak action of the heart. In such cases, therefore, with the diminished velocity of the current, there goes hand in hand a *decrease of the blood pressure* in the glomerulus, so that in this respect, too, a diminution of diuresis can be accounted for.

Composition of the Urine.—The urine which is secreted during an engorgement hyperæmia of the kidney shows a condition in keeping with the above-named experimental facts. *The volume for the twenty-four hours is diminished* (1,000 cc. and less), the *colour* is dark red, the *specific gravity high*, because the quantity of water has been disproportionately reduced in comparison to the solids excreted; the salts, especially the uric-acid salts, form a reddish precipitate in the cooled urine. The latter contains *albumin* and *urinary casts*. These are simple, *hyaline* casts so long as no inflammatory processes set in besides the engorgement. *Blood* is almost with-

out exception *absent* in simple congestion, apart from isolated blood corpuscles in the sediment, even if the engorgement reaches high grades; *if blood can be demonstrated in an undoubtedly existing engorgement, this almost always points to a complication*, to the simultaneous development of a nephritis or of a hæmorrhagic infarct.

It appears that the excretion of the solids of the urine is not *materially* disturbed; *at any rate, uræmia will never occur in simple engorgement.* This clinical fact, which is in certain contradiction to the circumstance that the secretion of urinary constituents is temporarily diminished in engorgement, may possibly be explained in the following manner: As we see that the renal epithelia in such conditions are able rapidly to reassume their full activity upon an improvement of the circulatory conditions, that they are, therefore, only *functionally* injured, we may have a right to assume that at times, during which a more considerable retention of excrementitious elements threatens to occur, the renal epithelia are able to display a greater activity and to cause a more profuse compensatory excretion.

The quantities of albumin in the urine are always only moderate, especially if the small quantity of urine in congested kidney is taken into consideration. It is of greater diagnostic importance than the relatively insignificant quantity of albumin, *that the latter, corresponding to the fact that albuminuria is dependent upon the energy of the heart, varies considerably with the present intensity of the latter.* Improvement of the cardiac activity by means of excitants (especially by digitalis) often causes the urine to become free from albumin in the course of a few days; this is the most marked symptom of engorged kidney. But we are not always able to cause the albumin to disappear; in such cases the diagnosis of congested kidney becomes doubtful, and a diagnostic reflection in various directions is necessary.

Dropsy.—The condition of the dropsy, is the first thing which should be observed. It always commences in those cases, in which it is a question of engorgement in the kidneys due to non-compensated affection of the heart or of the lungs, in the *lower extremities*, and is here more stationary than in nephritis, although in this affection, too, the laws of gravity very frequently co-operate regarding the localization of the œdema.

Condition of the Heart.—It goes without saying, furthermore, that *the heart and the lungs* are to be carefully examined and the condition of the pulse should be investigated. If emphysema is found or cardiac murmurs are present, it points at once to the presence of congested kidney. The demonstration of a simple hypertrophy of the heart without defects of the valves generally is more in favour of nephritis; but in the later stages of idiopathic hypertrophy of the heart, too, engorgement may occur and this is also the case in compensating hypertrophy of the heart in the course of nephritis. However, in the latter case, the greater tension of the pulse is still indicated in comparison to the small irregular pulse in an idiopathically hypertrophied heart, the energy of which has relaxed. Strong systolic functional murmurs may also occur in this stage of relaxed energy of a hypertrophied heart in nephritis, and they may give rise to a wrong interpretation of the conditions—i. e., they may cause the assumption of a congestion in the kidneys due to a mitral defect. The

observation of the entire course of the disease, the microscopical examination of the urine for casts and, above all, the ophthalmoscopic examination for albuminuric retinitis should, in such cases, clear the situation.

Other Symptoms.—The diagnosis of congested kidney becomes re-enforced by the demonstration of simultaneously existing manifestations of engorgement in other parts of the body, especially of cyanosis (which is in marked contrast to the pale colour of the skin in nephritis) and, above all, of the hard, distended, easily palpable liver.

Combined Congested Kidney and Nephritis.—If the above-named symptoms of engorgement disappear, owing to proper therapeutic measures, with an increase of the blood pressure; if, at the same time, the quantity of the urine increases and the specific gravity decreases, and the urine becomes clear and light coloured, but if, nevertheless, the albuminuria remains, although to a lesser degree, we should be careful with the diagnosis of simple congested kidney. ***In such cases there exists, besides the engorgement, a nephritis.*** The latter may be a more or less accidental complication, or the *consequence* of the stasis; in the latter case, then, it would be a question of interstitial inflammatory conditions in the kidney, of an engorgement nephritis ("chronic contracted kidney"), which, however, are not at all frequent, according to my experience (compare also p. 2).

CHOLERA KIDNEY, GESTATION KIDNEY

The renal affections which occur in the course of cholera and of gestation are on the border line between changes in the kidneys caused by circulatory disturbances and those which are brought on by inflammation. Common to both is the decrease of diuresis, the albuminuria, the excretion of hyaline casts with fatty epithelia, and the grave alteration of the nervous system (in pregnancy known and dreaded as eclampsia, in the course of cholera as cholera typhoid). Important as the question may be in a pathological respect as to the character of these renal changes (which, irrespective of the incidentally occurring genuine nephritides, are under all circumstances connected with changes in the circulation and an injury depending thereon of the anatomical condition and function of the epithelia), it is of little interest to the diagnostician, because the diagnosis itself never presents any difficulties owing to the unmistakable ætiology of such cases. There may only be a chance for a diagnosis in regard to the prognosis of the individual case.

Kidney in Pregnancy.—The more the condition of the urine during *pregnancy* approaches that in nephritis, therefore, the lower its specific gravity, and the more pronounced the admixture of blood and metamorphosed casts, the more doubtful is the prognosis. And the mere circulatory disturbances, engorgements in the abdominal veins should be probably less considered as the cause of dropsy and albuminuria than the nephritic changes in the kidneys. The dropsy in such cases does not, as in the former instance, attack the lower extremities exclusively or at least preferably, but rapidly extends to the upper portions of the body, or it may occur in these first.

Kidney in Cholera.—The renal changes in *cholera*, the weakness of the heart and the anuria characterize the prognosis. If an improvement occurs in these two factors, a favourable termination may be counted on, espe-

cially because the ischæmia of the kidneys in cholera is only exceedingly rarely followed by chronic nephritis. On the other hand, the longer the anuria persists, the scantier the urine voided after the resumption of the secretion, and the more profuse quantities of albumin it presents, the more certain should a lethal termination of the affection be expected (compare also Cholera).

DIFFUSE NEPHRITIS, BRIGHT'S DISEASE

Nomenclature of the Various Forms of Nephritis.—According to the precedence of Cohnheim we may divide the various forms of nephritis from a clinical standpoint, according to their rapid or slow course, into *two main classes*, into *acute* and *chronic nephritides*, and separate from the latter, as a distinct form of chronic nephritis, the "genuine *atrophied kidney*," in which the exquisitely focal, very slowly progressing, interstitial processes of inflammation from the onset, predominate over the parenchymatous changes, and these latter are restricted to the areas of induration, while (which is of importance for the clinical manifestations) more or less extensive *healthy* portions of renal tissue remain between the atrophied areas. A similar atrophy of the organ, however, occurs also in common chronic parenchymatous nephritis, especially in the so-called chronic hæmorrhagic form (the "large, red kidney"), if the affection is of long duration, a variety of atrophied kidney ("secondary atrophied kidney," "small red kidney"), which is anatomically distinguished from the focally indurated nephritis solely by the fact that the degeneration of the parenchyma predominates over the interstitial proliferation. An exact separation from an anatomical standpoint of the various forms of atrophied kidney is often quite difficult, while it is well possible upon observation of the clinical course of both forms of nephritis.

According to the above we distinguish:

I. Acute nephritis ("acute parenchymatous" nephritis).

II. Chronic nephritides:

1. *Chronic nephritis* sens. strict. (subchronic nephritis, "chronic parenchymatous nephritis," chronic diffuse nephritis without induration). Modified form: *Secondary atrophied kidney.*

2. *Atrophied kidney* sens. strict. ("focally indurating nephritis, primary, genuine atrophied kidney). Modified form: *Arteriosclerotic*, atrophied kidney (sclerosis of the kidney).

I find that this classification into acute, chronic nephritis (eventually with termination in secondary atrophied kidney) and (primary) atrophied kidney best answers the clinico-diagnostical requirements. A classification, which is more in accordance with the anatomical arrangement, into large white kidney, large red kidney, small red kidney, smooth atrophied kidney, is not advisable from a clinical standpoint, especially not, because it has also been acknowledged from the anatomical side that it is impossible strictly to separate the various forms of Bright's disease.

ACUTE NEPHRITIS

ACUTE PARENCHYMATOUS NEPHRITIS, ACUTE BRIGHT'S DISEASE

Composition of the Urine.—Quantity.—The symptoms of acute nephritis are so marked that a mistaking of the affection is not very well possible. The most characteristic pathological symptom here as well as in other renal diseases is the *condition of the urine*. The *quantity is small*; the secretion may even cease entirely.

The cause of this condition is to be looked for either in the diminished current velocity and blood pressure in the glomeruli caused by the inflammation or in a swelling and desquamation of the glomerulus epithelia which doubtless participate in the excretion of water.

observation of the entire course of the disease, the microscopical examination of the urine for casts and, above all, the ophthalmoscopic examination for albuminuric retinitis should, in such cases, clear the situation.

Other Symptoms.—The diagnosis of congested kidney becomes re-enforced by the demonstration of simultaneously existing manifestations of engorgement in other parts of the body, especially of cyanosis (which is in marked contrast to the pale colour of the skin in nephritis) and, above all, of the hard, distended, easily palpable liver.

Combined Congested Kidney and Nephritis.—If the above-named symptoms of engorgement disappear, owing to proper therapeutic measures, with an increase of the blood pressure; if, at the same time, the quantity of the urine increases and the specific gravity decreases, and the urine becomes clear and light coloured, but if, nevertheless, the albuminuria remains, although to a lesser degree, we should be careful with the diagnosis of simple congested kidney. *In such cases there exists, besides the engorgement, a nephritis.* The latter may be a more or less accidental complication, or the *consequence* of the stasis; in the latter case, then, it would be a question of interstitial inflammatory conditions in the kidney, of an engorgement nephritis ("chronic contracted kidney"), which, however, are not at all frequent, according to my experience (compare also p. 2).

CHOLERA KIDNEY, GESTATION KIDNEY

The renal affections which occur in the course of cholera and of gestation are on the border line between changes in the kidneys caused by circulatory disturbances and those which are brought on by inflammation. Common to both is the decrease of diuresis, the albuminuria, the excretion of hyaline casts with fatty epithelia, and the grave alteration of the nervous system (in pregnancy known and dreaded as eclampsia, in the course of cholera as cholera typhoid). Important as the question may be in a pathological respect as to the character of these renal changes (which, irrespective of the incidentally occurring genuine nephritides, are under all circumstances connected with changes in the circulation and an injury depending thereon of the anatomical condition and function of the epithelia), it is of little interest to the diagnostician, because the diagnosis itself never presents any difficulties owing to the unmistakable ætiology of such cases. There may only be a chance for a diagnosis in regard to the prognosis of the individual case.

Kidney in Pregnancy.—The more the condition of the urine during *pregnancy* approaches that in nephritis, therefore, the lower its specific gravity, and the more pronounced the admixture of blood and metamorphosed casts, the more doubtful is the prognosis. And the mere circulatory disturbances, engorgements in the abdominal veins should be probably less considered as the cause of dropsy and albuminuria than the nephritic changes in the kidneys. The dropsy in such cases does not, as in the former instance, attack the lower extremities exclusively or at least preferably, but rapidly extends to the upper portions of the body, or it may occur in these first.

Kidney in Cholera.—The renal changes in *cholera*, the weakness of the heart and the anuria characterize the prognosis. If an improvement occurs in these two factors, a favourable termination may be counted on, espe-

cially because the ischæmia of the kidneys in cholera is only exceedingly rarely followed by chronic nephritis. On the other hand, the longer the anuria persists, the scantier the urine voided after the resumption of the secretion, and the more profuse quantities of albumin it presents, the more certain should a lethal termination of the affection be expected (compare also Cholera).

DIFFUSE NEPHRITIS, BRIGHT'S DISEASE

Nomenclature of the Various Forms of Nephritis.—According to the precedence of Cohnheim we may divide the various forms of nephritis from a clinical standpoint, according to their rapid or slow course, into *two main classes*, into *acute* and *chronic nephritides*, and separate from the latter, as a distinct form of chronic nephritis, the "genuine *atrophied kidney*," in which the exquisitely focal, very slowly progressing, interstitial processes of inflammation from the onset, predominate over the parenchymatous changes, and these latter are restricted to the areas of induration, while (which is of importance for the clinical manifestations) more or less extensive *healthy* portions of renal tissue remain between the atrophied areas. A similar atrophy of the organ, however, occurs also in common chronic parenchymatous nephritis, especially in the so-called chronic hæmorrhagic form (the "large, red kidney"), if the affection is of long duration, a variety of atrophied kidney ("secondary atrophied kidney," "small red kidney"), which is anatomically distinguished from the focally indurated nephritis solely by the fact that the degeneration of the parenchyma predominates over the interstitial proliferation. An exact separation from an anatomical standpoint of the various forms of atrophied kidney is often quite difficult, while it is well possible upon observation of the clinical course of both forms of nephritis.

According to the above we distinguish:

I. Acute nephritis ("acute parenchymatous" nephritis).

II. Chronic nephritides:

1. *Chronic nephritis* sens. strict. (subchronic nephritis, "chronic parenchymatous nephritis," chronic diffuse nephritis without induration). Modified form: *Secondary atrophied kidney.*

2. *Atrophied kidney* sens. strict. ("focally indurating nephritis, primary, genuine atrophied kidney). Modified form: *Arteriosclerotic*, atrophied kidney (sclerosis of the kidney).

I find that this classification into acute, chronic nephritis (eventually with termination in secondary atrophied kidney) and (primary) atrophied kidney best answers the clinico-diagnostical requirements. A classification, which is more in accordance with the anatomical arrangement, into large white kidney, large red kidney, small red kidney, smooth atrophied kidney, is not advisable from a clinical standpoint, especially not, because it has also been acknowledged from the anatomical side that it is impossible strictly to separate the various forms of Bright's disease.

ACUTE NEPHRITIS

ACUTE PARENCHYMATOUS NEPHRITIS, ACUTE BRIGHT'S DISEASE

Composition of the Urine.—Quantity.—The symptoms of acute nephritis are so marked that a mistaking of the affection is not very well possible. The most characteristic pathological symptom here as well as in other renal diseases is the *condition of the urine.* The *quantity is small*; the secretion may even cease entirely.

The cause of this condition is to be looked for either in the diminished current velocity and blood pressure in the glomeruli caused by the inflammation or in a swelling and desquamation of the glomerulus epithelia which doubtless participate in the excretion of water.

Diminished Excretion of Urinary Constituents.—The *specific gravity* of the scanty urine is *high*, 10.20 to 10.30; later, when the secretion of urine again becomes more profuse, the specific gravity will become less. This depends as always, so also here, upon the amount of solids, especially of the *urea* in the urine. *The percentage of urea* is, it is true, actually *high in acute nephritis, but the total amount of urea is considerably diminished,* to one sixth of the normal and less. The excretion of *phosphates* and *chlorides* is also reduced, while it is remarkable that the amount of uric acid excreted was not found changed.

Albumin.—The urine always contains albumin, and that usually in quite considerable amounts ($\frac{1}{2}$ to 1 per cent).

According to the observation of several authors, albumin may be entirely absent temporarily. This fact appears to be at least remarkable, because the easier passage of the albumin through the vessel wall is a necessary consequence of the deficient nutrition and the abnormal permeability of the vessel wall which are brought about by the influence of the inflammation. If, nevertheless, urine free from albumin is found in certain exceptional cases—I personally have never observed one—this can be explained only in such a manner that the affected portions of the kidneys absolutely interrupt their water-secreting function temporarily, and the remaining healthy portions assume the secretion of urine independently.

Colour and Transparency of the Urine.—Of especial importance are the changes of the urine in regard to its *colour and transparency.* The colour is pale red, sometimes also *dark red* and always *turbid.* The red colour is caused by the admixture of blood, which appears in larger or smaller amounts, owing to the inflammation of the renal vessels. The turbidity is due to the profuseness of the solids, which were not dissolved in the scanty amount of urine. Upon letting the urine stand, they will precipitate in a more or less considerable *sediment.*

Composition of the Sediment.—The examination of this precipitate yields: red and white blood corpuscles (both combined may form "blood casts"), uric-acid salts, cast-off epithelia of the uriniferouse tubules, but, above all, *urinary casts.*

Casts.—Besides the blood casts named there are found in varying quantity *hyaline, metamorphosed, and, above all, purely epithelial casts*; the latter are a certain sign that the inflammation in the uriniferous tubules has become localized. We may observe in some cases, in which the occurrence of a nephritis may be expected with a certain amount of probability, as in scarlatina, that the excretion of blood corpuscles and casts sometimes precedes the onset of albuminuria for days.

The diagnosis of acute nephritis can be established at once from the above-described condition of the urine, if the latter shows the above changes *in toto*, which, however, is not always the case. It generally occurs that the described changes of the urine are the less pronounced the milder the form of the nephritis in the individual case, respectively the more the affection approaches a favourable termination in the graver forms.

Secondary Symptoms of Acute Nephritis—Fever.—In contradistinction to the above, other symptoms of nephritis are of minor diagnostic significance, because they may be prominently developed in some cases and be very distinct, while in

other instances they may be entirely absent—e. g., the pain, the desire to urinate, and the fever. The latter is usually caused by the original disease which produces the nephritis, pneumonia, scarlatina, sepsis, etc. But here, too, fever is rarely observed under such circumstances, because nephritis occurs in the last stages of those febrile infectious diseases, at a time when the toxiferous infectious material is eliminated from the body by the kidneys. However, it is certain, in my experience, that cases of febrile nephritis occur without preceding infectious diseases, but such cases are very rare. *The course of acute nephritis, almost without exception, is afebrile*; for instance, I did not meet with a single case of febrile nephritis for an entire decade, until recently several successive cases came under treatment.

Dropsy.—The most important and most frequent symptom of acute nephritis besides the changes in the urine is *dropsy* of the skin and of the serous membranes; which, however, is by no means constant. As there can be no question that the condition of the vessel walls of the skin also plays an important part in the origin of the anasarca besides the decreased diuresis, it is conceivable that a high degree of anasarca can really only be observed in acute nephritis after *colds* and *scarlatina, while it is almost always absent or less developed in that form of nephritis which is subsequent to other infectious diseases.* This is a rule which holds good at least in general, as appears from my observations at the bedside, which for years were directed upon this point, and which confirms the theoretical opinion of Cohnheim advanced in his time regarding the genesis of anasarca. *It is to some extent characteristic of anasarca in acute nephritis that it often changes its position* and does not develop first and most markedly in the lower extremities, as is the case especially in congested kidney. But not only the vessels of the skin, but those also of other regions of the body, in particular those of the serous membranes, may become *more permeable* under the influence of the infection, so that dropsy develops in the various cavities of the body. This also is more frequently the case in nephritides after scarlatina than after other infectious diseases. Besides the above-mentioned cause of anasarca another factor is to be taken into consideration which is in direct connection with the renal affection and essentially favours the occurrence of dropsy. This is the *retention of water—the "serous plethora"*—caused by the diminished secretion of urine and of the lymphatic resorption. In accord herewith is the fact that the intensity of the dropsy, as a rule at least, rises and falls with the increase or decrease of the quantity of the urine voided.

Hypertrophy of the left ventricle, which plays such an important part in the diagnosis and prognosis of chronic nephritis, *is almost without exception absent in acute nephritis.*

Case History.—The following case, which was recently observed by me, proves beyond doubt that *hypertrophy of the heart may occur in acute nephritis* in very rare cases.

C., tailor, sixteen years old, healthy until then, was attacked by dysphagia on December 12th; fever, 101.3° F., and angina follicularis were demonstrated when admitted to the hospital on December 26th. *The examination of the heart showed normal conditions; no albumin in the urine* The fever disappeared on the second day, also the swelling of the tonsils, so that the patient appeared cured on December 31st. *On January 1st* the patient noticed that his urine was of a darker colour; it contained much albumin and blood and many hyaline and few epithelial casts in

the sediment. Quantity, 700 cc. The face was slightly bloated, but there was no œdema elsewhere. *On January 5th the apex beat of the heart was accentuated in the fourth and fifth intercostal space in the mamillary line. Pulse regular, not hard to the touch; but a sphygmographic curve, taken on January 7th, showed slightly indicated back-stroke elevation and advance of the first catacrotic ascension towards the apex of the curve.* Considerable headache, sensorium clouded, dyspnœa (lungs were found to be normal), nausea. Diagnosis: *Incipient uræmia*; ordered caffeine sodium salicylate 0.1 subcutaneously. The locality of the apex beat in the fifth intercostal space was marked with an argentum crayon on January 9th. On the following day *the apex beat was markedly stronger and had advanced beyond the mark, about one finger's breadth outside of the mamillary line*; heart sounds pure. January 11th: The right border of the cardiac dulness extended a trifle beyond the left border of the sternum; profuse secretion of urine; the uræmic symptoms had disappeared. From now on progressive improvement, so that, on February 6th, the urine only contained traces of albumin; however, now and then there occurred fluctuations in the condition of the urine, especially could blood occasionally again be demonstrated, for the last time on March 15th. Patient left the hospital on March 27th at his urgent request; *the apex beat of the heart could still be felt externally from the mamillary line*; the urine still contained traces of albumin; the pulse had changed materially against its former condition—distinct back-stroke elevation.

The case is very remarkable in so far as the patient came under treatment with urine free from albumin and normal heart; *that the acute nephritis was observed from the first day on, and that, one to one and a half weeks after its onset, the acute development of a hypertrophy of the heart was positively demonstrated.*

Changes in the Pulse.—As rarely as such an acute occurrence of hypertrophy of the heart is observed in acute nephritis, as often is there, in my experience, a change of the pulse demonstrable which is to be regarded as the first beginning of the development of a subsequent hypertrophy, which usually does not occur owing to the short duration of acute nephritis. This is the *greater tension of the vessel wall* the early occurrence of which in the course of nephritis was first brought to the attention of the profession by Riegel. Although it cannot be demonstrated by the simple feeling of the pulse, it can be accomplished by means of the sphygmograph. If we accustom ourselves to examine the pulse of every patient suffering from acute nephritis sphygmographically, we shall observe that rarely the recession of the back-stroke elevation or the greater distinctness of the elasticity elevation, the advancing of the secondary ascension towards the top of the curve, etc., will be absent. However, we must not expect these changes of the pulse to occur always with the greatest distinctness, but we should also pay attention to slighter indications of this theoretically so important alteration of the pulse.

Uræmic Symptoms.—Other symptoms which may be observed in acute nephritis are almost always due to the retention of excrementitious substances. I count among these symptoms the gastric disturbances: *loss of appetite, nausea, vomiting*—the initial vomiting which is sometimes observed is of a reflex character—furthermore, *the diarrhœas, the pruritus, the drowsiness and the headaches.* If these symptoms increase, and if, finally, epileptoid convulsions occur and coma, the picture of "*uræmia*" in its usual meaning is completed. The details of the diagnosis of uræmia will be enlarged upon when discussing chronic nephritis. Inflammations of internal organs not infrequently develop in the course of acute nephritis:

Pleuritis, pericarditis, bronchitis, etc. The pain in the lumbar region, of which these patients often complain, is in connection with the enlargement of the kidney.

Ætiology.—The diagnosis of acute nephritis never encounters serious difficulties after what has been stated above, especially if the *ætiology of acute Bright's disease* is also considered: Preceding *colds, drenchings,* furthermore *poisoning* (with cantharides, especially, also after external application of the same, with turpentine oil, pyrogallol, naphthol, balsam of Peru, further, with various acids, especially sulphuric acid and salicylic acid, with mercury, etc.). Also the irritation of the renal parenchyma by the excessive use of acrid spices, of pepper, mustard, radishes, etc., may cause nephritis. However, preceding *infectious diseases* are most frequently the cause of the development of acute nephritis. Especially pneumonia, enteric fever, sepsis, Weil's disease, malarial fever, influenza, and diphtheria (besides, as I can confirm, simple angina); furthermore, measles, variola, varicella, and, above all, as is well known, scarlatina may lead to acute nephritis. In epidemics of diphtheria, as well as of scarlatina, cases have been observed in which nephritis occurred without the usual pathological manifestations in the throat, respectively without exanthema, and had to be explained as the expression of an unusual effect of the diphtheria, respectively scarlatina, virus which was restricted exclusively to the kidneys. The cause of nephritis arising in the course of infectious diseases is to be looked for, as has been demonstrated by clinical and experimental facts, either in the direct effect of the respective pathogenic bacteria upon the kidneys or in irritation of the latter by the toxine which is formed by these bacteria.

Various investigators have recently found pathogenic microbes in the urine of patients suffering from certain forms of acute Bright's disease, the origin of which could not be attributed to any of the above-named usual causes. These microbes were bacilli (Letzerich and others) as well as cocci: Staphylococci and streptococci which were never found in the urine of healthy persons nor in that of patients suffering from other diseases, which grew in pure cultures and, as shown by Mannaberg and others, when injected into the circulation of animals produced an intense nephritis. These specific nephritis streptococci (differing in their effect from other streptococci, also from those of erysipelas) do not multiply in the kidney, in spite of the injury to the renal tissue, and they disappear more or less rapidly from the kidneys and urine. Such acute *specific bacterial nephritides* terminate usually in rapid recovery, according to Mannaberg, while the cases of acute Bright's disease, in which streptococci in the urine are absent at the onset, appear to be more apt to pass into the chronic form. From my own experience I am able to confirm the occurrence of streptococci in some cases of acute nephritis which were observed in my clinic. The urine, which presents the other symptoms of nephritis, contains in some instances blood pigment without or with only isolated blood corpuscles; upon microscopical examination the pigment will be found in granules, flakes, or cylinders. This "*hæmoglobinuric nephritis*" has been observed either subsequent to *infectious diseases* (scarlatina, typhoid fever, pernicious malaria, etc.) or after extensive *burns* and *poisonings* and in the course of the so-called *paroxysmal hæmoglobinuria.* A separation of the blood pigment from the stroma of the blood corpuscles was also occasionally found as a secondary manifestation in some cases of severe hæmorrhagic nephritis. The explanation of the origin of hæmoglobinuric nephritis should be made differently in the individual cases; it appears that in some of the cases the hæmolysis of the cells as well as the inflammatory irritation of the kidneys is caused by the same noxa, while in other cases it seems that the hæmoglobin which enters the kidneys is the cause of the irritation of the epithelia (compare Hæmoglobinuria).

Differential Diagnosis—Acute Nephritis in the Course of Chronic Bright's Disease.—More difficult is the diagnosis of the *recurrences of acute nephritis,* which so frequently occur *in the course of chronic nephritis,* much more frequently than is usually supposed. If these recurrences appear in persons who, afflicted with chronic nephritis, have not been, so far, under treatment, a wrong diagnosis is very apt to be made, because, under such circumstances, a recent acute nephritis will be diag-

nosticated. Errors may be avoided if the anamnesis and the condition of the urine are carefully observed in certain respects. Anamnestically a distinct recent cause for the occurrence of the affection is absent in such cases, and it presents itself acutely (by acute dropsy, hæmaturia, etc.). The patient will rather state that lassitude, pallor, transitory œdema of the skin, headache, gastric disturbances, etc., have persisted for some time. Besides, the *urine*, although also hæmorrhagic, is cloudy and scanty in those cases in which acute and chronic nephritis are combined, is relatively larger in quantity than in primary acute nephritis, and the specific gravity comparatively lower; besides pure epithelial casts there are more fatty epithelia and abundant metamorphosed casts; eventually marked hypertrophy of the heart and albuminuric retinitis may be demonstrated, all of which are symptoms, the development of which requires, almost without exception, some length of time, and points directly to the presence of a previously existing chronic nephritis.

Febrile [Toxic] Albuminuria or Acute Nephritis.—As acute nephritis occurs in the course of febrile infectious diseases, on the one hand, and, on the other, sometimes, though rarely, its course as such is febrile, the question may suggest itself to the diagnostician, *whether an albuminuria which is accompanied with fever is a so-called "febrile" one* (see p. 347) *or whether it is to be referred to the existence of an acute nephritis.* It is advisable at first to separate diagnostically a febrile albumosuria from a febrile albuminuria accompanied by the excretion of serum albumin, and, furthermore, in the latter case to speak of acute nephritis only when the albuminuria uniformly persists for several days and shows considerable intensity—i. e., if traces of albumin appear in the urine not only transitorily. Furthermore, if blood can be demonstrated in the urine and if the microscopical examination of the urinary sediment reveals *epithelial*, besides hyaline, casts. *Dropsy* occurs too rarely in acute nephritis, if it appears in the course of febrile infectious diseases —except scarlatina—to make use of its absence in order to establish a differential diagnosis. Although these diagnostic rules are, in my experience, necessary for practical reasons, yet I wish to emphasize that a strict separation of febrile albuminuria from acute nephritis, at least in my opinion, is not proper from a theoretico-pathological standpoint. As "febrile" albuminuria is found in varying frequency in the different infectious diseases, and its occurrence is not at all always in proportion to the fever, and as, furthermore, an elimination of the infectious material through the kidneys may be assumed to be certain, albuminuria during fever, therefore, is in by far the majority of cases under all circumstances nothing else than the expression of the irritation of the renal tissue, especially of the vascular loops, by the virus of the febrile infectious disease. If the febrile albuminuria were merely the result of circulatory disturbances in the kidneys, incited by the weakening of the energy of the heart in consequence of the febrile affection, therapeutic measures to improve the weakness of the heart and against the fever should exert a much more marked effect upon the degree and the course of the albuminuria than is actually the case. If the irritation of the kidneys is increased by the virus, a marked picture of acute nephritis presents itself.

Corresponding to these views, we find at the autopsies of individuals, who *intra vitam* showed only symptoms of "febrile" albuminuria during the disease, almost always cloudy swelling of the epithelia; in patients who had suffered from scarlatina, possibly capsular epithelial desquamation, even scattered interstitial areas of infiltration in the renal tissue.

Glomerulo-Nephritis.—In scarlatina there occurs very frequently a form of acute nephritis which is designated as *glomerular nephritis*, characterized by the fact that the connective tissue and the epithelium are less affected, while the glomeruli are severely injured. Such glomerular nephritides might be differentiated diagnostically from common acute Bright's disease by observing the character of the *casts, which in this case should never have the appearance of epithelial casts*—i. e., of cylinder-like rows of epithelia. However, such diagnoses are of no *practical* value. At most, the presence of epithelial casts might be made use of for the diagnosis in a negative sense, inasmuch as it proves that the inflammatory process has not been restricted to the glomeruli alone in these cases, but has extended beyond them and led to the desquamation of the epithelia of the uriniferous tubules.

CHRONIC NEPHRITIS

1. *Chronic parenchymatous nephritis, chronic nephritis s. str., subacute and subchronic nephritis. Large white kidney. Large red, respectively red-mottled, kidney* (" *chronic hæmorrhagic nephritis* ").

Composition of the Urine—Quantity—Colour.—The *quantity* of urine is a little below normal, about 1 litre in twenty-four hours, in the usual form of chronic nephritis (in its earlier stages). It is generally the less, the more dropsy is developed, and, on the other hand, it may occasionally increase rapidly if the drospical transudates are quickly resorbed. The urine is *yellowish dirty* or of a *flesh-water colour*, and *cloudy* due to suspended urates or solid particles which precipitate upon standing.

Sediment.—The examination of the *sediment* reveals: Leucocytes, partly in a fatty condition, red blood corpuscles, fatty renal epithelia, and very abundant urinary casts of varying shapes, some hyaline and epithelial, others dark, granular, wax-like and shining, metamorphosed casts in all of which fatty degeneration is more or less prominent. Their surface is covered with fat granules, migratory cells, urates, and micrococci. The *specific gravity* of the urine is slightly increased or normal.

Albumin and Blood in Urine.—The most important alteration of the urine is the *presence of albumin* in the same; less albumin is usually excreted during the night than in daytime, less during rest than after bodily exercise. The quantity of the albumin excreted varies (0.5 per cent to 2 per cent and more), usually the less, the more the atrophy of the inflamed organ advances. *Blood* can be demonstrated in the urine, *as a rule*, not only microscopically but also chemically (best with Almén's test). The more blood appears in the urine, the more probable becomes the presence of a chronic hæmorrhagic nephritis (in contradistinction to large white kidney). The clinico-differential diagnosis, however, between large white and large red kidney is generally uncertain and of minor importance.

The normal *solid constituents* of the urine are excreted in abnormal quantities in chronic nephritis. The amount of *urea* excreted is not as much in general as that which is excreted by a healthy " control " person. However, this is not always the case; fluctuations in the daily quantity of urea excreted are quite usual. The excretion of uric acid appears to be within the normal limits, also in the chronic form of nephritis, the same as the ratios which refer to the excretion of uric acid to that of the alloxur bases; as has recently been demonstrated by Kolisch, the proportion of these two urinary constituents may, in some of the cases of nephritis, shift in such a manner that, in contradistinction to the normal condition, the quantities excreted of the alloxur bases grow at the expense of the excretion of uric acid.

Dropsy.—*Dropsy* is a symptom of chronic nephritis next in importance to the alterations in the urine. It is of an exceedingly high grade in this form of nephritis (the anatomical substratum of which is the large white or red kidney), and it affects the skin as well as the serous cavities of the body. The lower extremities and the scrotum in particular are swollen; the face appears pale and puffy.

Cause of Dropsy.—The *cause* of the œdema is to be looked for in various factors, so far as we are able to infer from clinical and recent experimental experiences: To a smaller degree the hydræmia due to the loss of albumin is responsible, to a greater extent the water retention (serous plethora), which is caused by the decrease of the lymphatic resorption and urinary excretion, provided the latter is not compensated by the stimulation of other secretions. These factors are the first, therefore, to be considered in the ætiological diagnosis of dropsy. However, it would not be correct to regard this as the sole cause of the dropsy formation in all cases; in some instances it is due, the same as in acute nephritis, to the greater permeability of the vessel wall (see p. 353), but, above all, to the mechanical factor of impaired circulation which undoubtedly co-operates, inasmuch as the compensatory increase of the activity of the heart is not accomplished sufficiently or relaxes temporarily. The latter factor is also to be taken into consideration when deciding upon the origin of the dropsy, if the latter grows with decrease of the energy of the heart and if the swelling of the lower extremities constantly predominates.

Heart and Pulse.—Particular attention is to be paid to the examination of the *heart* and of the pulse.

As mentioned in the discussion of acute nephritis, it is also often possible in chronic nephritis very easily to recognise an increased vascular tension from the behaviour of the pulse curve. The most probable reason for this fact is, in my opinion, an irritation of the vessel walls or of the vaso-motor centre by the altered blood mixture and the increase of the pressure in the vascular system caused thereby. The myocardium must become more active under the influence of the latter, and sooner or later it will hypertrophy (compare p. 54). It seems to me that it depends upon various factors whether the hypertrophy is brought about early or later in the disease: Whether the nervous system promptly reacts to the chemical irritation, but principally whether the stimulation to greater activity which is exerted upon the heart is a more constant one, or whether there are, besides periods of irritation, other periods in which the excretion of excrementitious substances has again become abundant for a while and thus no reason exists for an increased blood pressure, etc.

It may be considered as a general rule that *the longer the affection of the kidneys exists, the surer a hypertrophy of the heart may be expected, and vice versa*, although exceptions to this rule occur in both directions. The hypertrophy sometimes affects the entire heart, at other times—the usual occurrence—only the left ventricle is implicated. Dilatation of the heart is also found, as a rule, besides hypertrophy.

It is to be emphasized that the formation of a hypertrophy of the heart in insidious atrophy of the kidney, etc., appears to take place with greater certainty, because in this case the entire nutrition is less impaired than in large white kidney. It is rare in the latter disease that hypertrophy of the heart is plainly developed. This hypertrophy does not become prominent until later, when the sequences of the interstitial inflammation are more developed.

We find in chronic nephritis, the same as in the acute form, a tendency to inflammation of the serous and mucous membranes—bronchial catarrh, pleuritis, pericarditis, pneumonia, etc. Albuminuric retinitis occurs only rarely in the initial stages of chronic parenchymatous nephritis; it is much more frequent in the later course of the disease.

Uræmic Symptoms.—Gastric and intestinal symptoms occur in chronic parenchymatous nephritis, but they are less frequent than in atrophy of the

kidney. They are the consequence of gastric and intestinal catarrh or of an œdematous saturation of the stomach wall, but, above all, the expression of *uræmic intoxication* in a broader sense. The excretion of the urinary constituents by another route than by the kidneys, namely, by the intestine, exerts, in a certain number of cases, an inflammatory irritation upon the intestinal mucous membrane, and may thus often be the cause of extensive (croupous, ulcerative) alterations of the intestinal wall; in other cases, again, the gastric symptoms and the diarrhœa are purely nervous—i. e., manifestations brought about by the toxic irritation of the nerves of the digestive organs. Of an undoubted uræmic character are, further, the convulsions, the somnolence, the headache, etc. The aggregate of these symptoms produces the classical picture of fully developed uræmia.

The diagnosis of uræmia is easy in many cases, in others, again, the correct differentiation of the uræmic picture from other pathological conditions is one of the most difficult diagnostic problems.

The fully developed attack (vomiting, amaurosis, convulsions, coma, stertorous respiration, sometimes intermittent breathing in the Cheyne-Stokes type) is so characteristic that a mistaking of this pathological picture with another one should scarcely be considered possible. As the uræmic attack is undoubtedly accompanied with an irritation of the brain, it is obvious that diffuse cerebral irritations, which occur suddenly owing to other reasons, especially cases of changed relation of the circulation of the brain (see chapter on Cerebral Anæmia), are bound to show a similar picture. Mistakes can only be avoided by observation of the urine, which, although it may also become albuminous in these cases of cerebral anæmia owing to the general decrease of the aortic pressure, nevertheless, shows albumin only transitorily, and at any rate no epithelial, respectively metamorphized, casts, blood, etc., in the sediment.

But uræmia does not always occur as a fully developed affection. Thus we see at times only the occurrence of *psychical disturbances* (delirias, manias), tinnitus, baryecoia, etc., as uræmic manifestations. The expressions of limited nervous irritation: Contractions of individual muscle groups of one half of the body, in fact, actual hæmiplegias, have also been observed as an expression of uræmia. Furthermore, amaurosis may be the only symptom of a uræmic intoxication, the same as coma without its usual companion, convulsions.

Origin of Uræmia.—The most various theories have been set up in the course of the last decades regarding the *origin of uræmia*, but none that is satisfactory to explain all the details of the pathological picture. If we review the long list of clinical and experimental facts concerning the origin of the uræmic symptom-complex, it follows that uræmia depends, in the *first* place, upon a *retention of the urinary constituents* due to the impaired function of the kidneys; however, the toxic effect should not be ascribed to *one* of them exclusively. The intoxication manifests itself principally as an irritation of the nervous system, especially also of the vaso-motors and the brain. I consider the irritation of the vaso-motors as a compensating occurrence throughout, which causes an increase of the blood pressure and of the hypertrophy of the heart, on the one hand, and, on the other, a more thorough flooding of the cerebral capillaries. This compensation, however, has its limits, and when it is deficient or becomes relaxed, the effect of the intoxication becomes very conspicuous. If *certain transitory local symptoms* become prominent in the uræmic

picture, as signs of the special irritation of certain portions of the brain, the acute development of local cerebral œdema may be thought of in such cases, and its passing existence is analogous to the rapid occurrence and disappearance of œdema in other portions of the body in the course of nephritis.

The diagnosis of uræmia loses in certainty owing to the multiplicity of the pathological picture in which the affection may occur, more so because the quantity of the urinary secretion is by no means always an indication for the onset of a uræmia. Although this onset usually coincides with a considerable decrease in the quantity of the urine, yet uræmia has been seen to occur also in cases in which the secretion of urine and urea has not materially changed, and, *vice versa,* to fail to appear in long-lasting anuria. If we further consider that in some cases attacks of asthma (compare p. 59) become prominent and with insidious uræmia only very slight nervous symptoms manifest themselves—pruritus, headache, slight stupor, vertigo, nausea, etc.—it becomes sufficiently clear how difficult it is to recognise uræmic intoxication with certainty in every instance.

Differential Diagnosis between Uræmia and Similar Pathological Conditions.—A leading rule for the diagnosis is *to assume the uræmic intoxication to be the cause of nervous symptoms in patients with nephritis only when other causes for the same can be positively excluded.* This diagnosis by exclusion has often stood me in good stead in doubtful cases of uræmia, and it is the more necessary, because in the course of renal affections there occur often anatomically noticeable changes also in the central nervous system, like hæmorrhages and meningitis which may be the cause of a clinical picture similar to uræmia. The diagnosis in such cases may often be made only with a certain degree of probability.

Cerebral Disease.—Limited convulsions and, more so, hemiplegias almost always point decidedly more to the development of anatomical alterations in the central nervous system, especially if these symptoms remain uniform and are not of a transitory character, as in uræmia. It should not be forgotten that circumscribed thrombotic, softened areas of the brain have been found in rare cases as consequences of the uræmic attack. Diagnostic doubts occur further, often in my experience, between *meningitis* and *uræmia*, provided the former is accompanied with albuminuria. Isolated paralysis of cerebral nerves, stiffness of the neck, retraction of the abdomen, etc., of course point to meningitis; but there are cases in which the latter affection takes a course without local symptoms and in which a differential diagnosis is absolutely impossible.

Finally, *intoxications* and *severe infectious diseases* may simulate the picture of uræmia, because the latter affection, especially the acute form, is sometimes associated with *fever*, as has recently been particularly emphasized by Rosenstein, and which I can confirm from my own experience. Principally to be considered are severe cases, accompanied with cerebral manifestations, of miliary tuberculosis, enteric fever and cryptogenetic septicopyæmia, if their course is attended with albuminuria but without marked symptoms of a nephritis. To me, at least, these affections have often given considerable differentio-diagnostical difficulties. *Miliary tuberculosis* is most easy to exclude, comparatively, because the pulmonary symptoms, with inflammatory manifestations upon pleura and pericardium predominate, at least as a rule, and because the ophthalmoscopical examination eventually reveals tubercles in the fundus of the eye. The differentiation of uræmia from enteric fever may become much more difficult; but here the swelling of the spleen and roseola, the relative retardation of the pulse, the typical course of the fever, and, above all, the positive Gruber-Widal reaction will decide in favour of enteric fever, while in uræmia, the

fever, if at all present, is only moderate, and does not show the regular course as in enteric fever; furthermore, the swelling of the spleen is usually absent and the pulse does not follow any rule as to its frequency. The determination of objectively demonstrable multiple areas of inflammation points to *septicopyæmia*, especially of pustular and pemphigus exanthemata, endocarditis and arthritis, intermittent fever with chills, and, finally, the ophthalmoscopical finding (extravasations into the retina, eventually with a white centre).

Poisoning.—It is also possible that an *intoxication*, especially with *opium* or *alcohol*, gives rise to gross mistakes. The decision in these cases is arrived at by the anamnesis, by the observation of the condition of the urine, by the congestive irritation of the vascular nerves, and by the cheerful character of the delirium in alcohol intoxication. In doubtful cases the ophthalmoscopical examination should under no circumstances be forgotten, which reveals the quite frequent albuminuric retinitis in nephritis and thus furnishes a positive symptom for the diagnosis of uræmia.

Ætiology.—The *ætiology* yields but few points for the diagnosis of chronic parenchymatous nephritis. The causes named in the discussion of acute nephritis may, all of them, also lead to chronic parenchymatous nephritis, if the noxæ are brought to bear upon the kidneys for a *long time* or, affecting them only a short while, yet alter and weaken the tissue of the kidneys to such an extent that the diseased organ is now further irritated by insignificant, otherwise harmless irritants and becomes chronically affected. Thus it is possible that chronic infectious diseases which are present besides nephritis, as syphilis, malaria, tuberculosis, furthermore colds, which are anamnestically established and which affect the patient frequently and for some time, abuse of alcohol and mercury, etc., may give at least some support to the diagnosis of the chronic form of parenchymatous nephritis.

1. VARIETY: SECONDARY ATROPHIC KIDNEY, SECONDARY INDURATION OF THE KIDNEY

The picture of *secondary atrophic kidney* which represents the final stage of chronic parenchymatous nephritis—i. e., which gradually develops after a long duration of the same—differs in various respects from that of the latter affection, so that the diagnosis of the former usually does not present any difficulties.

Composition of the Urine.—Above all, *the quantity of the urine increases* in comparison to that in chronic parenchymatous nephritis; normal or larger quantities of urine are excreted, while the specific gravity decreases (about 10.15 to 10.10). *The urine, although relatively abundant, is still slightly cloudy, bloody, and contains sediment*; the sediment contains, besides white blood corpuscles and epithelia, *still many casts* in their various forms, and also *red blood corpuscles*. The *albuminuria* is generally *more profuse than in primary atrophied kidney*. Neither is *dropsy* entirely absent, in contradistinction to the condition in the latter affection, in which dropsy does not occur—at least not during the greater part of the disease. The occurrence of uræmic symptoms is the rule, retinitis is a very common manifestation, and hypertrophy of the heart has also developed in almost every instance. However, there cannot be any question of a strict differentiation between secondary and primary indurative nephritis based upon clinical symptoms; the diagnosis is usually possible only if the course of the disease is known to us in the individual case, and if we have personally observed the earlier stages of the affection.

Cause of Dropsy.—The *cause* of the œdema is to be looked for in various factors, so far as we are able to infer from clinical and recent experimental experiences: To a smaller degree the hydræmia due to the loss of albumin is responsible, to a greater extent the water retention (serous plethora), which is caused by the decrease of the lymphatic resorption and urinary excretion, provided the latter is not compensated by the stimulation of other secretions. These factors are the first, therefore, to be considered in the ætiological diagnosis of dropsy. However, it would not be correct to regard this as the sole cause of the dropsy formation in all cases; in some instances it is due, the same as in acute nephritis, to the greater permeability of the vessel wall (see p. 353), but, above all, to the mechanical factor of impaired circulation which undoubtedly co-operates, inasmuch as the compensatory increase of the activity of the heart is not accomplished sufficiently or relaxes temporarily. The latter factor is also to be taken into consideration when deciding upon the origin of the dropsy, if the latter grows with decrease of the energy of the heart and if the swelling of the lower extremities constantly predominates.

Heart and Pulse.—Particular attention is to be paid to the examination of the *heart* and of the pulse.

As mentioned in the discussion of acute nephritis, it is also often possible in chronic nephritis very easily to recognise an increased vascular tension from the behaviour of the pulse curve. The most probable reason for this fact is, in my opinion, an irritation of the vessel walls or of the vaso-motor centre by the altered blood mixture and the increase of the pressure in the vascular system caused thereby. The myocardium must become more active under the influence of the latter, and sooner or later it will hypertrophy (compare p. 54). It seems to me that it depends upon various factors whether the hypertrophy is brought about early or later in the disease: Whether the nervous system promptly reacts to the chemical irritation, but principally whether the stimulation to greater activity which is exerted upon the heart is a more constant one, or whether there are, besides periods of irritation, other periods in which the excretion of excrementitious substances has again become abundant for a while and thus no reason exists for an increased blood pressure, etc.

It may be considered as a general rule that *the longer the affection of the kidneys exists, the surer a hypertrophy of the heart may be expected, and vice versa*, although exceptions to this rule occur in both directions. The hypertrophy sometimes affects the entire heart, at other times—the usual occurrence—only the left ventricle is implicated. Dilatation of the heart is also found, as a rule, besides hypertrophy.

It is to be emphasized that the formation of a hypertrophy of the heart in insidious atrophy of the kidney, etc., appears to take place with greater certainty, because in this case the entire nutrition is less impaired than in large white kidney. It is rare in the latter disease that hypertrophy of the heart is plainly developed. This hypertrophy does not become prominent until later, when the sequences of the interstitial inflammation are more developed.

We find in chronic nephritis, the same as in the acute form, a tendency to inflammation of the serous and mucous membranes—bronchial catarrh, pleuritis, pericarditis, pneumonia, etc. Albuminuric retinitis occurs only rarely in the initial stages of chronic parenchymatous nephritis; it is much more frequent in the later course of the disease.

Uræmic Symptoms.—Gastric and intestinal symptoms occur in chronic parenchymatous nephritis, but they are less frequent than in atrophy of the

kidney. They are the consequence of gastric and intestinal catarrh or of an œdematous saturation of the stomach wall, but, above all, the expression of *uræmic intoxication* in a broader sense. The excretion of the urinary constituents by another route than by the kidneys, namely, by the intestine, exerts, in a certain number of cases, an inflammatory irritation upon the intestinal mucous membrane, and may thus often be the cause of extensive (croupous, ulcerative) alterations of the intestinal wall; in other cases, again, the gastric symptoms and the diarrhœa are purely nervous—i. e., manifestations brought about by the toxic irritation of the nerves of the digestive organs. Of an undoubted uræmic character are, further, the convulsions, the somnolence, the headache, etc. The aggregate of these symptoms produces the classical picture of fully developed uræmia.

The diagnosis of uræmia is easy in many cases, in others, again, the correct differentiation of the uræmic picture from other pathological conditions is one of the most difficult diagnostic problems.

The fully developed attack (vomiting, amaurosis, convulsions, coma, stertorous respiration, sometimes intermittent breathing in the Cheyne-Stokes type) is so characteristic that a mistaking of this pathological picture with another one should scarcely be considered possible. As the uræmic attack is undoubtedly accompanied with an irritation of the brain, it is obvious that diffuse cerebral irritations, which occur suddenly owing to other reasons, especially cases of changed relation of the circulation of the brain (see chapter on Cerebral Anæmia), are bound to show a similar picture. Mistakes can only be avoided by observation of the urine, which, although it may also become albuminous in these cases of cerebral anæmia owing to the general decrease of the aortic pressure, nevertheless, shows albumin only transitorily, and at any rate no epithelial, respectively metamorphized, casts, blood, etc., in the sediment.

But uræmia does not always occur as a fully developed affection. Thus we see at times only the occurrence of *psychical disturbances* (delirias, manias), tinnitus, baryecoia, etc., as uræmic manifestations. The expressions of limited nervous irritation: Contractions of individual muscle groups of one half of the body, in fact, actual hæmiplegias, have also been observed as an expression of uræmia. Furthermore, amaurosis may be the only symptom of a uræmic intoxication, the same as coma without its usual companion, convulsions.

Origin of Uræmia.—The most various theories have been set up in the course of the last decades regarding the *origin of uræmia*, but none that is satisfactory to explain all the details of the pathological picture. If we review the long list of clinical and experimental facts concerning the origin of the uræmic symptom-complex, it follows that uræmia depends, in the *first* place, upon a *retention of the urinary constituents* due to the impaired function of the kidneys; however, the toxic effect should not be ascribed to *one* of them exclusively. The intoxication manifests itself principally as an irritation of the nervous system, especially also of the vaso-motors and the brain. I consider the irritation of the vaso-motors as a compensating occurrence throughout, which causes an increase of the blood pressure and of the hypertrophy of the heart, on the one hand, and, on the other, a more thorough flooding of the cerebral capillaries. This compensation, however, has its limits, and when it is deficient or becomes relaxed, the effect of the intoxication becomes very conspicuous. If *certain transitory local symptoms* become prominent in the uræmic

picture, as signs of the special irritation of certain portions of the brain, the acute development of local cerebral œdema may be thought of in such cases, and its passing existence is analogous to the rapid occurrence and disappearance of œdema in other portions of the body in the course of nephritis.

The diagnosis of uræmia loses in certainty owing to the multiplicity of the pathological picture in which the affection may occur, more so because the quantity of the urinary secretion is by no means always an indication for the onset of a uræmia. Although this onset usually coincides with a considerable decrease in the quantity of the urine, yet uræmia has been seen to occur also in cases in which the secretion of urine and urea has not materially changed, and, *vice versa,* to fail to appear in long-lasting anuria. If we further consider that in some cases attacks of asthma (compare p. 59) become prominent and with insidious uræmia only very slight nervous symptoms manifest themselves—pruritus, headache, slight stupor, vertigo, nausea, etc.—it becomes sufficiently clear how difficult it is to recognise uræmic intoxication with certainty in every instance.

Differential Diagnosis between Uræmia and Similar Pathological Conditions.—A leading rule for the diagnosis is *to assume the uræmic intoxication to be the cause of nervous symptoms in patients with nephritis only when other causes for the same can be positively excluded.* This diagnosis by exclusion has often stood me in good stead in doubtful cases of uræmia, and it is the more necessary, because in the course of renal affections there occur often anatomically noticeable changes also in the central nervous system, like hæmorrhages and meningitis which may be the cause of a clinical picture similar to uræmia. The diagnosis in such cases may often be made only with a certain degree of probability.

Cerebral Disease.—Limited convulsions and, more so, hemiplegias almost always point decidedly more to the development of anatomical alterations in the central nervous system, especially if these symptoms remain uniform and are not of a transitory character, as in uræmia. It should not be forgotten that circumscribed thrombotic, softened areas of the brain have been found in rare cases as consequences of the uræmic attack. Diagnostic doubts occur further, often in my experience, between *meningitis* and *uræmia,* provided the former is accompanied with albuminuria. Isolated paralysis of cerebral nerves, stiffness of the neck, retraction of the abdomen, etc., of course point to meningitis; but there are cases in which the latter affection takes a course without local symptoms and in which a differential diagnosis is absolutely impossible.

Finally, *intoxications* and *severe infectious diseases* may simulate the picture of uræmia, because the latter affection, especially the acute form, is sometimes associated with *fever,* as has recently been particularly emphasized by Rosenstein, and which I can confirm from my own experience. Principally to be considered are severe cases, accompanied with cerebral manifestations, of miliary tuberculosis, enteric fever and cryptogenetic septicopyæmia, if their course is attended with albuminuria but without marked symptoms of a nephritis. To me, at least, these affections have often given considerable differentio-diagnostical difficulties. *Miliary tuberculosis* is most easy to exclude, comparatively, because the pulmonary symptoms, with inflammatory manifestations upon pleura and pericardium predominate, at least as a rule, and because the ophthalmoscopical examination eventually reveals tubercles in the fundus of the eye. The differentiation of uræmia from enteric fever may become much more difficult; but here the swelling of the spleen and roseola, the relative retardation of the pulse, the typical course of the fever, and, above all, the positive Gruber-Widal reaction will decide in favour of enteric fever, while in uræmia, the

fever, if at all present, is only moderate, and does not show the regular course as in enteric fever; furthermore, the swelling of the spleen is usually absent and the pulse does not follow any rule as to its frequency. The determination of objectively demonstrable multiple areas of inflammation points to *septicopyæmia*, especially of pustular and pemphigus exanthemata, endocarditis and arthritis, intermittent fever with chills, and, finally, the ophthalmoscopical finding (extravasations into the retina, eventually with a white centre).

Poisoning.—It is also possible that an *intoxication*, especially with *opium* or *alcohol*, gives rise to gross mistakes. The decision in these cases is arrived at by the anamnesis, by the observation of the condition of the urine, by the congestive irritation of the vascular nerves, and by the cheerful character of the delirium in alcohol intoxication. In doubtful cases the ophthalmoscopical examination should under no circumstances be forgotten, which reveals the quite frequent albuminuric retinitis in nephritis and thus furnishes a positive symptom for the diagnosis of uræmia.

Ætiology.—The *ætiology* yields but few points for the diagnosis of chronic parenchymatous nephritis. The causes named in the discussion of acute nephritis may, all of them, also lead to chronic parenchymatous nephritis, if the noxæ are brought to bear upon the kidneys for a *long time* or, affecting them only a short while, yet alter and weaken the tissue of the kidneys to such an extent that the diseased organ is now further irritated by insignificant, otherwise harmless irritants and becomes chronically affected. Thus it is possible that chronic infectious diseases which are present besides nephritis, as syphilis, malaria, tuberculosis, furthermore colds, which are anamnestically established and which affect the patient frequently and for some time, abuse of alcohol and mercury, etc., may give at least some support to the diagnosis of the chronic form of parenchymatous nephritis.

1. VARIETY: SECONDARY ATROPHIC KIDNEY, SECONDARY INDURATION OF THE KIDNEY

The picture of *secondary atrophic kidney* which represents the final stage of chronic parenchymatous nephritis—i. e., which gradually develops after a long duration of the same—differs in various respects from that of the latter affection, so that the diagnosis of the former usually does not present any difficulties.

Composition of the Urine.—Above all, *the quantity of the urine increases* in comparison to that in chronic parenchymatous nephritis; normal or larger quantities of urine are excreted, while the specific gravity decreases (about 10.15 to 10.10). *The urine, although relatively abundant, is still slightly cloudy, bloody, and contains sediment*; the sediment contains, besides white blood corpuscles and epithelia, *still many casts* in their various forms, and also *red blood corpuscles*. The *albuminuria* is generally *more profuse than in primary atrophied kidney*. Neither is *dropsy* entirely absent, in contradistinction to the condition in the latter affection, in which dropsy does not occur—at least not during the greater part of the disease. The occurrence of uræmic symptoms is the rule, retinitis is a very common manifestation, and hypertrophy of the heart has also developed in almost every instance. However, there cannot be any question of a strict differentiation between secondary and primary indurative nephritis based upon clinical symptoms; the diagnosis is usually possible only if the course of the disease is known to us in the individual case, and if we have personally observed the earlier stages of the affection.

2. PRIMARY, GENUINE ATROPHIC KIDNEY ("FOCAL, INDURATING" NEPHRITIS, "CHRONIC INTERSTITIAL" NEPHRITIS); RENAL SCLEROSIS

Contracted Kidney.—Regarding, finally, *primary atrophic kidney*, this is characterized by the *extremely slow, insidious development of the disease.*

Composition of the Urine—Polyuria.—The *quantity of the urine* is usually *very profuse*, it may amount to 3 or 4 litres and above. The more frequent desire to urinate, especially at night, and the increased thirst are the first symptoms to call attention to the disease. The cause of the polyuria can in most instances be ascribed to the hypertrophy of the heart which develops almost in every case; if the power of the heart decreases, the quantity of the urine diminishes.

However, the excessive diuresis cannot be explained solely by the hypertrophy of the left ventricle. A very important *rôle*[1] is also played, in my opinion, by anatomical alterations in the kidney proper (above all, the destruction of numerous cortical capillaries and the increased pressure and current velocity caused thereby in the preserved glomeruli); a decreased resorption of water in the medullary substance might possibly be of influence also (Ribbert).

Specific Gravity, Colour, Etc.—The *specific gravity* of the abundant urine is *low*, even if the quantity becomes temporarily less, 10.05 to 10.10; its *colour* is pale, light-green yellow. At the same time it is *clear* or but slightly cloudy, *leaving no sediment.* Only after standing for some time the urine will show a precipitate in which can be demonstrated a few casts, mostly hyaline, narrow and broad, sometimes also renal epithelia, leucocytes, and very rarely red blood corpuscles.

Albumin.—The *amount of albumin* is very small and increases only upon decrease of the quantity of the urine; it is possible, in some cases, which, however, are extremely rare, according to my experience, that the albumin is *entirely absent*, especially in the night urine. Occasionally the casts may temporarily disappear while the albuminuria persists; the cause of this symptom should be looked for, according to Sehrwald and others, in a temporary dissolution of the casts by the pepsin which is contained in the urine. *As to the quantity of the solid constituents of the urine, the kidney, in spite of a plentiful excretion of water, tends decidedly to a decrease in the excretion of urea, uric acid, phosphoric acid, chlorides, and ammonia.* However, it is quite a common occurrence that larger amounts of these substances are excreted between the periods of lesser secretion; in fact, it may be possible, as demonstrated by von Noorden and Ritter, that the excretion of nitrogen temporarily exceeds the intake of this substance considerably (obviously in consequence of excretion of nitrogenous products of decomposition which were previously retained). Considerable fluctuations can be observed especially during the uræmic attacks.

---

[1] Regarding details I refer to my deductions in the text-book "Die Lerke vom Harn," p. 317, published by Salkowsky and myself.

Dropsy, in contradistinction to other forms of nephritis, is *always slight, or is entirely absent,* so long as a copious diuresis exists and the hypertrophied heart displays the required energy. If the latter decreases in the course of the disease, the quantity of the urine diminishes also, and now œdema sets in which occurs principally in the lower extremities and which is to be interpreted as engorgement œdema.

Hypertrophy of the heart is present in by far the majority of cases and is demonstrable as such. The diagnosis should consider the degree of its development and the completeness of the compensation by the same during the entire course of the disease, especially because the prognosis of the individual case is determined principally by the prevailing intensity of the development of the hypertrophy of the heart. The second aortic sound is intensified, the *pulse* usually *hard,* the alteration of its force and rhythm is generally a good indicator of the threatening remission of the compensation by the hypertrophied heart and thus of the eventual occurrence of a cardiac asthma and of the uræmic attacks. The pulse curve often plainly shows anadicrotism and a recession of the back-stroke elevation.

The ophthalmoscopical examination usually reveals the characteristic signs of the so-called *albuminuric retinitis,* which may even be the very first sign of the pathological picture—i. e., it is sometimes present before any urinary changes can be positively demonstrated. *This albuminuric retinitis occurs in no other form of nephritis as frequently as in atrophied (contracted) kidney.*

Hæmorrhages from the internal organs do not infrequently occur during the course of chronic nephritis; nose-bleed, especially, is a frequent symptom of the affection. Quite usual, furthermore, are disturbances in the digestive tract, especially *diarrhœas* and *dyspepsia*; sometimes it is the latter pathological manifestation which causes the patient to consult the physician. It is in the majority of cases, in my opinion, a uræmic symptom and of nervous origin, as is proved by the digestion experiments.

Uræmia.—*Headache,* and doubtless also the other nervous manifestations—vertigo, pruritis, etc.—are likewise of an essentially uræmic character. I would advise particularly never to omit the examination of the urine in headache, even if it is of a purely intermittent character or unilateral. We shall thus avoid subsequent self-reproaches; a headache which so far appears to be harmless attains a grave diagnostic and prognostic significance upon the presence of albumin in the urine. It is especially a *chronic uræmia* manifesting itself only by the slightest disturbances, which is quite common in atrophic kidney, and it is not noticed until a sudden weakening of the activity of the heart changes it into a fully developed one, of apparently acute onset, or until an engorgement of the urinary constituents occurs to such a degree that the consummation of the stimuli precipitates a severe attack.

Cerebral Hæmorrhage.—Death may occur, besides from uræmia, from a serous pneumonia or quite in particular also from *cerebral hæmorrhage* which in a large percentage of cases owes its existence generally to atrophic kidney. It is necessary, therefore, to examine the urine for albumin in every case of apoplexy (in comatose individuals after it has been withdrawn by means of the catheter), but

it should not be forgotten that albuminuria occurs in cerebral hæmorrhage also without nephritis, simply in consequence of the change of the circulation caused by the cerebral affection. Although the determination of broad hyaline casts and of fatty renal epithelia in the urinary sediment, the low specific gravity of the urine, and the eventual demonstration of œdemas usually allow us under such circumstances to recognise an atrophic kidney as the cause of cerebral hæmorrhage, yet the question is to be left undecided in some cases whether an atrophic kidney is present besides apoplexy, and not until later does it become manifest whether the albuminuria was the consequence of the apoplexy or whether, conversely, the latter owes its origin to a granular atrophy of the kidneys which until then had taken a latent course. The same as in the brain, there occur hæmorrhages also at other places of the body, due to the influence of the hypertrophy of the heart and the vascular affection, like nose-bleed, uterine-gastric hæmorrhages, etc.

The ætiology of chronic interstitial nephritis allows us but rarely to draw conclusions which are of value in a support of the diagnosis. It is true, the presence of an atrophic kidney becomes probable at once if arthritis ("gouty kidney"), lead intoxication, alcoholism, diabetes mellitus, or a later stage of syphilis are demonstrably present, while, on the other hand, after malaria and after pulmonary phthisis, for instance, chronic parenchymatous nephritis may be expected most frequently. It is obvious, however, that such diagnostic considerations are of no value in comparison to the objective results of the urinary examination, etc.

Neither will it be possible to expect any help ætiologically for the diagnosis of renal sclerosis, which will be discussed supplementarily, because the just-mentioned noxæ, alcoholism, arthritis, etc., are acknowledged to lead to arteriosclerosis generally and with it also to arteriosclerotic atrophic kidney.

VARIETY: ARTERIOSCLEROTIC ATROPHIED KIDNEY, ARTERIOSCLEROTIC INDURATION, SCLEROSIS OF THE KIDNEYS

This variety of chronic interstitial kidney is characterized anatomically by sclerotic thickening of the small vessels of the kidney, by hyaline degeneration of the glomeruli, and degeneration of the glomerular and uriniferous tubules with *moderate* proliferation of the interstitial connective tissue. The *clinical* symptoms of the affection are essentially the same as those of common primary chronic atrophic kidney, so that it is not possible to distinguish it positively from the latter. However, a probable *diagnosis* of arteriosclerotic induration of the kidneys is permissible at least, if it is a question of elderly persons, if the symptoms of arteriosclerosis are very marked generally, and if hypertrophy of the heart, disturbances of compensation, and cardiac asthma become prominent early in the pathological picture. The amount of albumin in the urine is very small particularly in this form of atrophic kidney, and very often it is entirely absent.

AMYLOID DEGENERATION OF THE KIDNEYS—FATTY KIDNEY

The diagnosis of *amyloid kidney* is always difficult, according to my experience, because the urine is never changed so characteristically as in the nephritides.

The quantity of the urine, to begin with, is found to vary: normal, *increased* or decreased, and also in such a manner that increase and decrease of the diuresis alternate.

Of importance, above all, are cases in which absolutely pure amyloid kidney—i. e., vascular amyloid without epithelial and interstitial changes—are found post mortem. Rosenstein has recently described such cases, and he has observed in them an *increase of the amount of urine,* especially in amyloid degeneration of the vessels

of the medullary substance. The specific gravity of the urine was low at the same time, the colour pale. It is true, however, that Wagner and others, conversely, have seen a decrease of diuresis also in pure amyloid kidney, especially towards the end of life.

Quantity of Urine—Specific Gravity.—The *specific gravity* also varies, it is usually *decreased* (10.02 to 10.05), especially in cases of pure amyloid kidney; the *colour* is bright yellow and, what I consider to be the main point, *the urine is clear*, so that it is usually difficult to obtain a sediment. Casts are generally absent in the latter; however, there are cases, on the other hand, in which they are very abundant (hyaline and granular); besides, leucocytes are found in the urine but no blood. Casts which give an amyloid reaction have been demonstrated but very rarely. Their occurrence in the urine is more than doubtful. I have never been able to find them in spite of the most careful search in the urine of patients with the most marked amyloid degeneration of the kidneys.

The *albuminuria* also varies, being sometimes large, at other times slight; in some cases it was entirely absent in spite of indubitable amyloid degeneration of the renal vessels. Of course, such observations are of value only if long-continued examinations of the urine were made with always the same negative result. The following case, which was observed in my clinic, presents an example of constant absence of albumin in the urine in an instance in which the onset of amyloid kidney was awaited for months:

A boy, seven years old, admitted to the clinic April 18, 1885 (died July 16, 1886), suffered from *tuberculous spondylitis* of the lumbar vertebral column, *compression myelitis*. Liver and spleen appeared to be of normal size upon his admission, urine free from albumin. In the middle of January a gravitating abscess perforated in the left flank, and diarrhœa set in. From this time on until May a *hard, smooth hepatic tumour* developed with sharp border, deep interlobular incisure; no ascites. The spleen was not palpable. As an amyloid affection of the organ had to be diagnosticated according to the ætiology, to the consistence, size, and smoothness of the liver, we were daily expecting a diagnosticable amyloid degeneration of the kidney—i. e., the appearance of albumin in the urine. *The examination of the urine, however, which was carefully made therefor every day, gave an absolutely negative result*—except ten days in April, when tracelike cloudiness of the urine was found upon the reaction for albumin; especially during the last months of life of the patient the urine was surely *absolutely free from albumin*.

The autopsy showed, besides tuberculous caries of the spinal column and myelitis, *sago spleen* (size $10 \times 5\frac{1}{2} \times 2\frac{1}{2}$), enormous *lardaceous liver* (weight 1,400 gm.), *kidneys large and pale, the cortex paler than the medullary substance*. The amyloid test applied to the fresh organ *in toto* proved negative; the *microscopical examination*, however, gave an undoubtedly positive result—namely, an *amyloid degeneration of the glomeruli and of the vasa afferentia*.

We should expect to find albumin in the urine in cases of amyloid degeneration of the glomeruli, and that it *is bound* to be absent upon restriction of the degeneration to the medullary vessels. This, however, is not the case; albuminuria may eventually also be absent in marked amyloid degeneration of the glomeruli. It may be possible that in the latter case it has not advanced sufficiently, qualitatively, to render the glomerular wall permeable for the albumin, or that, as is proved by Hansemann's recently reported results of examination, at first only the loops become affected, while the covering epithelium still remains entirely intact.

The cases of amyloid kidney without albuminuria are the exception, generally, and the usual occurrence is an *abundant* albuminuria; besides the serum albumin there is also found globulin in the urine, the same as in diffuse nephritis. *Investigations of metabolism* are of little value in amyloid kidney regarding the estimate of its influence upon the *quantity of secretion of the individual urinary constituents*, because the renal affection is in this case only a partial manifestation of severe constitutional disorders. But this much appears to be certain at least, that *the amyloid degeneration of the kidneys does not prevent the passing out of secretable* substances. With this, the fact would agree that *uræmia* probably *never occurs* in pure amyloid kidney, and also that *hypertrophy of the heart is absent*. The conditions are different, however, if parenchymatous nephritis, and especially atrophic processes (as is frequently the case), become associated with amyloid degeneration. Then the hypertrophy of the heart does not fail to appear, and gradually uræmic symptoms will set in. Retinitis has also been observed in this combination.

It is *impossible* according to the above considerations to establish a positive diagnosis of amyloid kidney from the urinary picture alone; however, it is possible in most cases to make a correct diagnosis by observation of the concomitant manifestations. *Anasarca* and serous *cavity-dropsy* are present at least in the majority of cases, often very much developed, but in other cases again no dropsy is present. It is quite conceivable that high-graded *anæmia* and *cachexia* can also be observed very often. But more important for the diagnosis than all the above-named symptoms is the demonstration of a synchronous *amyloid degeneration of the liver and spleen*. As the kidneys alone were found to be affected by amyloid degeneration in only a few isolated cases, and in over 60 per cent of the cases liver, spleen, and kidneys simultaneously so, the diagnostic importance of this generalization of amyloid degeneration in the body becomes manifest, especially as amyloid spleen and liver are generally very easy to diagnosticate. The obstinate *diarrhœa*, a consequence of the amyloid degeneration of the intestinal wall, may lead to the diagnosis, although it may sometimes be due to other causes, especially to tuberculous enteritis.

Ætiology.—Finally, it is the *ætiology, the observation of which is primarily determining for the diagnosis. Phthisis* with ulcerative processes in the lungs and intestines, *bone suppurations*, long-lasting suppurations in general, and *inveterate syphilis* are the main sources of amyloid degeneration in general and therefore also of amyloid kidney. All other (besides, positively determined) causes of amyloid degeneration are exceedingly rare, on the other hand, like carcinoma, rhachitis, gout, and obstinate malarial fevers. In very isolated cases it was not possible at all to find any source whatever of amyloid degeneration. But it is advisable, nevertheless, to diagnosticate amyloid kidneys *only when phthisis, suppurations, or syphilis exist for some time, and if, at the same time, the liver and spleen (or at least one of these organs, especially the spleen) are enlarged and hard, in short, show the symptoms of amyloid degeneration plainly, and the urine contains albumin, and also shows the above-described conditions of the specific gravity, etc., especially when it is also remarkably clear, pale, and free from sediment in spite of the presence of much albumin.*

It seems that the occurrence of amyloid disease is rare in some localities and more frequent in others. I can only thus explain the fact that, for instance, in Würzburg, in spite of the enormous frequency of phthisis and tuberculous bone affections, comparatively few cases of amyloid disease are noted at the autopsy in the course of a year.

As the differential diagnosis between amyloid kidney, engorged kidney, and the various forms of nephritis is so very frequently forced upon the diagnostician, I have tabulated (on pages 368 and 369) the characteristic symptoms of the various diffuse affections of the kidneys which have been discussed so far.

SUPPURATIVE NEPHRITIS, SEPTIC AND PYÆMIC NEPHRITIS, PYELONEPHRITIS, RENAL ABSCESS

The inflammatory processes of the kidneys now to be considered, which may be traced to an *entrance of bacteria into the kidney*, either by means of the vascular passage or through the urine-secreting ducts, are characterized in so far as the inflammatory process is less frequently diffuse, but always is a focal one or generally affects only *one* kidney; a portion of the renal parenchyma still remains functionable, therefore.

Urine.—This may be explained, in the first place, because in such cases a complete anuria is never reached which is due to a suspended activity of the kidneys, and it is just as explicable that the urine may eventually be free from abnormal constituents. The latter, however, is but rarely the case; the appearance of the urine is usually turbid, and more or less *pus corpuscles* and many *bacteria* are found in the same; staphylococci, streptococci, bacterium coli communis, and proteus (Hauser) have been demonstrated as generators of suppuration of the kidney. The urine contains but little albumin at first in these processes, only enough to correspond to albuminuria in febrile (infectious) diseases or to the admixture of pus to the urine. *Urinary casts are usually entirely absent in the sediment*, while in some cases they can be demonstrated when, owing to the bacterial inflammation, the epithelia of the glomeruli and uriniferous tubules become necrotic and are excreted in larger quantities. In these cases we shall then find a larger amount of albumin than corresponds to the quantities of pus admixed. *Blood* is almost always absent in the urine in the suppurative, septic form of nephritis. However, if the origin of suppurative nephritis is traumatic, and especially if the suppurative nephritis is caused by stone, hæmaturia will not be absent.

Sudden profusion of pus in the urine points to the perforation of a renal abscess into the renal pelvis, or otherwise to the sudden detachment of a purulent plug which obturates the ureter and behind which the purulent urine accumulated (a cause for the origin of hydronephrosis). It is true, however, that the single or repeated excretion of large masses of urine may be caused by the perforation into the urinary canal system of a perinephritic abscess or of an abscess in the neighbourhood of the ureters or the bladder.

Renal Tissue.—If necrotic *particles of the renal tissue proper* become detached in abscesses of the kidney, it is possible that they may be passed

DIAGRAM FOR THE DIFFERENTIAL DIAGNOSIS

		ENGORGED KIDNEY.	ACUTE NEPHRITIS.
Ætiological factors:		Affections of the heart or lungs, thrombosis of the renal vein or of the inferior vena cava.	Severe cold, acute poisoning (cantharides, etc.), acute infectious diseases (scarlatina, pneumonia, enteric fever, diphtheria, sepsis, etc.), infection by specific bacteria.
Condition of the Urine:	Quantity:	Scanty.	Scanty.
	Colour:	Dark red.	Pale red to dark red, cloudy.
	Specific gravity:	High.	High.
	Blood:	Absent (outside of a few isolated red blood corpuscles).	Abundant.
	Albumin:	Moderate, varying amounts.	Abundant.
	Sediment (casts, etc.):	Moderate.	Marked.
		Urates, hyaline casts, a few red blood corpuscles.	White and red blood corpuscles, blood casts, epithelial casts (pure and metamorphized), urates.
	Total solids, urinary constituents:	Almost normal. Absolute quantity of urea slightly diminished; secretion of sodium chloride unchanged.	Great reduction of secretion of urea, of chlorides and phosphates in the urine.
Hypertrophy of the heart:		Caused by the original affection.	Almost without exception *absent*.
Dropsy:		Caused by original affection, rather stationary, especially at the lower extremities.	Marked (rarely entirely absent), location changing.
Uræmia:		Absent.	Frequent, especially in scarlatinal and cold nephritis.
Secondary symptoms:		General symptoms of engorgement (hyperæmia of the liver, etc.).	Symptoms of infectious diseases and intoxications.
Death caused by:		Heart failure, infarcts, etc.	Uræmia or inflammation of internal organs, pulmonary œdema, serous pneumonia.

Chronic Nephritis.			
CHRONIC PARENCHYMATOUS NEPHRITIS.	SECONDARY CONTRACTED KIDNEY.	CHRONIC NEPHRITIS, SLOWLY DEVELOPING FROM THE ONSET, "PRIMARY CONTRACTED KIDNEY."	AMYLOID KIDNEY.
Acute nephritis (scarlatina, etc.), slowly acting refrigeration (damp rooms, etc.), intermittent fever, phthisis.		Gout, saturnism, alcoholism, diabetes mellitus, arteriosclerosis and, probably, also syphilis.	Suppurations, especially caries, pulmonary phthisis, syphilis (especially amyloid contracted kidney). Rarely: Malaria, carcinoma, foot ulcers, etc.
Slightly less than normal, about one litre.	Abundant or at least normal.	Very abundant.	Almost normal, varying, increased in genuine amyloid kidney.
Of flesh-water colour, cloudy.	Rather clear.	Clear, pale.	Pale yellow, *clear*.
Slightly higher or normal.	Slightly below the normal.	Low.	Normal or *less*.
Usually present	Usually small amounts.	Red blood cells usually entirely absent.	*Absent*.
Abundant.	Moderately abundant.	Scanty.	Sometimes absent, but usually abundant.
Marked.	Rather marked.	Very sparse.	Usually entirely absent.
White and red blood-corpuscles, casts of all kinds, numerous casts affected by fatty degeneration.	Numerous casts of all kinds.	Especially hyaline casts (narrow and wide).	Sparse—hyaline and granular, also waxy casts, leucocytes.
Decrease of the secretion of solid constituents.	Great decrease of the secretion of solid constituents.	Great decrease of the secretion of solid constituents.	Normal, unless formation of urates decreased by constitutional deterioration.
Sometimes present.	Usually present.	Present almost without exception.	Absent, except in combination of amyloid degeneration with chronic nephritis.
Marked, cavity dropsy.	Moderate, developed as anasarca and cavity dropsy.	Usually not at all, but later present in cardiac insufficiency.	Usually quite marked.
Rather frequent.	Frequent.	Very frequent.	Absent, except in amyloid contracted kidney.
Marked pallor of the skin, retinitis, bronchitis, etc., inflammations of internal organs.			Retinitis absent, the symptoms of the underlying affection. (See ætiology.)
Uræmia or, more often, by inflammation of internal organs.	Uræmia, cerebral hæmorrhages, cardiac insufficiency, inflammation of internal organs.		The original affection, cachexia, exhaustion.

with the urine. This will furnish the possibility of a positive diagnosis, provided that uriniferous tubules, etc., can be demonstrated microscopically in these particles. However, the last-named occurrences are rarities; the diagnostic employment of the urine in this respect is not possible in the vast majority of cases.

The reaction of the purulent urine is *acid at first*, later it may become *alkaline* upon ammoniacal decomposition; however, the decomposition of the urea into carbonate of ammonia is not essential to infectious pyelitis and ascending nephritis. Schmidt and Aschoff reported that the ammoniacal decomposition of the urine was regularly absent even in cystitis, respectively pyelonephritis, which was caused by infection with the coli bacterium and was found only when other kinds of bacteria, for instance staphylococcus pyogenes, became active beside the bacterium coli or without the same.

Composition of the Urine in Pyelonephritis.—If *pyelitis* is supervened by an inflammation of the kidney due to contiguity of structure in such a manner that the pus cocci enter from the papillæ into the straight uriniferous tubules and gradually advance to the surface of the kidney inciting inflammation in the uriniferous tubules and in the interstitial tissue, *urinary* casts will also be found in the urine, although rarely, besides *epithelia* and *pus cells*, which are usually *imbricated* and which originate in the renal pelvis. The glomeruli being situated in the middle of the pus area and the epithelia being destroyed necrotically, a more frequent occurrence of casts could, *a priori*, be expected; but it should not be forgotten that the uriniferous tubules become obturated by the pus and the dead epithelia and thus prevent the deflux of the albumin-containing and cast-containing urine from the affected parts of the kidney. So soon as the latter is not the case throughout, casts and larger quantities of albumin than correspond to the admixture of pus will actually appear in the urine, and in such cases the pyelonephritis which has become associated with the pyelitis will now be diagnosticable.

If it is clear, after the above statements, that the conduct of the urine in suppurative nephritis, in contradistinction to the diffuse nephritides, allows in the great majority of cases unreliable diagnostic conclusions at most, the question remains whether the other symptoms of this affection do not offer better points of support for the diagnosis.

Pus Fever.—It is, in the first place, the *high fever, interrupted by chills*, which points to a purulent process in the body and at once indicates an affection of the urinary tracts as the cause of the intermittent fever in those cases in which the urine has assumed a purulent condition. The diagnosis becomes more certain when the region of the kidneys becomes painful spontaneously and also to pressure, or when (which requires an enormous abscess formation) a *fluctuating tumour in the renal region* becomes palpable from behind or from the abdominal walls.

Fluctuation in the Renal Region—Differential Diagnosis—Paranephritic Gravitation Abscess, etc.—Confounding this condition with paranephritic or with gravitation abscesses originating in the spinal vertebræ are liable to occur, especially as they, too, the same as true renal abscesses, perforate suddenly into the urinary

passages and evacuate the pus with the urine. The more circumscribed the abscess, the more probable is the presence of a renal abscess in cases of doubt, especially if no œdematous swelling of the lumbar region sets in—i. e., if the abscess formation is restricted to the kidney within its capsule, if pyuria and hæmaturia are present from the beginning, and if painfulness and flexion of the vertebral column are absent. If the *kidney* is recognised to be the seat of the fluctuating tumour, it is to be considered whether an *abscess of the kidney, hydronephrosis*, or *echinococcus* of the kidney are present; but these are differentio-diagnostical questions which are best considered in the discussion of the diagnosis of hydronephrosis and of renal echinococcus. The often-debated *paraplegia of the lower extremities*, which is explained either as a reflex paralysis or as a sequence of an ascending neuritis, and which is often observed, especially in renal abscess, is of very little diagnostic significance.

Ammoniæmia.—Dryness of the tongue and of the buccal mucous membrane, vomiting, general debility, stupor, and coma develop in some cases, a condition which, although remotely similar to the picture of uræmia, is differentiated from the same by the constant absence of convulsions, retinitis, and dropsy. The coma, after the manner of the diabetic coma, is sometimes associated with dyspnœic respiration (Senator). Treitz and Jaksch have previously designated the condition as "*ammoniæmia*," and interpreted it as the product of an intoxication with the ammonia of the decomposed urine. It is true, the idea of an auto-intoxication should not be rejected in such cases; but it is not at all clear as yet in what it may consist. On no condition are we entitled to ascribe it to an intoxication of the organism by ammonia; it is rather evident that there are several intoxicating substances of the decomposed urine or pus, possibly also bacterial toxines, the resorption of which produces fever and other pathological symptoms.

Symptoms upon which the Diagnosis Depends.—If the above-named manifestations are found in a case, the diagnosis of suppurative nephritis may then be made, and that with greater certainty if most of the symptoms are simultaneously present. I recapitulate them in the order of their diagnostic value: *Excretion of renal particles besides pus and eventually blood in the urine (rarely urinary casts), circumscribed tumour, fluctuating deeply (abscess) in the renal region without œdematous swelling of the adjacent tissue, perforation of the pus into the renal pelvis and transitory secretion of large pus masses with the urine, fever accompanied with chills, auto-intoxication with substances which are derived from decomposed urine and pus, paraplegia, pain in the region of the kidneys, alkaline reaction of the urine.*

Ætiology.—But even if several of the above-named manifestations coincide in the pathological picture, the rule should be observed not to establish the diagnosis of suppurative nephritis positively until the *ætiology* of the case has been thoroughly investigated and well considered, because the diagnosis becomes firmly established only by the demonstration of a cause which conditions the eventual suppuration in the kidney; the supposition of a "spontaneous" renal suppuration embodies *a priori* the "*germs*" of a wrong diagnosis.

Cystitis, prostatic hypertrophy, urethral stricture, and affections of the spinal marrow, with vesical torpor, most frequently give rise to suppuration in the kidneys. The bacteria which cause the inflammation and suppuration enter the urinary passages from outside (most frequently brought on directly by catheterization) in such cases and gradually extend upward in the direction of the urinary current, especially if the deflux of the same downward is obstructed and the bladder is not yet excessively distended—i. e., if it is still sufficiently contractile to throw a flood wave

to the ureter (Lewin and Goldschmidt). The origin of suppurative nephritis in *formations of concrement in the renal pelvis* (calculous pyelitis) and in *trauma* affecting the kidneys, is to be accounted for in a slightly different manner. A partial necrosis of renal tissue and portions of mucous membranes being brought about owing to these noxæ, a point of attack is created for the effect of the micro-organisms which are introduced by the blood current. Such a miscarriage of the micro-organisms and of their products into the kidneys by way of the circulation is indubitable, furthermore, in those cases in which a suppurative nephritis supervenes, in the course of *septicopyæmia*. It may be that in some cases of *long-lasting coprostasis* this contributes to the origin of suppurative nephritis (especially that which is caused by the bacterium coli) owing to the auto-intoxication which may eventually be connected with it. It should likewise be observed in the diagnosis of suppurative nephritis whether a *suppuration in the tissue adjacent to the kidney* (in the renal capsule, psoas muscle, peritonæum, or retroperitoneal connective tissue, furthermore, a gravitation abscess arising from hepatic and splenic suppurations or from gastric abscesses) *might not have continued into the kidney simply by contiguity.* Finally, cases have been observed in which the manner of invasion of the pyogenic agent (especially of the bacterium coli communis) into the kidney could not be demonstrated.

Differentiation of Suppurative Pyelitis from Renal Abscess.—The diagnosis of suppuration of the kidney is almost always difficult according to the above explanations, even if we proceed as cautiously as we possibly can. The diagnosis is easier upon accumulation of large masses of pus in the kidney; but here the question again suggests itself whether an abscess exists in the renal tissue proper or a simple *accumulation of purulent masses in the renal pelvis* is present, a question which usually cannot be decided unless exceptionally the secretion of urinary casts or even renal particles points directly to abscess of the kidney. The latter condition would also be indicated if an eventual fluctuation can be felt posteriorly from the renal region rather than from the abdominal walls. But if this differentio-diagnostic factor is to be applicable to the assumption of a renal abscess, it must be possible to exclude a paranephritic abscess. The diagnosis of the latter requires a more detailed discussion so that I will devote a special chapter to the same.

PARANEPHRITIS

Diagnosis.—Paranephritis is characterized by *pain* in the lumbar region, concentrated in the latter or radiating to the surrounding parts, intermittent *fever, tumour in the lumbar region with œdematous swelling of the skin above and adjacent to it*, more or less plainly palpable upon bimanual examination. The tumour does not become displaced during respiration and allows deep fluctuations to be recognised according to the size and stage of development of the paranephritic suppurative area; in most cases there is also pain upon contraction of the psoas muscle. The pus may perforate into the peritonæum, intestine, bronchi, etc., or even to the surface, unless operative interference causes it to be evacuated. *The urinary secretion remains absolutely normal* except in those cases in which perforation of the pus occurs into the renal pelvis (the urine does not contain pus at first which appears suddenly later) or, which is not infrequent, in which the paranephritis follows secondarily upon a pyelitis and pyelone-

phritis, an abscess of the kidneys, or upon a tuberculous infiltration of the kidneys.

Differential Diagnosis.—The diagnosis is not very difficult, at least in the majority of cases if the above symptoms are observed. The differentiation of paranephritic abscesses from *those of the kidney* is successful if we consider that a rather *diffuse* infiltration and œdema of the integument in the renal region occur in the course of paranephritis; that downward burrowing of the pus is very apt to occur, and that the urine shows normal conditions aside from the exceptions mentioned. The same as in suppurations in the kidney, so it is conversely possible that abscess formations in adjacent organs, especially in the *psoas* muscle, may be confounded with paranephritic abscesses, more so because an impairment of the function of the *psoas* is also observed in paranephritis. However, the latter does not from the onset become so markedly prominent in this case as in psoas abscess, in which, besides, the pain and the tumefaction are concentrated upon the course of the psoas. It is furthermore possible that *fæcal tumours* and *paratyphlitic abscesses* simulate paranephritis; in the latter affection, however, the seat of the abscess is so different from that of paranephritic abscess at the beginning that a mistake is scarcely possible. This is more apt to occur later when the pus in paranephritis has sunk towards the inguinal region. In such cases the *course of development* of the affection, the condition of the stools, etc., must determine the diagnosis in regard to the starting-point of the abscess.

If doubts are present as to the prevalence of *pus* at the bottom, an exploratory puncture is indicated.

HÆMORRHAGIC INFARCT OF THE KIDNEY

Hæmorrhagic infarct of the kidney arises in the well-known manner in valvular defects, by emboli from the heart, in atheroma of the arteries, etc. Traumas also, it appears, may lead to the formation of infarcts by a breaking of the wall of the renal artery. If, *in the presence of such a source for embolus, a sudden pain occurs in the renal region* (*owing to an expansion of the renal capsule*) *combined with hæmaturia or the latter alone, and if these symptoms entirely disappear again after a short while,* the diagnosis of hæmorrhagic renal infarct may be made. However, in by far the majority of cases of embolisms of the renal arteries their course is entirely *without symptoms during life* (as is shown by the autopsies).

Those cases are very rare in which hæmorrhagic infarcts of the kidney are diagnosticated during life. I succeeded in only a single instance, about twelve years ago, in making the diagnosis intra vitam in a case of insufficiency of the valves of the heart in which the autopsy confirmed the diagnosis. I regret very much that I do not possess the details of this case any more; but in another instance the symptoms were so marked that the diagnosis may undoubtedly be considered correct in spite of the favourable termination of the affection. The history of the case is as follows:

Case of Renal Embolism in the Course of Acute Endocarditis.—A carpenter, seventeen years old (admitted February 9th), suffered from the symptoms of an acute endocarditis (aortic insufficiency) which occurred in the course of an articu-

lar rheumatism. *The urine contained albumin and blood; the region of the left kidney was very sensitive to pressure*; no œdemas. The blood disappeared entirely after two days, the albumin after six days. On March 6th the patient complained of a feeling of constriction about the chest and of headache, and presented a slight elevation of temperature (on March 6th, 101.2° F. per axilla, on the 8th, 101° F., on the 9th, 100.3° F., on the 10th normal temperature); *on March 7th the urine, which until then was entirely normal, showed again blood and albumin and the region of the left kidney was again sensitive. The presence of blood and albumin in the urine persisted this time for seven days*; from that time on the urine became normal and the region of the kidney lost its sensibility to pressure. Chills were absent in the first as well as in the second attack.

I believe that in this case the diagnosis of *hæmorrhagic infarct of the kidney* was justified. At least I consider any other explanation of the occurrence of hæmaturia and albuminuria, which suddenly set in, with painfulness in the renal region, and rapidly disappeared again, in the course of an acute endocarditis, to be vastly less probable than the assumption of an embolus of the renal arteries.

TUBERCULOSIS OF THE KIDNEY—NEPHROPHTHISIS

Tuberculosis of the kidneys occurs in two essentially different forms: 1, *as a secondary acute miliary metastatic tuberculosis*, which represents a partial manifestation of a tuberculosis which is widely disseminated in the body, and which affects both kidneys but is not diagnosticable, because it does not cause appreciable symptoms, and 2, *as primary chronic tuberculosis localized in the kidneys*. In this second form there arise, besides gray nodules, owing to their becoming confluent and in consequence of progressing infiltration, larger nodes, which become caseous, which causes the gradual destruction of the renal tissue, forming larger areas of softening which are here and there connected with the likewise degenerated renal pelvis. This form of renal tuberculosis produces marked clinical manifestations and is easily diagnosticable as such. It is combined with tuberculosis of the ureter and bladder and almost exceptionally also with a caseous tuberculosis of the prostate, testicles, and epididymis, of the seminal vesicles, ovaries, and tubes, so that the process is also designated as *urogenital tuberculosis*. The mode of importation of the tubercle toxine is not quite clear as yet; probably it takes place from within by emboli through the blood passage, as well as from without through the urethra, vagina, prostate, and bladder.

Composition of the Urine.—The *specific alteration of the urine* is diagnostically the most important symptom of the affection. The urine, secreted in abundance, is usually *cloudy*, of *acid* reaction, rarely contains more *albumin* than corresponds to the admixture of pus and blood, and always leaves a sediment which consists of *blood, epithelia* (rarely urinary casts), *pus*, and *detritus*. In some cases it contains desquamated *shreds of connective tissue and elastic fibres*. If they are demonstrable, this state will be proof that a deep destruction of the ureter, of the renal pelvis, or renal parenchyma, especially of the interstitial tissue of the kidney and of the vessels entering the substance has taken place. However, these formations do not prove anything as to the *tubercular* character of the nephrophthisis, because they are bound eventually to appear in the urine in every instance of greater disintegration of the above-named tissues by suppuration or tyrosis (which seems to occur in the kidneys upon a non-tuberculous basis also). *But, on the other hand, the demonstration of tubercle bacilli in these cheesy, brittle masses or in general in the sediment of the urine of patients who are suspected to be affected with nephrophthisis* (especially on account

of the excretion of blood, pus, and detritus in the urine) *furnishes an absolute help in the diagnosis of renal tuberculosis.*

Diagnostic Value of the Proof of Tubercle Bacilli in the Urine.—It is not difficult to find the tubercle bacilli in the urine. *I was the first* to observe them shortly after Koch's discovery in a case of localized tuberculosis of the kidneys, and they have since been demonstrated by a great number of investigators, and by the same method which we employ for the demonstration of tubercle bacilli in the sputum. By this method we are enabled to make a positive diagnosis of a tuberculous process in the urinary passages. It is true though that a few isolated tubercle bacilli have since also been found in the urine in cases of disseminated miliary tuberculosis; but such findings are rarities in comparison to the enormous majority of cases in which *no* tubercle bacilli are passed with the urine in spite of evident disseminated tuberculosis in the body. Besides, the urine does not contain pus, etc., in these cases, in addition to the bacilli, as is the case in tuberculous nephrophthisis. The great value for the diagnosis of the demonstration of tubercle bacilli in the urinary sediment holds good, therefore, *if they are found on repeated examination, besides pus and detritus.* If repeated examinations of the urinary sediment for tubercle bacilli give negative results, we may, according to the example of Damsch, inoculate the pus excreted with the urine into the anterior chamber of the eye of a rabbit. If nodules appear in the iris in the third week, the tuberculous character of the urogenital affection is thus demonstrated. An error in the diagnosis may arise when tuberculous areas in the neighbourhood of the urinary passages (especially in the prostate) perforate into the same. The subsequent appearance of pus, shreds of tissue, and tubercle bacilli in the urine may simulate a tuberculous affection of the urinary organs. This may occur also when smegma bacilli mix with the urine, and their differentiation from tubercle bacilli presents great difficulties, as was explained on p. 131, and the diagnostic exclusion of which is necessary in all instances before the diagnosis of a tuberculous affection of the urogenital system can be made.

Other Symptoms.—All other symptoms, for instance pain in the renal region, etc., are far less significant for the diagnosis in comparison to the occurrence of tubercle bacilli in the urine. Nevertheless, the tuberculous character of the affection becomes probable if we succeed in demonstrating a thickening of the kidney, ureter, or of the bladder wall. Of extreme importance is the demonstration of a simultaneous *induration and tyrosis of the testicles* and especially of the *epididymis*, as well as of the *prostate*, in the male, of the *ovaries* and *tubes* in the female; the careful examination of these organs should therefore never be neglected in suspected cases of nephrophthisis (especially exploration per rectum and per vaginam). *Fever*, though slight, is probably always present, at least periodically; profuse diaphoresis and diarrhœa, also tuberculous joint affections and tuberculous meningitis complicate tuberculosis of the urogenital apparatus in its later stages. Pulmonary tuberculosis is also found at times, besides tuberculous tyrosis in the urinary organs. It may either form the source of the "excretion tuberculosis" in the urinary organs or it may follow secondarily upon a primary nephrophthisis. It need scarcely be mentioned that, the same as in other tuberculous processes, so also in urogenital tuberculosis, it is possible that an *amyloid affection* of the abdominal glands may develop in the course of the disease.

The diagnosis is sometimes rendered difficult or impossible in that the urine appears temporarily or constantly free from abnormal constituents. This may occur when a disintegration of the tuberculous infiltrates has not yet taken place in the

beginning of the disease, or when a transitory occlusion of the ureter of the affected kidney occurs by caseous material, thus preventing the passage of the characteristically altered urine from the affected kidney, while the other non-tuberculous kidney still secretes normal urine. In such cases there develops a *hydronephrotic* dilatation of the lumen of the urinary passages behind the obturation.

Although frequently *both* kidneys and ureters are affected by the tuberculous process, yet often enough it occurs that only *one* kidney is the seat of the tuberculous affection, as has been proved by recent, especially surgical, experiences. The decision of this point, however, is of the utmost importance for the therapy, especially on account of the necessity of a nephrectomy in the latter instance. Determining for the diagnosis is here: the restriction of pain and tumefaction in the region of the kidney and ureter to *one* side, the temporary excretion of *entirely normal urine* with transitory obturation of the ureter of the affected side, and, above all, *the result of cystoscopy,* which, if operation is considered, should always precede the latter. [The urine segregator should be employed in this instance.]

TUMOURS OF THE KIDNEY

The diagnosis of renal tumours almost always presents certain difficulties, and requires in all cases a systematic examination and a careful consideration of the differential diagnosis. The more or less extensive tumour of the kidney shows varying locations and extension, according to its origin in the upper or lower region of the kidney. The *development of the tumour from the upper regions of the right or left kidney* causes a bulging of the right or left hypochondriac region and later of the right or left iliac region. If the tumour has its starting-point in the *lower half of the kidney,* the bulging occurs at once in the iliac region. According to their location we should expect the principal extension of renal tumours to take place posteriorly, while in reality the palpable growth of the same occurs much more anteriorly, because posteriorly the psoas muscles offer much more resistance than the soft intestines, which are easily displaced by the growing tumour. But it should always be remembered, above all, if we want to diagnosticate a tumour of the abdomen as occurring in the kidneys, that it grows *postero-anteriorly,* displacing all movable organs of the abdomen (especially the intestines) forward or aside.

Position of the Colon as Regards the Tumour.—Such a locomotion by the renal tumour does not affect, in the iliac region, the ascending and descending colon, intestinal portions which, having no peritoneal cover in their posterior thirds, are firmly connected by cell tissue with the quadratus lumborum muscle and the corresponding kidney. Upon anterior or posterior enlargement of the kidney, therefore, the ascending and descending colon remain more or less unaltered in their natural position to the kidney—i. e., they are only slightly pushed forward or aside, and that in such a manner that usually *the ascending colon is placed from the right below to the left upward, the descending colon from the left above outward to the right within downward* as a portion of the intestine which passes *over* the tumour. *This varying conduct of the two portions of the intestine is*

caused, according to my opinion, by the natural direction of the fæcal displacement in the intestine, especially in the flexures of the colon, forcing the right flexure more within and the left more outward. But as the portion of the intestine which passes obliquely down over the tumour has a tympanitic sound in the natural gas-containing condition or in that in which it is to be filled artificially from below, in contradistinction to the absolutely dull sound of the tumour, this position of the intestine on the surface of the tumour undoubtedly obtains diagnostic significance, although deviations from this usual position of the colon occur quite frequently in tumours of the kidney, according to my experience, and I wish to lay especial stress upon this fact.

The position of the renal tumour to liver and spleen is such that the uppermost portion of the colon, respectively the flexure of the colon, passes *between* the tumour and the corresponding unchanged organs, that we are usually able to place the hand between tumour and liver or spleen and that below the same we feel the upper margin of the tumour retroflexed.

Bimanual Examination.—An important rule, never to be left unobserved in the diagnosis of abdominal and especially renal tumours, is, furthermore, *not only to palpate the tumour anteriorly but also bimanually from behind forward.* Tumours which originate in the kidney are then felt, especially upon deep respiration, to press more distinctly and to a greater extent towards the palpating hand than is the case in abdominal tumours which originate in other organs ("ballottement").

Immovability of Renal Tumours upon Respiration.—It is also generally assumed that *an immobility exists of the lower margin of renal tumours upon respiration*, because it is said not to follow the movements of the diaphragm. This is generally correct; but as the kidney is normally adjacent to the lower surface of the liver and spleen, a slight transmission of the movement of these organs, which is caused by the descent of the diaphragm, is unavoidable.

Renal tumours can be *displaced by palpation* only when it is a question of degenerated enlarged floating kidneys, in which the diagnosis usually encounters insurmountable difficulties, because the most essential characteristics are absent to recognise the tumour as a renal one.

Neuralgic Pains in the Abdominal Wall.—The normal position of the kidney is over the anterior branch of the twelfth dorsal nerve and over twigs of the lumbar plexus which extend in the abdominal wall; it is therefore not to be wondered at that an increase in volume of the organ causes *neuralgic pains in the abdominal wall* which may radiate to the thighs and genital organs. These painful spots, which are *anatomically* founded, are of diagnostic importance, while otherwise pains should not be considered of which the patient complains as originating in certain internal organs.

Differential Diagnosis.—*Tumours of the liver* differ from those of the kidney principally in so far as they also grow upward into the thoracic cavity, usually attack the left lobe uniformly with the right one; the ribs of the lower aperture of the thorax also bulge out much from above and the surface of the tumour below the costal arch appears as the direct con-

tinuation of the circumference of the thorax. But tumours of the kidney, on the other hand, can only in case they are very large cause a slight extension of the thorax and then only gradually from below, and they usually allow us to recognise a retroflexion of their apex or at least an indication of it between the tumour and the border of the liver. In tumours of the liver we also distinctly feel, without exception, the lower border as a more or less sharp edge, and can follow it in its natural course; the

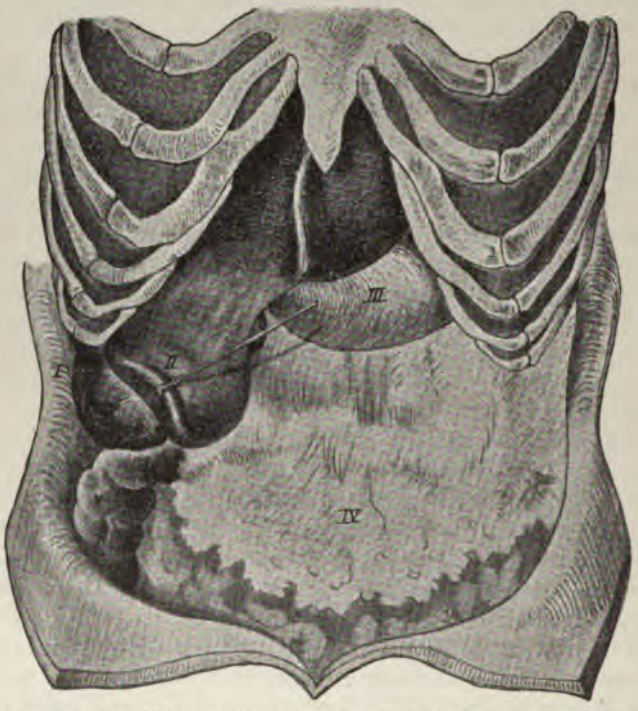


FIG. 11.—I, renal tumour; II, liver with corset lobe, covering the tumour of the kidney—the lower border of the liver turned up; III, stomach; IV, omentum; V, ascending colon displaced downward, flexed to the left in the middle third, continuing upward and to the right under the omentum.

border of the liver, upon deep inspiration of the patient, will almost always spring over the finger of the examining physician, and the downward displacement of the tumour is usually distinct and considerable.

Tumours of the spleen are less apt to be confused with renal tumours. They always, even if they are extremely large, show the normal shape of the spleen—i. e., the longitudinal axis of the tumour is directed from the umbilicus towards the axillary line, the anterior end can be distinctly felt as a point. The tumour can be easily grasped laterally, is very movable on inspiration, and is, almost always, situated *over* the colon (see p. 377). As to the remainder, I refer to the diagnostic rules which I laid down in the discussion of the diagnosis of hepatic carcinoma and splenic tumours (see pp. 202 and 227).

Besides tumours of the liver and spleen, the neoplasms on p. 376 are practically to be considered in the diagnosis of tumour of the kidneys: *Ovarian tumours* and enlargement of *retroperitoneal glands*, whereas intestinal tumours, according to the already-mentioned symptoms which are especially peculiar to them, can scarcely be mistaken for renal neoplasms.

Ovarian tumours differ from renal tumours in so far as they ascend from the pelvis. Accordingly, they generally cannot, if palpated from the abdominal wall, be demarcated inferiorly, but a connection of the tumour with the genital apparatus can be recognised upon examination per vaginam; they will force the intestinal coils *laterally and posteriorly*, so that a clear tympanitic percussion sound appears just in the neighbourhood of the renal region posteriorly. On the other hand, a differentiation of renal tumours from large **neoplasms** *originating* **in the retroperitoneal glands** is very difficult, in fact, almost impossible, as the growth of retroperitoneal tumours will cause exactly the same displacement of adjacent organs as will that of renal tumours—i. e., here, too, the intestines are laterally displaced, the descending or ascending colon passes downward over the tumour as a strip with tympanitic sound; the tumour itself is immovable. However, there exist some points of support to distinguish retroperitoneal tumours from those of the kidneys. To begin with, the urine, in the latter, shows, at least usually, changes, generally *admixture of blood*; but it should not be forgotten that, on the one hand, the urine in renal neoplasms may be normal during the entire course of the disease, and, on the other, changes in the urine may also occur in retroperitoneal tumours owing to their pressure upon the ureter and the renal vessels. It is of greater importance differentio-diagnostically, therefore, that renal tumours, even if they are large, are always situated more laterally from the vertebral column than retroperitoneal tumours.

After the tumour, by means of the above characteristics, has been recognised as belonging to the kidney, the question suggests itself, What is the character of the tumour? a question usually much easier to decide than to determine that an abdominal tumour is a tumour of the kidneys. Diagnostically to be considered in this case are, as will be discussed in particular, renal cysts, especially hydronephrosis, echinococcus, carcinoma and sarcoma of the kidneys; whereas the other renal neoplasms: adenoma, fibroma, myxoma, syphiloma, lipoma, etc., are of no clinical, but only of pathologico-anatomical interest.

Case of Sarcoma of the Right Kidney, which was completely Covered by a Corset Lobe of the Liver.—To illustrate the above diagnostic rules, I report the case history of an instance of *sarcoma of the right kidney*, which was observed in the Julius Hospital, and which was difficult to diagnosticate.

The patient was a woman, fifty-four years old, who was healthy until eight months previous to admission to hospital, when she had an attack of hæmaturia (according to the patient's statement), which, however, rapidly disappeared; the urine remained entirely normal, especially during the three months of her stay in the hospital. One month previous to her admission to the clinic she had pains in the arm, near the shoulder joint, upon active and passive movements. She had no other morbid disturbances, except inclination to constipation.

The physical examination showed poor nutrition, the subcutaneous layers of fat had disappeared; at the upper end of the humerus sensitiveness and a swelling hard

to the touch, which gradually increased; one month before death spontaneous fracture at this place; no glandular swelling. All the organs were normal. *Examination of the abdomen: In the right abdominal region a tumour the size of a small child's head visible and palpable.* The stone-hard tumour commenced immediately below the costal arch, *its upper end could be grasped indistinctly.* To the right the tumour extended to the lateral contour of the abdomen, to the left almost to the median line, downward to within one finger-breadth below the umbilicus. *It was remarkable that the neoplasm could be felt anteriorly in the depth through a softer surface, but laterally immediately below the abdominal walls as a hard tumour* (see Fig. 11, p. 378). Upon closer examination it could be determined that it was possible slightly to raise the softer surface as a *sharp-edged lobe* from the deeper-seated tumour, and that the sharp-edged lobe extended triangularly into the lower third of the abdomen, to rise towards the median line into the border of the liver. The percussion sound over the tumour was absolutely dull; during the first month of observation a tympanitic sound could occasionally be noted over the right lateral portion of the tumour; but by inflating the rectum with air no portion of the intestine could be filled over the neoplasm. The hard tumour was immovable on respiration, but it could be displaced slightly by pressing it forward from the renal region, in which case the soft lobe situated above the tumour did not move.

The *diagnosis* could be made, after some deliberation, of a *tumour of the right kidney,* because the tumour could easily be palpated from the renal region direct, and because it was immovable on respiration, and also because the upper contour of the tumour could be grasped below the costal arch posteriorly. It was remarkable that the intestine, especially the ascending colon, did not pass over the tumour, but a tympanitic sound was only demonstrable for a short distance laterally over the same, and also that the urine did not show any changes during the entire course of the affection. An ovarian tumour could be excluded because the neoplasm did not extend down to the ovarian region; a hepatic tumour could be excluded because the neoplasm, although it passed into the liver dulness which superiorly remained approximately within the normal boundaries, remained entirely immovable during respiration; an intestinal tumour was not likely to be present because no obstacles were observed in the passage of the intestinal contents; a retroperitoneal tumour, finally, was at least improbable in comparison to a renal tumour because the neoplasm did not extend beyond the median line into the other side, but was developed towards the right renal region posteriorly and laterally. The sharp-edged, soft lobe situated over the tumour *could not possibly represent anything else, owing to its triangular shape and its passage into the sharp liver border, than a malformed liver,* and nothing else, under the circumstances, than a *tight-lace lobe* of the liver with a lacing furrow, which rendered it possible to palpate the upper contour of the tumour and to grasp the same posteriorly below the costal arch.

The *diagnosis* was accordingly: *Tumour of the right kidney covered by tight-lace liver, metastasis in the left humerus.* The patient died with the manifestations of marasmus. The autopsy (Rindfleisch) showed: *Sarcomatous tumour of the right kidney, dislocation of the right lobe of the liver (tight-lace liver), situated over the renal tumour,* covering the same and showing it only at the lateral border of the tight-lace lobe; downward displacement of the ascending colon, so that no portion of the intestine was situated over the tumour; the middle third of the ascending colon was bent to the left, situated below the renal tumour, connecting with its lower contour. Metastasis of the retroperitoneal glands and of the left humerus with fracture of the latter. The cut on page 378 illustrates plainly the position of the renal tumour in comparison to the liver and other abdominal organs.

CYSTIC TUMOURS OF THE KIDNEY—RENAL CYSTS, HYDRONEPHROSIS, ECHINOCOCCUS

Common to cystic tumours of the kidneys, to hydronephrosis, and to renal echinococcus is the soft, fluctuating condition of the tumour observed in these affections.

RENAL CYSTS

The *cysts* proper (of which the smaller ones, originating in the distended uriniferous tubules and situated especially in the cortical substance, are of very frequent occurrence in chronic nephritis) attain only exceptionally such a marked size that they become an object of diagnosis. But they may sometimes reach the size of a child's head; they occur in *one* or *both* kidneys. They are sometimes found in adults, but usually it is a question of small children in whom cystic degeneration of the kidneys appears congenitally and which, upon more marked development of the affection, may be an obstacle at birth.

The *diagnosis of cystic kidney* is possible only in very well-developed cases, best when it is a question of the congenital form, in which other malformations are known to occur; hydrocephalus, defective development of the extremities, malformation of the urinary bladder, etc. In adults we must think of renal cysts if a bilateral tuberous renal tumour with elastic, globular prominences is felt on the surface. It has happened several times that upon these symptoms the diagnosis was made *intra vitam*, and was confirmed by the autopsy. The urine generally contains albumin (and blood), as interstitial nephritis and nephrolithiasis were usually found associated with a cystic kidney, or probably were the cause of its origin in most instances. But it may also be that the urine is of a normal condition during the entire course of the disease. It is obvious that, in case a fluctuating tumour is felt at all, hydronephrosis and renal echinococcus, the diagnostic characteristics of which will be discussed presently, should always be taken into consideration in the diagnosis.

HYDRONEPHROSIS

If an obstruction exists below the renal pelvis to the passage of urine, an accumulation occurs above the obstacle owing to the continuous secretion of the kidney, and with it an expansion of the affected portion of the urinary passages takes place. Therefore, according to the higher or lower position of the obstruction, either the renal pelvis alone, or the latter and part of the ureter, or the bladder, together with both ureters and the renal pelvis, are distended by the accumulating urine. The urine which has collected in the renal pelvis, gradually exerts an atrophic pressure backward upon the renal substance, therefore, the secretion of urine ceases gradually and the mucous membrane of the renal pelvis will soon secrete nothing into the sac but mucus and serum or, upon supervening inflammation, pus. This condition is designated as *hydronephrosis*, and it cannot become the subject of diagnosis until the tumour has assumed large dimensions. But a diagnosis usually offers great difficulties even if a tumour containing fluid can be palpated, and confusions with other soft abdominal neoplasms, especially ovarian cysts, are very apt to occur.

Symptoms of Hydronephrosis.—The symptoms especially peculiar to hydronephrosis are by no means characteristic. The *excretion of urine* is quite varying. It may be that, in unilateral hydronephrosis and with complete occlusion of the ureter, the quantity and condition of the urine may be entirely normal—in fact, polyuria may be present, because the other healthy kidney, which under these circumstances becomes hypertrophied, functionates vicariously to the fullest extent. But in bilateral hydronephrosis oliguria or anuria may exist and uræmia may set in as a consequence. Accordingly, therefore, whether the obstacle to the flow of urine becomes manifest, or whether it *is temporarily overcome*, oliguria alternates with profuse excretion of urine, and for the same reason does the size of the tumour vary (" *periodic*," " *intermittent* " hydronephrosis)—i. e., an intermittent filling and emptying of the hydronephrotic sac occurs if a valve-like occlusion forms in the

lumen of the ureter, which is temporarily suspended by the pressure of the accumulating urine from behind, also if, as has been mentioned, hydronephrosis occurs in a movable kidney by torsion of the ureter, and if, upon retrotorsion, the flow of urine again takes place without obstruction. It is also conceivable, in view of such conditions, that the *quality* of the urine varies; the urine is normal at one time, at another time it contains mucus, pus, blood, and casts, and it is cloudy. The general health is but little disturbed; at most *fever* occurs, caused by pus formation in the hydronephrotic sac. *A compensatory hypertrophy of the heart* has also sometimes been observed.

Differential Diagnosis.—As to the *differential diagnosis*, the best mode of procedure is *first to determine whether the tumour actually belongs to the kidney*; all previously mentioned characteristics of renal tumours are to be considered in this case, and above all, the *position of the intestine (especially of the ascending and descending colon) in comparison to the tumour.*

Ovarian Cysts.—The hydronephrotic tumour, if retroperitoneal, presses the intestine forward or to the side, whereas ovarian cysts press it backward. But it should be expressly mentioned that portions of the intestines are found between the tumour and the abdominal wall in ovarian cysts also, the same as happens in renal tumours. *The movability of the tumour* is conclusive diagnostically in so far as *ovarian cysts, at least those that are not very large, are usually subject to decidedly passive movements, while hydronephrotic sacs are not.* However, it should not be forgotten that this rule does not hold good for hydronephroses which relatively often develop in *movable kidneys* due to partial torsion of the organ (Landau). It is further in favour of hydronephrosis, in contrast to ovarian cysts, that a hydronephrotic sac is palpable according to the starting-point of its formation in the renal region, especially upon bimanual examination, while ovarian cysts situated near the uterus are always especially easy of palpation from the vagina. Besides, in the latter case it may eventually be possible to feel the uterine adnexa pass into the tumour, and to observe the pedicle of the ovarian cyst especially from the rectum.

If these differentio-diagnostic measures are not sufficient, an examination is to be made with the patient in chloroform narcosis (passing the entire hand into the rectum), in order to be able better to palpate the contours of the tumour and its starting-point. If this should not be sufficient either, nothing remains but to make an *exploratory puncture*, which, however, should not be done without urgent reason, as the chances for operation of ovarian cyst become worse if previously an often unavoidable suppuration of the cyst occurs after the exploratory puncture.

Condition of the Contents of the Hydronephrotic Sac in Contrast to that of Ovarian Cyst.—The *fluid* which is obtained by puncture by means of a thin trocar differs in ovarian cysts considerably from the contents of the hydronephrotic sac in the majority of cases. *The fluid from ovarian cysts is usually viscid, colloid,* of *high specific gravity* (about 10.20), contains much albumin, and also paralbumin and metalbumin; furthermore, in the sediment, there are *epithelial casts of normal and colloid appearance.* The hydronephritic fluid, on the other hand, shows in the sediment *stratified pavement epithelia* from the renal pelvis, is of *low* specific

gravity, rarely colloid, and contains, above all, besides albumin, very many urinary constituents—*urea, uric acid,* etc. However, the chemical constituents of the fluid are by no means pathognomonic of one or the other of the two affections. For the characteristic constituents of the urine disappear gradually in older hydronephrotic sacs, so that urea and uric acid are no longer demonstrable; on the other hand, considerable quantities of urea are also found in the contents of ovarian cysts. Metalbumin and paralbumin, which were for some time considered characteristic of the latter, have also been demonstrated in the fluid from hydronephrotic sacs. This proves that not too much should be expected for the differential diagnosis from the result of an exploratory puncture in general.

The facts that disturbances of menstruation, swelling of the breasts and secretion of milk occur sometimes in ovarian cyst, while disturbances in the secretion of urine take place in hydronephrosis, may give direction to the diagnosis.

Besides, with ovarian cysts, hydronephrosis may be confused with *ascites, mesenterial cysts, renal abscess, echinococcus of the kidney,* and *renal cysts.*

Ascites.—In regard to *ascites,* a wavering in the diagnosis can only be possible in bilateral hydronephrosis. But the differential diagnosis is easy even then, owing to the great movability of the ascitic fluid. The lateral dull areas which are caused by ascites usually disappear very rapidly and completely if the patient assumes the lateral posture, while the dulness of hydronephrosis remains.

Sacculated Peritoneal Exudate.—Slightly less easy is the differentiation between hydronephrosis and *sacculated peritoneal exudates.* However, these latter fluids always show, aside from the anamnetic data, pains, etc., more diffuse and irregular boundaries than the sharply defined hydronephrotic sacs; it is also mostly possible in exudates, in contradistinction to the latter, that the tympanitic sound of the subjacent intestines can be observed by strong percussion.

Renal Abscess.—Hydronephrosis is easily differentiated from the explicitly described *renal abscess* which also, like the former, forms a fluctuating tumour. For in renal abscess there exists a more marked fever, accompanied with chills, and the investigation of the ætiology of the case must give a reason for the presence of suppuration if the diagnosis of renal abscess shall rest on a firm basis.

Renal Cysts.—*Renal cysts,* however, cannot be differentiated diagnostically from hydronephrosis. It is true, if a fluctuating renal tumour is felt in the newborn, that we may first think of the existence of a renal cyst, especially if the fluctuation is only felt posteriorly in the lumbar region; however, it should not be forgotten that hydronephrosis may also occur in the newborn due to congenital anomalies of the ureters, and that in cystic kidney the renal pelvis may also be widened; in such cases, of course, a differential diagnosis is impossible.

Renal Echinococcus.—Finally, it is a question whether *echinococci of the kidneys* present any differentio-diagnostical criteria in contrast to hydronephrosis. In referring to the special diagnosis of renal echinococcus, I now wish to remark only that the echinococcus becomes diagnosticable only upon the passage of echinococcic cysts in the urine, and even

then care should be taken in the diagnosis, because echinococcic cysts may perforate into the urinary passages from other sources. If we decide upon puncture of a tumour which is recognised as a renal cyst, the examination of the fluid thus obtained will remove all doubts, if it does not contain any albumin and if stratified cystic membrane parts or hooklets can be demonstrated. Regarding the differentiation of hydronephrosis from *mesenteric cysts*, I refer to the diagnostic criteria given in the discussion of the latter (p. 421).

Application of the Ætiology to the Diagnosis.—Finally, it must always be considered a rule in the diagnosis of hydronephrosis never to make it with certainty unless *ætiological* reasons speak in favour of the formation of the same—i. e., a preceding lithiasis, pyelitis, cured peritonitis, movable kidney, or abdominal tumours, which, owing to their position, may compress the ureter (especially carcinoma of the bladder which obstructs the entrance of the ureter). Bilateral hydronephroses are brought about either by the above ætiological factors, if accidentally both ureters become occluded, or by causes which *regularly* prevent the flow of urine from both ureters, such as retroflexion and prolapse of the uterus, prostatic hypertrophy, and urethral stenosis. But the most careful taking of the history and examination does *not* in some cases supply any cause for the formation of hydronephrosis. Even the autopsy sometimes fails to answer the question as to its occurrence.

ECHINOCOCCUS OF THE KIDNEY

There can only be a question of diagnosis of renal echinococcus if it forms a large, firmly elastic, fluctuating cyst. But this occurs only in less than one half of the cases, and even when a large cystic tumour is felt, still the diagnosis usually presents great difficulties, for the tumour in such instances has no characteristic properties. The hydatid thrill which for some time was considered pathognomonic, is absent in some of the cases of echinococcus, and, on the other hand, it may also be observed in other cysts. After the fluctuating tumour has been *diagnosticated a cystic tumour of the kidney*, in accordance with the previously stated rules, the diagnosis of the cyst as renal echinococcus becomes feasible: (1), by the spontaneous perforation of the echinococcus cyst into the stomach, intestine, or even bronchi (and evacuation of the cystic contents with the vomit, fæces, or sputum), or, most frequently, by a perforation into the renal pelvis, and the passage of the cystic contents with the urine, and, (2), by the result of the exploratory puncture of the cyst. The fluid obtained in the latter case is entirely *water-white*, reacts alkaline and contains, which is very remarkable, *no* (or but very little) *albumin*; less remarkable is the eventual presence of succinic acid, inosite, reducing substance and of much sodium chloride. Positively demonstrative of the echinococcic character of the cystic fluid is only the occurrence of *hooklets, scolices* and *transversely striped membrane parts in the sediment.* It may sometimes occur that, besides the characteristic echinococcic elements, a bloody or purulent contents of the cysts is found.

Consequences of Spontaneous Discharge of the Echinococcus Cysts.—If the contents of the echinococcus cyst discharges into the *urinary passages*, the excretion of the echinococcus cysts takes place with attacks of renal colic, and it may happen that the urethra finally becomes occluded with particles of the echinococcic cyst.

Then, either spontaneously or artificially, a clear, or milky, or blood-tinged urine is voided, which mostly contains albumin and also the characteristic elements of echinococcus formation. A microscopical examination will show, besides hooklets and membrane parts, pus corpuscles, blood corpuscles, triple phosphate crystals, and other formations of inferior diagnostic significance. Of importance is the decision of the question whether the palpable cystic tumour *becomes* correspondingly *smaller* with the discharge of the above-named sediments. However, the determination of a diminution of the tumour is by no means a proof of the presence of a renal echinococcus, because a rapid decrease of the tumour may naturally also be caused by the perforation of an echinococcus tumour from an organ adjacent to the urinary passages into the latter. However, it is also possible that, in place of the expected diminution, on the contrary an *enlargement* of the fluctuating tumour will occur after the perforation, inasmuch as an acute hydronephrosis may secondarily be brought about in consequence of an occlusion of the ureter by the discharged echinococcus particles, thus producing a swelling of the fluctuating renal tumour.

In conclusion, it may be emphasized that, the same as in other renal cysts, so also in renal echinococcus, the *immovability of the tumour* is of the greatest importance in the differential diagnosis between echinococcus and other cysts in the abdominal cavity, especially ovarian cysts. However, exceptions to this rule occur, because, evidently, in consequence of the growth and the gravity of the echinococcus cyst in the kidney, the organ gradually becomes movable or the echinococcus develops in a previously movable kidney. The differential diagnosis may become an extremely difficult task under such circumstances.

SOLID TUMOURS OF THE KIDNEY—CANCER OF THE KIDNEY—SARCOMA OF THE KIDNEY

Of the solid tumours of the kidney, only sarcoma and carcinoma are to be considered diagnostically, whereas the other neoplasms of the kidney, fibroma, lipoma, myxoma, adenoma, gumma, etc., are of no clinical, but only of a pathologico-anatomical interest.

CARCINOMA OF THE KIDNEY

Renal carcinoma, a practically rare affection, remains concealed sometimes during the life of the affected patients, especially when it is a question of secondary development of the carcinoma. The larger renal cancers, however, form such great tumours that the examining physician cannot fail to observe them upon palpation of the abdomen, and they are characterized as renal tumours by their immovability, by their position in relation to the liver and spleen and, above all, to the intestine, by their growth from behind, in short, by the entire list of previously described manifestations.

Consistence of Renal Cancer.—Their *consistence*, in contradistinction to the just-described renal cysts, is *hard*, firm; sometimes, it is true, they are *soft in some areas*, owing to the fatty degeneration of the carcinomatous nodes, or even fluctuating, owing to hæmorrhages which take place into the interior of the highly vascular tumour. The surface of the carcinomatously degenerated kidney is, according to the seat and the dissemination of the cancerous masses, either smooth or distinctly tuberous.

Besides the most important symptom, the tumour, the demonstration of which alone is sufficient to render the diagnosis of renal carcinoma feasible, the *pains* in the renal region and certain *changes of the urine* are to be considered only secondarily in a diagnostic respect. The urinary changes, it is true, may often be entirely absent, but, if present, they serve to render the diagnosis considerably more certain. As renal cancers are distinguished from other carcinomata by their large amount of thin-walled vessels, there occur, besides the above-named intrarenal hæmorrhages, extravasations of blood into the renal pelvis in about half the cases and, with these extravasations a **periodic hæmaturia,** either at the onset of the disease or, the more frequent case, during its later course.

Condition of the Urine.—The passage of bloody urine occurs spontaneously, sometimes, possibly, owing to traumatic effects upon the renal tumour. The urine may be entirely normal—i. e., free from any admixture of blood or albumin between the attacks of hæmaturia. In fact, the secretion of urine from the diseased kidney may cease entirely if the growing cancerous mass obstructs the ureter and thus eventually secondarily produces also hydronephrosis. Pus corpuscles and stratified pavement epithelial cells are contained in the sediment only if the carcinoma originates in the renal pelvis, or if the mucous membrane of the renal pelvis or of the ureters also become affected in the course of the disease. The latter is very apt to occur because renal carcinomata are very much inclined to grow towards the point of least resistance—i. e., in the direction of the ureters. In such cases not only the symptoms of a simultaneous pyelitis and hæmaturia occur, but, eventually, the *detachment of carcinomatous masses, which may appear in the urine,* a condition which is theoretically possible, but which up to now could not be utilized free from all doubt in practice as a pathognomonic symptom in the diagnosis of carcinoma of the kidney.

Secondary Symptoms of Renal Carcinomata.—A carcinomatous mass may also proliferate, the same as into the urinary passages, into the renal veins, and, finally, reaching the inferior vena cava, cause metastasis *in the lungs*; metastases also occur secondarily in other organs. Pressure of the tumour upon the large abdominal veins produces varices and *œdema of one or both lower extremities, varicocele and ascites,* pressure upon the nerves which are within reach of the tumour, *pain in the lower intercostal spaces and in the femurs.* Spreading of a renal tumour to the vertebrate column and to the spinal cord may cause *paraplegia,* with violent pains.

A very important diagnostic symptom of cancer of the kidney, especially on account of its malignant character, is *cachexia,* which occurs in the course of the affection. However, in some cases the condition of the strength remains relatively good for a remarkably long period.

Differential Diagnosis.—It has already been explicitly stated in the discussion of the differential diagnosis of renal tumours in general, that carcinoma of the kidney may be confused with other abdominal tumours, also how such mistakes can best be avoided. I only wish to mention here also that the differential diagnosis of renal carcinoma almost always causes considerable difficulties, and that a confusion with ovarian tumour has been particularly frequent. Determining in the differential diagnosis between renal and ovarian tumours are the movability of the latter (carcinoma is only rarely found accidentally in movable kidneys)—I have only seen one

case—their development from the pelvis, in contrast to the growth of renal carcinomata from the lumbar region, the connection of ovarian tumours with the uterus, and the simultaneous disturbances of menstruation in the same. Intercurrent hæmaturiæ render the presence of a renal carcinoma probable, and it is also important to remember that, contrary to other cancers, it occurs not only in advanced age but also in youth and *especially in children*. Statistics show that more than one third of all renal carcinomata occur between the first and the eleventh years.

SARCOMA OF THE KIDNEY

The same as carcinoma of the kidney, so does sarcoma occur secondarily and primarily, in the latter case principally in children. About two thirds of the so far reported primary sarcomata of the kidney occurred in children of less than ten years of age. The tumour, which reaches a considerable size and is almost always unilateral, is mostly soft, sometimes even fluctuating, owing to the formation of areas of softening, uneven on the surface.

Differential Diagnosis between Sarcoma and Carcinoma of the Kidney.—The question whether the tumour is of a carcinomatous or sarcomatous character—another question is not to be considered diagnostically—can be decided with some degree of probability, as youthful age, the absence of hæmaturia, and the relatively smaller disturbance of the general health are decidedly more in favour of sarcoma. However, these characteristics may not always be depended upon, as is proved by the previously reported case of renal sarcoma, and as, on the other hand, the above-named symptoms, which generally speak for sarcoma, also occur in carcinoma. The diagnosis remains doubtful, therefore, until sarcoma cells appear in the urine, or until an exploratory puncture and a microscopical examination of tumour particles thus obtained remove any doubt as to the sarcomatous character of the tumour. Not the softest areas should be selected for exploratory puncture, because then only fluid will be obtained for examination, or tissue which is so changed that the diagnosis cannot be made from the microscopical finding.

If we have to deal with primary sarcomatosis of the skin or of other parts of the body which are accessible to direct examination, the occurrence of the solid renal tumour in the course of the disease will naturally point at once to the fact that the renal tumour is of a sarcomatous nature. However, the secondary development of sarcomata in the kidneys may take place without any symptoms, as I have recently again been taught by a case observed in the Julius Hospital, in which, in spite of profuse formation of secondary sarcomatous nodes in the kidneys, neither the volume of the organ increased in size nor any change of the urine indicated a secondary sarcomatosis of the kidneys.

The differential diagnosis between carcinoma and sarcoma is generally of no value until a microscopical examination of the tumour tissue obtained by exploratory puncture has taken place. The same holds good also for *renal adenoma*, which has recently been found more frequently and the close connection of which with carcinoma does not permit of a differential diagnosis during the life of the patient.

NEPHROLITHIASIS—FORMATION OF CONCREMENT IN THE KIDNEYS AND URETERS—[STONE IN THE KIDNEY.]

According to the size of the concretions found in the kidneys and ureters, are they distinguished as *sand*, *gravel*, or *stones*. Common to all these formations is the fact, first discovered by Ebstein, that they do not represent simple precipitations of stone-forming masses (salts, etc.), but rather upon dissolution of the latter leave an organic framework which is formed either in concentric layers or more irregularly, and into which the stone-

forming substances are deposited. It appears that the framework, which consists of albuminous substances, owes its origin to an inflammatory irritation of the epithelia of the urinary passages and to deficient secretion of the inflammatory products by the urine.

Symptoms of Use in Diagnosis—Pains.—It is rare that renal stones do not cause any symptoms or disturbances. The patients usually complain of *pains* in the renal region which radiate downward towards the bladder and the femurs, and which are aggravated in particular by movements which shake the body (horseback-riding, etc.), and, on the other hand, are made easier by certain postures of the body which are taken purposely. Larger quantities of sand-like or slightly larger sediments from the size of a poppy to that of a lentil seed are passed with the urine in shorter or longer intervals and precipitated in the freshly voided urine.

Condition of the Urine.—The urine shows at the same time sparse or even profuse, sometimes periodically recurring, admixtures of blood and, upon inflammatory irritation of the renal pelvis, of pus corpuscles, mucus and desquamated, often stratified, pavement epithelia—in fact the characteristics of the urine in pyelitis; the *reaction* of the urine is almost always *acid* in such cases. If the stones in their passage through the ureter meet with an obstacle, or if the larger stone occludes the ureter, the classical picture of **renal colic** will occur: A lancinating pain, radiating from the renal region along the ureter to the bladder and the urethral opening, extremely violent, which diffuses to various points, especially to the testicles, but which is concentrated mostly upon the region of the urinary passages. There exists at the same time a violent, continuous desire to urinate, but, nevertheless, usually only very few drops of a concentrated, mostly bloody urine full of sediment can be voided. Of general symptoms there are observed: Sensation of anxiety, cold perspiration, anxious facial expression, vomiting, weakness of the pulse, chills. The paroxysm ceases with the entrance of the stone into the bladder, with its passage in the urine, or with the return of the stone from the upper opening of the ureter into the renal pelvis.

After the passage of the stone, a profuse discharge of urine follows—i. e., of that quantity of urine which accumulated above the stone obstructing the ureter and causing the temporary distention of the same and an acute hydronephrosis. In other unfortunate cases the occlusion of the urinary passages by stone is followed by *uræmia* when both kidneys are affected and both ureters are obstructed, or, in the absence of one kidney, if the lumen of one ureter becomes occluded. The obvious consequence under such circumstances is complete anuria, which is found, sooner or later, to lead to uræmia, but which, it is true, in exceptional cases, as has been positively determined, may persist for weeks without the occurrence of uræmia. It is exceptional, also, that anuria occurs in connection with an attack of colic if only one of the two ureters is occluded. The cause of this condition is either due to the fact that the other kidney was also diseased even before the impaction of the stone or that the healthy kidney with its unobstructed ureter (probably due to radiating irritation of the splanchnic nerve, the vascular nerve of the kidney) has temporarily entirely suspended its activity. Other consequences of ureteral occlusion by stones, tearing of a ureter with following peritonitis, perforation of the stone into the intestine, to the external skin, etc., are only very rarely to be expected (I have seen only one case of external perforation of the stone).

Although the above-described symptoms allow a very probable conclusion upon the presence of nephrolithiasis, a *positive* diagnosis by reason of the same cannot be thought of. For, it may occur, although rarely, but undoubtedly without the presence of stones and without any anatomical change in the kidneys, that paroxysms of pain occur which are entirely like renal colic and which are due either to nervous irritations originating in the central nervous system (especially in the *crises nephrétiques* of tabetic patients), or to a neuralgia of the peripheral nerves (*nephralgia*); but it may also be that other foreign bodies besides stones, such as coagulated blood, (which, if formed in the ureter, are discharged as worm-like formations), echinococcus cysts, etc., will cause renal colic upon passing into the ureters. The diagnosis becomes certain only when (which will be very rarely possible) a stone can be palpated in the renal pelvis or if even the rubbing of several stones can be distinctly felt or when, the usual case, *concretions are passed with the urine.* Unless the latter are discovered before the onset of renal colic, the diagnostic question may sometimes suggest itself whether the paroxysm of colic is a renal one at all. It is almost always easy to recognise the latter as such from the seat of the pain and from its radiation, even if renal colic is combined with vomiting. The matter becomes difficult if, as a consequence of renal stones, no renal colic pains at all, but only gastric symptoms (vomiting and cardialgia), manifest themselves by way of nervous radiation. In such cases only the *examination of the urine for concretions* can elucidate the diagnosis. [The value of the X-rays is here of great use, as stones have been seen in the kidney by its employment.]

Examination of Urinary Concretions.—Freely voided urine is at first caused to form a sediment, then the lowest portion of the sediment which has formed in the test tube, is removed with a pipette for further examination. [The centrifuge should be employed.] The grating of the sediment upon glass and pipette usually betrays the fact that we are dealing with hard, sharp-edged bodies. The presence of stones in the urine becomes entirely clear if larger concretions have passed the urethra. The microscopic preparation will then show, besides the characteristic crystals of uric acid, calcium oxalate and cystin, which form renal sand, pus corpuscles, epithelial and blood corpuscles, especially when the affection persists for some time, owing to a supervening pyelitis. The macroscopically visible concretions, to determine their constituents, require the preparation of thin, transparent sections for microscopical investigation, and a more precise chemical examination.

It may be stated here in general only as a diagnostic guide that *uric-acid* concretions, by far the most frequent stones, present a smooth or only slightly uneven surface; they are of a yellowish-brown or reddish-brown colour, and are hard. If particles of such a stone are heated with nitric acid, upon addition of caustic potash or ammonia, the well-known purple coloration (murexit test) appears. *Oxalate stones* are very hard, of a brownish to black colour, and usually show a rough, nodular surface ("mulberry calculus"). They are soluble in mineral acids; upon addition of ammonia to the solution they crystallize into octahedral crystals of calcium oxalate. *Phosphate stones*, consisting mostly of calcium phosphate and ammoniated phosphate of magnesium are relatively soft, of a white or clay colour, and have a sand-like, rough, often glistening, surface. The various stones are usually not composed of only one of the above-named chemical substances alone. For instance, the nucleus may consist of uric acid, the outer portion of calcium oxalate, or *vice versa.* [The nucleus may also contain some of the various common forms of micro-organisms.] Often the phosphates form only the material for the outer portion of the

stone. They are easily soluble in acids, and, in contradistinction to oxalate stones, also in organic acids. However, only the precise chemical and microscopical examination of the stones gives exact information as to its composition in the individual case. This is especially the case in rare forms of stone, which are composed of indigo, cystin, and xanthin. As an accessory constituent of renal stones, calcium carbonate is often found.

Confusions with other pathological conditions occur frequently, if we venture to make the diagnosis from isolated symptoms of nephrolithiasis, hæmaturia, renal colic or from the symptoms of pyelitis, without concretions being demonstrable in the urine. It must be established as a rule that the demonstration of the latter is absolutely necessary for a positive diagnosis, and that, if they are absent, the diagnosis is best left in suspense. If this rule is not observed, bad diagnostic errors are liable to occur: Confusions with renal tuberculosis, paranephritic abscesses, lumbago, gastralgia, etc., but even if concretions are passed with the urine, diagnostic errors are still possible, either on account of gall-stones in rare cases forcing a wrong passage into the urinary tracts, being then discharged with the urine, or because it is not a question of renal calculi but of stones of the bladder.

Differential Diagnosis between Renal and Bladder Stones.—It is true that the disturbances and pains in the latter case are concentrated upon the region of the bladder, especially upon that of the neck of the bladder; but radiations of the pain to the renal region occur also. It is important in the decision whether renal or bladder stones are present, that, in stones which have formed primarily in the bladder, the pain which eventually radiates to the renal region, besides the bladder disturbances, is not restricted to *one* side and does not precede the former. The *reaction* of the urine, which usually becomes alkaline in long-lasting cystitis, is of no value, because an alkaline reaction of the urine may also occur in calculous pyelitis. Of course, the most important diagnostic means is the direct examination of the bladder with the bladder sound.

Cystoscopic Examination.—Owing to the possibility of a nephrectomy, the question sometimes confronts us whether formation of stone is to be assumed in *one* kidney only or in both. *This question can never be decided with absolute certainty* without cystoscopic examination; for, although all symptoms in a case may point to the isolated affection of *one* kidney, it is not impossible that concretion may be present in the other also, as *concretions may, exceptionally, develop without any symptoms at all.* The perfection of cystoscopy [and the X-rays] have rendered it feasible to make a positive diagnosis in this respect, and, therefore, operative interference for the removal of a kidney affected with stone should not be undertaken until we are satisfied, from the condition of the urine collected isolatedly from the one ureter [and the other diagnostic aids], regarding the state of health of the one kidney.

ANOMALIES OF FORM AND POSITION OF THE KIDNEYS

Hypertrophy of the Kidneys.—*Partial or general hypertrophy of the kidneys* can be diagnosticated only if the kidneys are movable and if we are able, in the latter case, to palpate through the abdominal walls an organ enlarged *in toto*, with smooth surface, in the former case, a tubercled surface of the kidney. To make a diagnosis it is necessary to observe the patient for a long time to be sure that no neoplasm in the renal tissue simulates such, generally rare, anomaly of the kidney. The history of the case may also be of some help, inasmuch as a preceding embolism may cause partial hypertrophy. Diabetes may give rise to diffuse hypertrophy of both kidneys; occlusion of the ureter of one kidney by stones, etc., may produce diffuse hypertrophy of the other kidney. On the other hand, I have observed unilateral and bilateral hypertrophy of both (movable) kidneys at the bedside in two cases in which nothing whatever could be found ætiologically to explain the occurrence of these anomalies.

Horseshoe Kidney.—The coalescence of both kidneys in some places, so-called *horseshoe kidney*, has also become diagnosticable in some instances, inasmuch as such anomalous kidneys usually also assumed an abnormal position, descended (as far as the true pelvis), and, with thin abdominal walls, could also be felt in front of the vertebral column as a tumour which was caused to pulsate by the adjacent abdominal aorta.

Single Kidney.—Finally, the *congenital absence of one kidney* can be diagnosticated if one renal region shows constantly tympanitic sounds, the other normal dulness, and if, upon repeated examination, no movable kidney is palpable. Attention is called to the presence of this anomaly, either accidentally when percussing the renal region for other reasons, or in cases in which the impaction of a renal stone of one side takes place with pains which are distinctly localized in the latter, and if complete anuria and, eventually, uræmia follow the occlusion of the ureter.[1] The absence of one kidney does not cause any functional disturbances, as the kidney which is present hypertrophies and fully compensates and transacts the secretion of urine.

MOVABLE KIDNEY, REN MOBILIS, WANDERING KIDNEY, FLOATING KIDNEY, NEPHROPTOSIS

Movable Kidney.—Of much greater practical importance to the diagnostician is the acquired change of position of the kidney—*movable kidney* (*Ren mobilis, wandering kidney*)—which anomaly has recently been accorded especial attention by various authors, so that, according to my experience and that of others, it may be considered a relatively frequent occurrence. The diagnosis of floating kidney is easy, almost without exception, provided the examination is done bimanually and with the abdomen relaxed, i. e., if, in palpating or in executing slight massage from the abdominal walls antero-posteriorly, one hand pressed from the lumbar region towards the other hand. We determine then eventually an oval, smooth, firm tumour of the size and shape of a kidney which is usually slightly sensitive to stronger pressure; sometimes it is possible to palpate even the hilum of the kidney and to feel the pulsation of the renal artery which enters here. Floating kidney occurs principally in women, favoured by the changes of

[1] That anuria may also occur in the presence of two kidneys with occlusion of only one ureter has been stated previously in the discussion of the diagnosis of nephrolithiasis (p. 388).

position of the uterus and relaxation of the abdominal walls after pregnancies or possibly also by pressure of the corset. Displacement of the kidney is found much oftener on the right than on the left side; the displaced organ can be moved more or less by the palpating finger *and be felt especially well during deep respiration of the patient.* If the patient is made to assume various postures of the body, the position of the tumour will also change, in the knee-elbow posture especially will it advance towards the anterior abdominal wall, whereas in the dorsal position of the patient it will fall back and can, if very movable, be replaced to its normal location. Percussion of a movable kidney does not furnish a practical serviceable result for the diagnosis; however, it is necessary for diagnosis that the renal region of the respective place, at least upon *repeated* examination, occasionally shows *distinct* clear tympanitic sounds.

Differential Diagnosis.—Confusions with other tumours of the abdominal cavity which are of about the same size and consistence as the kidney occur frequently, especially if only one examination takes place and if both kidneys are not movable (the usual case), but only one of the organs has become displaced from its normal position. *Tumours of the pylorus*, especially, may show an extremely marked movability. I was able, in one of my cases, to displace, with the greatest ease, a carcinoma of the pylorus, which was absolutely smooth to the touch and of the size of the kidney, from the right umbilical region into the left hypochondrium—in fact, slightly below the left costal arch! It was due particularly to the unusual smoothness and movability of the tumour that a carcinoma of the pylorus was considered only in the second or third place; an exploratory incision of the abdominal cavity only proved the actual condition. It would be quite reasonable first to think of a confusion of pyloric tumours with movable kidney, since recently it was believed that a subsequent manifestation of tumours of the pylorus, gastrectasis, could also be attributed to the pressure of a floating kidney. But, in my experience, we are not entitled to ascribe gastrectasis found in movable kidney to the effect of the latter. The following facts, besides the presence of gastrectasis (eventually with an alteration of the hydrochloric-acid secretion of the stomach contents), guard against confusion with neoplasms of the pylorus: permanent bilateral dulness in the renal region, the relations of the tumour to the contours of the stomach filled and emptied by means of the stomach-tube, and, above all, the shape of the tumour itself, which, in floating kidney, is bound always to show on palpation the exact shape of the kidney. This latter fact also applies to the differentiation of *ren mobilis* from *tumour of the gall-bladder*, which, besides, beginning at the border of the liver, cannot be grasped superiorly and which is almost never as movable as wandering kidney.

Slightly more difficult upon a single examination is the differential diagnosis between movable kidney and *movable spleen.* However, percussion of the splenic region, which gives a tympanitic sound in floating spleen, and also the not bean-like shape of the tumour, will soon elucidate an eventual error. It may be especially mentioned that both organs, the left kidney and the spleen, may be movable simultaneously, as I was recently able to determine with certainty during life in a case which, later, came to autopsy. A confusion of movable right kidney with a *tight-lace lobe of the liver* is more liable to occur, as has been explained on p. 205. I have become convinced that a considerable portion of floating kidneys, which are so frequently diagnosticated and which are not controlled by autopsy, are tight-lace lobes of the liver, and that wrong diagnoses are very common in this respect. The best mode to guard against such occurrences is to require for a *positive* diagnosis of movable kidney that *the organ in question shows the exact shape of the kidney, can be fully grasped from above, therefore passes by continuity into the resistance of the liver and is easily and markedly to be displaced downward even without deep respiration of the patient.* It is premised that the tight-lace lobe is movable, can be turned eventually, and is thick and round to the touch (as is the case in circu-

latory disturbances)—i. e., accordingly has no sharp, thin, lower border. If the contrary is the case, a confusion of tight-lace lobe of the liver with floating kidney is *a priori* impossible.

A confusion with other abdominal tumours is not very well possible if we adhere to the above-named characteristics of movable kidney. Besides, a floating kidney often shows symptoms which are especially peculiar to this condition. It is true, the sensations of heaviness, tugging, and slight pains in the abdomen are of too ambiguous a nature to be at once applied to the diagnosis; but the fact alone that all more violent movements of the body—horseback riding, driving, dancing, etc.—markedly aggravate this disturbance, gives a slightly better support to the diagnosis. But it is of especial importance that so-called *symptoms of impaction* occur from time to time: collapse, violent pains, inclination to vomit, sensation of anxiety, chills, and disturbances of urinary secretion, especially scanty diuresis, sometimes with the development of an acute temporary hydronephrosis; symptoms which, in my opinion, are easiest ascribed to a temporary torsion of the ureter, of the renal vessels, and nerves in the pedicle of the kidney, due to more marked movements of a floating kidney. It also appeared to me that transitory inflammations in the neighbourhood of the organ were the source of "symptoms of impaction" in rare cases. Whatever else has been stated to be a consequence of movability of a kidney (for instance, hæmaturia without previous impaction) is, according to my experience, very doubtful as to its connection with floating kidney; only temporary *albuminuria* may, in my experience, be the consequence of circulatory disturbance which is brought about in the dragged—i. e., in part twisted, renal vessels, and which causes an insufficient function of the glomerulus epithelia. A movable kidney rarely becomes the seat of cancer or of concretions, as has been explained in the respective chapters.

DISEASES OF THE URINARY BLADDER

CATARRH OF THE BLADDER—INFLAMMATION OF THE BLADDER—CYSTITIS

Symptoms that may be Utilized Diagnostically.—*Inflammation of the bladder* (*cystitis*) usually causes such pronounced symptoms that the diagnosis does not present any difficulties. The local symptoms, above all, are of importance, whereas the general manifestations, which are sometimes present in this condition, are not to be considered in the diagnosis: Fever, loss of appetite, vomiting, etc. The local symptoms in *acute* cystitis are different from those in the chronic condition, inasmuch as in the former spasm and pains in the bladder region and surroundings, predominate, whereas these symptoms are only indicated in chronic catarrh of the urinary bladder and only a troublesome desire to frequent urination exists. External pressure in the bladder region is more or less felt, especially on bimanual examination from the vagina and rectum respectively. Catheterization also causes disturbances or violent pains, especially if the neck of the bladder is inflamed.

However, a sure support for the diagnosis is found only, apart from cystoscopy, in the condition of the urine and in the microscopical examination of the same. The urine is more or less cloudy, contains mucus and, eventually blood, white blood corpuscles which partly show lively amœboid manifestations of contraction, bladder epithelia, and almost always *bacteria*.

The investigations regarding the ætiology of cystitis which were made

during the last years by various authors (Rovsing, Guyon, Hallé, Reblaub, J. Müller, Schnitzler, Barlow, and others) of which the experimental investigation of Rovsing contributed principally to improve the views we held regarding the pathogenesis of cystitis and thus to shape the diagnosis of the various forms in a more precise manner, have shown that *cystitis is caused almost without exception by microbes.*

Bacteria which cause Cystitis.—The bacteria which are to be considered in this respect are either bacilli (in the first place, bacteria coli commune, tubercle bacilli, coccobacillus ureæ, typhoid bacilli, proteus) or various kinds of cocci (staphylococci, primarily staphylococcus pyogenes aureus, streptococci, diplococcus pyogenes). If pure cultures of these bacteria are introduced, with the necessary precautionary measures, into the bladder of *healthy* animals, sometimes a cystitis develops, at other times it does not, or a cystitis of a more marked degree, when simultaneously, by constriction of the urethra at the neck of the bladder, care is taken that the bacteria are detained in the bladder for some length of time. *Retention of urine*, therefore, is an *auxiliary cause* of the occurrence of cystitis, the same as are *hyperæmia of the vesical mucous membrane* and *traumatic effects upon the bladder.* The significance of these factors in the causation of cystitis was recognised principally by Guyon's school.

Some of the bacteria which cause cystitis are able to decompose urea into ammonium carbonate; others do not have this property, for instance, the tubercle bacillus, the gonococcus, the bacterium coli commune, etc. *There is no question that the ammoniacal decomposition of the urine* which is observed in many, especially the severe, cases of cystitis, *is a product of micro-organisms, and this transformation of urea into ammonium carbonate always takes place without the aid of a ferment developed by them,* a fact which *I* demonstrated years ago in regard to bacteria which were obtained by Graser and *myself* from decomposed urine as well as in regard to the pulmonary sarcines which also decompose urea. The assumption of Rovsing that ammonia is the first cause of inflammation in cystitis was not confirmed. The systematic investigations made in my clinic (J. Müller, Baer) have shown that the reaction of urine in cystitis is, relatively often, acid, and that the importation of bacteria into the bladder may produce cystitis, even without the transformation of urea into ammonium carbonate being a necessary accompaniment. Besides, injections into the bladder of ammonium solutions, the strength of which corresponded to the contents of NH_3 of urine decomposed by ammonium in severe cystitis, were not followed by inflammation of the mucous membrane of the bladder, a result which was also obtained by the experiments of other investigators.

Importation of Bacteria.—The question *how the microbes which produce cystitis usually reach the urinary bladder* is to be decided according to clinical and experimental experiences in such a manner *that, without question, cystitis is in most cases a consequence of a carelessly executed catheterization.* Bacteria which excite cystitis are then introduced into the urinary bladder from an external source, in part also from the urethra, in which pathogenic bacteria are present in large numbers, by means of the catheter (for the latter reason also, sometimes, when it has been thoroughly disinfected). Bacteria may enter the urinary bladder without *preceding catheterization*: If the orifice of the bladder is patulous, in dripping of urine, in high-graded strictures in which the stream of urine, according to the view of Rovsing, when coming from the bladder, rebounds at the place of the stricture, thus washing into the urinary bladder those bacteria which lie centrally in the urethra. Another mode of origin of cystitis is, that an abscess perforates into the neighbourhood of the bladder, or, as has become probable from the investigations of Reymond, that infectious microbes enter, from inflammatory areas in the neighbourhood of the bladder (for instance, from a metritis), through the wall of the bladder, the continuity of which remains intact, and thus produce, at first circumscribed, later diffused cystitis. Another mode for the immigration of bacteria into the bladder is that *they enter from the blood and from the kidney into the bladder* after the kidney

has been put into a condition of inflammation and thus becomes suitable for the passage of bacteria. It is especially the staphylococcus pyogenes aureus, which is able secondarily to cause cystitis in this manner—i. e., by means of a suppurative nephritis—which has been primarily produced by this bacterium.

According to the virulence of the bacteria and according to the conditions being favourable to their increase in the bladder in the given case, mild or severe forms of cystitis develop. The signs of the mild form are: absence or slight development of local and general manifestations (fever, etc.), few leucocytes in the sediment, eventually crystals of ammonium and magnesium phosphate (in coffin-lid form) and (thorn-apple-shaped) crystals of ammonium urate; the chemical examination of the urine often shows acid reaction, mucus, and no albumin, or only traces of the same. In the *severe* form, on the other hand, numerous pus corpuscles are found in the urinary sediment (up to 150,000 in one cubic centimetre of urine) and usually profuse amounts of crystals of salts of ammonium.

The reaction of the urine is in most cases strongly alkaline (ammoniacal) and contains as much albumin as 0.1 per cent and more. As large quantities of mucus and pus are contained in the urine, a thick sediment, which sometimes assumes a jelly-like consistence with alkaline-ammoniacal reaction of the urine, forms upon standing. This can be determined best particularly when the urine is poured from one vessel into another, in which case the adhesive, glue-like, sediment is drawn out in long threads. If gangrenous changes of the vesical mucous membrane develop, the urine, which has a putrefying odour, contains desquamated shreds of the bladder wall. The fever may attain high grades in the severe forms of cystitis, and may be accompanied with chills. The general health may be markedly disturbed, and the patient may succumb with manifestations of poisoning.

Thickening and Distention of the Bladder.—In case of long duration of a severe cystitis, the inflammatory infiltration of the bladder wall gives rise to a thickening of the same, so that the bladder may be felt as a globular, hard tumour from the abdominal walls over the symphysis pubis and from the rectum. This is especially easy if a paralysis of the musculature of the bladder produces an excessive filling of the bladder and its vertex reaches up to the umbilicus and farther. It is possible, when examining the interior of the bladder by means of a metal catheter, to observe the often beam-like, thickened muscles of the bladder wall as such. A distention of the bladder wall may also occur in *acute* catarrh of the bladder —namely, by consecutive spasm of the sphincter, which is associated with violent spasmodic pains radiating to the glans penis, to the testicles, and to the perinæum; whereas the retention of urine in cases of chronic cystitis mostly develops insidiously and painlessly, and the presence of a bladder tumour is usually accidentally discovered upon palpation of the abdomen.

If the symptoms of cystitis are as pronounced as just described, the disease cannot be mistaken. This is different, however, if it is a question of insignificant degrees of catarrh or of the last remnants of a healing cystitis.

Differential Diagnosis between Cystitis of a Mild Degree and Contracted Kidney.—The correct explanation of such cases has often given rise to difficulties, at

least to me. The examination of the urine, even if the microscopical examination of the urine shows only a few pus corpuscles, always gives a slight but distinct albumin reaction under such circumstances. Now the question suggests itself whether these quantities of albumin are caused by cystitis or by chronic nephritis. The question is so much more difficult to decide, as numerous white blood cells occur also in nephritis, and simple hyaline casts are also found in normal urine. However, numerous investigations which I made with the urine of healthy individuals have shown that, unless urinary sediment contains only some isolated hyaline casts, this always represents a pathological symptom—i. e., that the kidneys are inflammatorily affected in such cases. Then it is always necessary to make a careful examination of the heart for hypertrophy, and of the pulse for increased tension in the aortic system, also to examine with the ophthalmoscope for eventual retinal changes, in order to make a positive diagnosis of nephritis. Posner has recently availed himself of *the number of pus corpuscles per cubic centimetre in comparison to the percentile quantity of albumin* to determine in the given case whether the excretion of albumin in the urine should be regarded simply as a consequence of the admixture of pus or not. It was shown that, with 80,000 pus corpuscles to the cubic centimetre, one per mille of albumin is present in the urine; if much more albumin is found than corresponds to this proportion, for instance, one quarter of one per mille in a case in which the counting of the pus corpuscles with the Zeiss apparatus gave only 5,000 per cubic centimetre, another source of the albumin in the urine is to be thought of, especially of nephritis; positive information, however, will only be obtained in most cases by the cystoscopic examination according to Nitze. The diagnosis becomes especially difficult if both conditions, nephritis and cystitis, are present at the same time. A diagnosis in such cases is possible only if the sediment is very profuse and if large quantities of pus cells and pavement epithelia are found besides the epithelial casts.

Differential Diagnosis between Cystitis and Pyelitis.—As the stratified basement epithelium of the mucous membrane of the urinary bladder is entirely like epithelium of the ureter and of the renal pelvis morphologically, a confusion of cystitis with *pyelitis* is also possible in such cases, in fact, it cannot be avoided if the diagnosis is based simply upon the result of the urinary examination. For the reaction of the urine is also of no significance, because alkaline or ammoniacal reactions are quite common in pyelitis, if this affection has arisen in connection with a cystitis, or if it has developed from the kidneys by pyogenic, urea-decomposing microbes. If we mean at least to try to make a diagnosis in such a case, it is necessary to consider the other symptoms of the disease, especially the pains in the renal region, which are absent in catarrh of the bladder which is not complicated by pyelitis; but a diagnosis based upon the seat of the pains is always a doubtful one. Nor is spasm of the bladder pathognomonic of cystitis, because a painful desire to urinate exists also in pyelitis, although not quite as often as in the former disease. It is of greater significance in the diagnosis of pyelitis that real attacks of renal colic may occur in its course, with temporary occlusion of the ureter by mucus and pus, and it is of especial importance that hydronephrosis may develop under such circumstances, with unilateral development of the disease, and that entirely clear urine (originating in the healthy kidney) may be voided temporarily. This is never observed in catarrh of the bladder; it is only a pity that this condition is by no means constant in pyelitis. Nor does the ætiology give much information in regard to the differential diagnosis between these two affections, as the same causes of origin generally hold good for both.

Ætiological Diagnosis of Catarrh of the Bladder.—Nevertheless, it is necessary always to consider the *ætiological factors* in every case of cystitis, and not to regard the diagnosis as complete until the cause of the catarrh of the bladder has become clear in every instance. We must investigate in this respect whether chemical irritants (food, medicines, etc.), thermic effects or traumatism were active, or whether an inflammation has spread

to the bladder from neighbouring organs, or, finally, whether an infectious virus gave rise to the catarrh of the bladder.

Gonorrhœal Cystitis.—Especial discussion is required in this respect by "*gonorrhœal*" *cystitis* and also by that form which is caused by stones of the bladder. As to catarrh of the bladder occurring *in the course of gonorrhœa,* there can be no question, according to Krogius, Barlow, and others, of the occurrence of genuine gonococcic cystitides—i. e., of catarrhs of the bladder which are brought about by the action of gonococci which have entered the bladder—but, on the other hand, the well-known investigations of Bumm have proved that gonococcic cystitides are, *unquestionably, only very rarely of a specific nature*—i. e., caused by gonococci.

These latter possess the peculiarity of entering only into mucous membranes which are covered with cylindrical epithelium. If diplococci are found in the urinary sediment in cystitis of patients with gonorrhœa, this, of course, is no proof that the cystitis was caused by gonococci, because they may also come from the urethra affected by gonorrhœa, and may, especially in the later stages of gonorrhœa, be excreted, with the so-called gonorrhœa threads ["Tripperfäden"]. But even if this source of the admixture of diplococci to the urine can be excluded, caution should be used in drawing any conclusions, as the pus of gonorrhœa contains profuse quantities of pyogenic microbes, staphylococci and diplococci, which are able to cause cystitis. The clinical experience that the administration of balsam copaiba caused a rapid cure of gonorrhœal cystitis, a fact which I had often a chance to determine, is no direct proof of the specific character of cystitis, as this balsam not only acts favourably in gonorrhœa, but also in such catarrhs of the bladder as are surely not of gonorrhœal origin.

Calculous Cystitis.—Catarrhs of the bladder which are produced by *stones in the bladder* are distinguished from the usual cystitides by the more frequent complication with hæmaturia, by the frequent presence of gravel and crystals in the urine, and by the *pains which, in spite of the chronic course of the disease, are violent* and which are produced especially by vigorous movements of the body, radiating usually into the urethra as far as the point of the glans penis, but also farther into the neighbourhood of the bladder. Particularly towards the end of urination, stranguria manifests itself as a sign of irritation of the neck of the bladder by the stones; sometimes the stream of urine is suddenly interrupted, rarely is the flow entirely prevented. The distinct sensation of the patient, in some cases, of a moving foreign body in the bladder may sometimes give rise to the thought of the presence of a stone, but it is of as little use for a *positive* diagnosis as all the above-named symptoms. *The diagnosis becomes positive only by the result of cystoscopy and by the examination of the bladder by means of the sound.* The latter (after previous disinfection of the urethra) is to be done repeatedly if the result is negative at first, because stones which become impacted in a diverticulum of the bladder or at unusual places (especially above the symphysis) are often discovered only upon repeated examination with the sound. As to the remainder I must refer to what has been stated in the discussion of renal calculi.

Croupous Diphtheritic Cystitis.—In rare cases it may happen that, owing to the action of an infectious virus in the course of scarlatina, small-pox, etc., or to a very intense irritation by chemical substances (for instance, cantharides or putre-

urine), or, finally, by catheterization and importation of micro-organisms into the bladder (according to an observation of Bumm, of the yellowish-white diplococcus), very severe forms of inflammation may occur, viz., *croupous diphtheritic cystitis.* Chills and high fever may accompany this form of cystitis, and the diagnosis will become possible by the passage of white shreds in the urine, which consist of fibrinous threads, pus cells, epithelia, and bacteria.

Submucous Parenchymatous Cystitis.—If the inflammation is not restricted to the mucous membrane of the bladder, but located in the bladder wall, the latter is thickened, its contraction is impeded, and the secretion of urine is impaired and very painful. According to the seat of the pus collection in the wall of the bladder occlusion of the ureters or of the internal orifice of the urethra may be brought about, thus causing accumulation of urine upward towards the renal pelvis, and, eventually, temporarily a complete interruption of the discharge of urine. At the same time there exist chills, high fever, pressure and pain in the region of the bladder, and the signs of suppuration in the depth of the pelvis (*cystitis submucosa "parenchymatosa"*). After perforation of pus into the bladder, rapid improvement of the symptoms occurs, with passage of the pus with the urine. The manifestations become modified correspondingly upon perforation into another direction (towards the peritonæum, rectum, vagina, etc.). If these suppurative areas communicate with the interior of the bladder, the urine mixes with the pus and decomposes; the results of this infiltration of urine are: an increase of the inflammation in the neighbourhood of the bladder, œdematous swelling of the perineal and anal regions, etc.

Paracystitis.—*Paracystitis* is accompanied with similar symptoms—i. e., the signs of suppuration in the depth of the pelvis, and its consequences. Only catheterization is less difficult in this case, and the purulent infiltration of the neighbourhood of the bladder is felt from the abdominal walls, perinæum, rectum, or vagina as a firm, later fluctuating tumour, the further development of which may cause hypostatic abscesses, perforations into the rectum, bladder, etc.

TUBERCULOSIS OF THE BLADDER

The diagnosis of tuberculosis of the bladder, which occurs almost exclusively as a partial manifestation of tuberculosis of the renal pelvis and genitalia, coincides with the diagnosis of renal tuberculosis. The urine contains, the same as in the latter condition, *blood*, epithelia, pus, and detritus, sometimes also *elastic fibres* and *shreds of connective tissue*, if the disease has led to ulcerative disintegration of the bladder wall, especially at the neck of the bladder. But the most important constituents of the urinary sediment, which determine the diagnosis, are *tubercle bacilli*, as has been explained in the discussion of the diagnosis of nephrophthisis. But, as found by Rovsing, the demonstration of tubercle bacilli succeeds only if the reaction of the urine is not ammoniacal at the same time, evidently because tubercle bacilli in ammoniacal urine lose the property to stain with the usual methods of tinction. But a regular staining can be obtained if, by the internal administration of boric acid (three times daily 0.5), the reaction of the urine is changed into an acid one. The diagnosis becomes considerably more certain, as previously stated, if a simultaneous hardening and cheesy degeneration of the testicles, epididymis, or ovaries, or a secondary affection of the lungs can be demonstrated.

Diagnosis of the Special Participation of the Bladder in the Tuberculous Process of Inflammation of the Urinary Passages.—It is usually only a question in the given case whether the participation of the bladder in the process can be diagnosticated as such. This, however, will not always be possible. But if (contrary to the usual cases of tuberculosis of the kidneys) a urine is *constantly* voided which leaves much sediment and contains tubercle bacilli; if marked strangury exists, and if the catheter, upon entering the bladder, causes pain at a certain locality and produces hæmorrhages, we may diagnosticate a participation of the bladder in the urogenital tuberculosis. If the surface of the ulcer is encrusted with mineral deposits, we may be misled, so long as no tubercle bacilli are demonstrated in the sediment, to assume that stones of the bladder are the cause of the catarrh.

The *occurrence of tuberculous cystitis* has recently been explained by Rovsing. Injection of pure cultures of the tubercle bacillus into the healthy bladder of rabbits does not exert a pathogenic action upon the latter, not even if a retention of urine, lasting twenty-four to twenty-six hours, caused by ligature of the urethra, is made to follow the injection. *Furthermore, a decomposition of urea by the tubercle bacillus never occurs,* because, although pyogenic, it does not decompose urea. The *reaction of the urine* in uncomplicated tuberculous cystitis, therefore, remains *acid* under all circumstances. If the experimental production of tuberculous cystitis is to succeed, a mechanical injury of the mucous membrane of the bladder must take place and direct inoculation of the bacilli into the tissue of the vesical mucous membrane, or the bacilli must be injected into a bladder affected with suppurative cystitis, and a twenty-four-hour-retention of urine follow the injection.

The *usual mode of origin of tuberculous cystitis in man is probably that the tuberculous process spreads by contiguity into the tissue of the bladder*, or that the tubercle bacilli reach the parenchyma and mucous membrane of the bladder *metastatically*—i. e., from the blood current—advance to the free surface of the same, and at this place cause processes of ulceration. If the urine is ammoniacal in tubercular cystitis, it appears that a mixed infection is always present—i. e., there is no question that in such a case microbes which decompose urea have become active besides the tubercle bacilli.

NEOPLASMS OF THE BLADDER—CANCER OF THE BLADDER

Some of the neoplasms which occur in the bladder, such as myoma, myxoma, sarcoma, etc., are more of a pathologico-anatomical than of a clinico-diagnostic interest. But *papillomatous fibroma* ("villous cancer") and also *carcinomata*, which usually spread secondarily into the bladder from the neighbourhood, are objects of the clinical diagnosis. The usual symptom of carcinoma of the bladder is *hæmaturia;* of course, an ambiguous manifestation which occurs in various affections of the urinary tracts. Nor is the pain of much value in the diagnosis; but it is suspicious, to a certain extent, if pain and hæmorrhage do not take place after movements, as in bladder-stone diseases, but appear without any external cause. However, a diagnosis can be made only when (besides the pain in the bladder region and surroundings, besides the hæmaturia and the symptoms of chronic cystitis in general) a tumour is felt from the rectum or from the vagina, or by means of the catheter. If, as occurs in rare cases in women, a pedunculated papilloma advances into the urethra, the tumourous mass may become visible. It occurs relatively often that cancerous masses become desquamated and are discharged with the urine, which then usually is in a putrefying condition. The desquamated carcinomatous particles can be recognised as such by a microscopical examination, unless they are in a condition of complete necrosis.

If, nevertheless, a diagnosis cannot be made, we may, definitely to determine the same, make an *endoscopic examination*. There can be no doubt that swelling of the inguinal glands and increasing cachexia also are in favour of the presence of carcinoma of the bladder; but only a very limited diagnostic importance is to be granted to the occurrence of cachexia, because long-lasting affections of the bladder, which are accompanied with hæmaturia, are, of course, also followed by cachexia. This is also true of hæmorrhoids of the bladder, which will be discussed presently.

HÆMORRHOIDS OF THE BLADDER

All affections of the bladder which we have enumerated so far may occasionally lead to hæmorrhages and hæmaturia; this is most frequently the case with stone and carcinoma of the bladder. Besides these hæmaturias, which are caused by general affections, scurvy, etc., there occur isolated cases of vesical hæmorrhage which can be ascribed only to *varicose dilatation of the bladder veins*. The diagnosis of such "hæmorrhoids of the bladder," which has often given rise to great mischief, should only be made if the possibility of an existence of other kinds of bladder hæmorrhages can be *excluded* with sufficient certainty, as otherwise wrong diagnoses are unavoidable. But, on the other hand, there are certainly cases (I have recently observed one of this category) in which death was precipitated in elderly individuals in consequence of simple varices of the bladder—i. e., of the hæmorrhages caused by the same. It is necessary to know that such cases occur, in order to avoid the mistake, under such circumstances, to assume carcinoma of the bladder as certain in case of a fatal termination. This should only be done generally when *positive* points of support are present favouring the diagnosis of carcinoma (tumour, carcinomatous particles in the urine, see Cancer of the Bladder).

NERVOUS DISTURBANCES OF THE FUNCTION OF THE BLADDER

Physiological Preliminary Remarks.—The distention of the bladder causes a reflex irritation of the muscles of the urinary bladder, both of the smooth muscle fibres which are designated as *detrusor* vesicæ and which run vertically, as also of the transversely striped *sphincter* of the bladder and urethra. In such cases the reflex contraction of the latter predominates over the effect of the detrusor with moderate tension of the bladder. If the filling of the bladder increases, the sphincter can only succeed in retaining the urine in that it becomes voluntarily and very energetically contracted. This is accomplished by the pudendal nerve from the sacral plexus. The nerve fibres for the voluntary contraction of the urethral sphincter, which are stimulated from the brain, pass through the pedunculus through the anterior columns and the posterior portions of the lateral columns of the spinal marrow. The same as the reflex contraction of the urethral sphincter can be voluntarily increased by these fibres, so also is a *voluntary inhibition* possible of those reflex contractions by special fibres of inhibition, the voluntary stimulation of which causes a relaxation of the urethral sphincter. The tract of these reflex inhibitory fibres is also situated in the pedunculi and anterior columns.

Severing of the spinal cord above the lumbar enlargement causes *retention of urine* with distention of the bladder, owing to the removal of inhibition of the reflex contraction of the sphincter. Only after the distention of the urinary bladder becomes very marked, so that the posterior urethral opening also becomes mechanically dilated, excretion of urine occurs *with a full bladder*. Incontinence is also caused by *a severing of the sensory and motor-nerve fibres of the urethra* which, passing into the lower sacral nerves, transmit the reflex of the sphincter and the voluntary contraction of the urethral sphincter. As the sensory nerves of the urethra and of the bladder which produce the sensation of a filled bladder and, with it, that of the *desire to urinate*, ascend in the spinal marrow to the brain (because they probably pass into Goll's columns soon after their entrance into the spinal marrow), the severing of the spinal cord, and especially the degeneration of Goll's columns, abolish the sensation of the desire to urinate.

If these conditions concerning the innervation of the bladder are properly considered, it is easy to explain the alterations in the function of the bladder depending upon nervous disturbances and, with it, to make a diagnosis of conditions of paralysis and spasm of the musculature of the bladder in detail.

PARALYSIS OF THE MUSCULATURE OF THE URINARY BLADDER—"PARALYSIS OF THE BLADDER"—CYSTOPLEGIA

PARALYSIS OF THE DETRUSOR

The consequence of paralysis of the detrusor is the inability of voiding urine normally. A *moderate filling* of the bladder causes a reflex irritation of the sphincter—*retention of urine.* If the tension of the bladder and, with it, *the desire to urinate* become more intense, the reflex contraction of the sphincter will be removed by voluntary stimulation of the reflex inhibitory fibres of the sphincter, and *the discharge will take place by means of the paralyzed detrusor* through *straining of the abdominal muscles,* which takes place with great exertion in a deficient stream (and with a feeling of aggravation) and furnishing unsatisfactory results, so that, upon the *introduction of the catheter, large quantities of urine are found in the urinary bladder after urination.*

Example: Compression myelitis. Patient feels the filling of the bladder, has the sensation of the desire to urinate, is able to discharge urine, but not with the necessary force and only by means of straining of the abdominal muscles; no dripping of urine. A moderately large amount of urine remains in the bladder after discharge, as is proved by catheterization after the discharge of urine, which was as complete as possible.

Variety of Detrusor Paralysis.—Paralysis of the Sensory Tracts.—It is presupposed in such cases that only the motor portion of the reflex tract of the detrusor is paralyzed; if its centripetal portion is also unable to transmit or if *only* the sensory tracts are interrupted, the picture of disturbance of the discharge of urine is slightly different: *Absence of the desire to urinate, excessive accumulation of urine in the bladder, dilatation of the latter to the umbilicus and above,* with reflex contraction of the sphincter. If the latter relaxes finally under the growing tension of the bladder, *dripping of urine sets in with an excessively filled bladder* (overflow of the full bladder, "ischuria paradoxa"). The patients *may be able temporarily to suppress the dripping of urine* by voluntary contraction of the sphincter and, in the interval, to force the passing of moderately large quantities of urine by means of a straining of the abdominal muscles.

PARALYSIS OF THE SPHINCTER

Paralysis of the Reflex Activity of the Sphincter, with Paralysis of the Voluntary Innervation of the Sphincter.—Normally the elasticity of the surroundings of the urethra is sufficient, with moderate filling of the bladder, to retain the urine in the bladder; with a more pronounced distention the reflex activity of the sphincter is brought into play. *But if the reflex of the sphincter is paralyzed, involuntary discharge of urine occurs with moderate desire to urinate, especially during sleep, or if the attention is diverted*—i. e., when the urine is not retained by energetic, voluntary contraction of the sphincter.

If the voluntary innervation of the sphincter is also disturbed, a retention of urine is no longer possible, even with full attention of the patient and while he is awake—i. e., *involuntary discharge of urine occurs as soon as the bladder is more markedly filled.*

In *paresis of the sphincter* the patient is urged to discharge urine upon *moderate* desire to micturate and moderate filling of the bladder, because the weak sphincter is only able to offer slight resistance of short duration to the activity of the detrusor, respectively to the greater tension of the bladder. Neither does the weak sphincter hold out upon coughing and other more marked movements of straining—i. e., involuntarily a stream of urine is discharged by starts (especially in women).

SYNCHRONOUS PARALYSIS OF THE DETRUSOR AND THE VESICAL SPHINCTER

Combined Paralysis.—If the detrusor and sphincter are paralyzed simultaneously, *the bladder fills to a moderate degree* so long as the elasticity of the surroundings of the urethra are able to retain the urine. Now, owing to the inability of the sphincter to interfere, *involuntary discharge of urine occurs with moderately filled bladder, showing itself as dripping of urine owing to paralysis of the detrusor.* Temporary suppression of the same is not possible, in contrast to simple paralysis of the detrusor. At the same time, it may be possible that the sensation of a beginning filling of the bladder may be preserved so long as the sensory tracts are capable of conduction.

SPASM OF THE BLADDER MUSCULATURE—CYSTOSPASM

HYPERKINESIS OF THE VESICAL DETRUSOR

Spasm of the Detrusor.—Suppose the excessive irritability does not affect the sensory portion but the centre, respectively the motor, portion of the reflex arc of the detrusor, upon increasing dilatation of the bladder, not the usual, but a *spasmodic contraction of the detrusor* will occur. The consequence is that the patient has an urgent desire to urinate, which even voluntary contraction of the sphincter cannot in the long run resist. *The patients, therefore, are urged to urinate with violent pressure and with moderate filling of the bladder.*

HYPERKINESIS OF THE SPHINCTER VESICÆ

Spasm of the Sphincter.—A *spasmodic contraction of the sphincter occurs, with moderate filling of the bladder,* under the same presumptions as in hyperkinesis of the detrusor, instead of the normal reflex contraction of the sphincter. The patient endeavours to remove the spasmodic contraction by voluntary stimulation of the reflex inhibitory fibres of the sphincter, *but he is only partially or not at all successful.*

Dysuria and Ischuria Spastica.—In the former case he is still able to pass small quantities of urine (*dysuria spastica*), in the latter case he will

not succeed in doing so, in spite of the greatest efforts on the part of the abdominal muscles (*ischuria spastica*). The bladder fills constantly, with an increasing desire to urinate, the excessive distention is felt in a most disagreeable manner, simultaneously with inability to discharge urine; this, finally, becomes associated with a spasm of the bulbo-cavernous muscle and of the sphincter ani. If the spasm ceases temporarily, small quantities of urine are discharged in a stream, with a continual repetition of the above-described scene. If it ceases permanently, a large quantity of accumulated urine is voided at one time. *When attempting to catheterize the bladder, the catheter meets an obstacle which it is very difficult to overcome in the upper portion of the urethra.*

SIMULTANEOUS SPASM OF THE DETRUSOR AND SPHINCTER VESICÆ

Slight Development of Combined Spasm of Detrusor and Sphincter.—Upon *slight development of the spasm of both muscles,* the patient has a constant desire to urinate, owing to the (although weak) spasm of the detrusor; he is urged but, upon attempting to discharge urine, obstacles manifest themselves to the action of the reflex inhibitory fibres of the sphincters. It is difficult, therefore, for the patient to discharge the urine.

Highest Degree of Intensity.—An *increase of the spasm* will lead to *spastic enuresis,* to spasmodic dripping of urine, until, upon still more pronounced intensity of the spasm, the discharge of urine ceases entirely and the vain struggle of the vesical musculature to discharge the bladder contents attains the highest degree of intensity. At this stage it is possible that dangerous general symptoms may follow: collapse, weakness of the pulse, cold sweat, trembling, and general spasms.

The above-described spasm of the muscle fibres of the detrusor and sphincter muscles may arise as a combination of both spasms in the same manner as each of these is brought about as an isolated spasm—i. e., by synchronous, excessive irritability of both reflex centres and of the motor part of both reflex arcs.

Hyperæsthesia of the Vesical Mucous Membrane as Cause of Spasm of Detrusor and Sphincter.—But the cause is usually to be looked for in a *hyperæsthesia of the vesical mucous membrane. In consequence of this condition, even a slight filling of the bladder will be followed, instead of by the normal reflex contraction of the detrusor and sphincter muscles, by a reflex spasm of both,* in which, according to normal conditions, the spasm of the sphincter predominates, and now the above-described picture results. *The desire to urinate will be felt very painfully in this condition from the very beginning, owing to the excessive irritation of the sensory nerves.*

SENSORY DISTURBANCES OF THE MUCOUS MEMBRANE OF THE URINARY BLADDER

The diagnosis of *hyperæsthesia* of the vesical mucous membrane usually coincides with that of the above-described spasm of the sphincter and detrusor muscles. However, there are apparently cases in which, owing to

hyperæsthesia, the accumulation even of the *smallest* amounts of urine is felt as being very troublesome, and, before a reflex spasm occurs, urine is discharged voluntarily, respectively the reflex contraction is so little spasmodic as yet that the contraction of the sphincter muscle is spontaneously removed in this stage by the inhibitory fibres. The patient, therefore, experiences almost a continuous, disagreeable desire to urinate, but if only he yields sufficiently often to the desire to urinate, he discharges the urine without difficulty and without a pronounced sensation of spasm.

Anæsthesia.—*Anæsthesia of the bladder* manifests itself by *absence of the desire to urinate*; at the same time a suspension occurs of the reflex contractions, both of the detrusor and of the sphincter muscles. Now the previously described picture of simultaneous paralysis of sphincter and detrusor muscles develops, but, in contradistinction to that condition, *every sensation of filling of the bladder has ceased in this case.*

It would be erroneous to assume that we shall succeed in every instance in making a diagnosis of the individual form of nervous disturbance of the bladder without objection. But I hope that, with the aid of the above statements, it will be possible, at least in the majority of cases, to be guided in the, often quite complicated, diagnostic situation.

DIAGNOSIS OF DISEASES OF ADRENAL BODIES

ADDISON'S DISEASE

The diseases of the adrenal bodies are not as yet a subject of diagnosis —at most they are the subject of a provisional diagnosis.

The clinical study of affections of the adrenal bodies and, at the same time, the physiological investigation of the function of these bodies were occasioned, in 1855, by the description by Addison of a peculiar disease which terminated fatally. The discoverer of the disease, which has since been named after him, brought it into relation with changes in the adrenal bodies, and stated as characteristic manifestations of the same: anæmia, with adynamia and apathy, gastro-intestinal and nervous disturbances, bronzing of the skin, and progressive cachexia.

Addison's disease has since been observed repeatedly. Although regarding the various symptoms valuable details were discovered during the last forty years, nothing has been changed in the outlines of the pathological picture. We find in Addison's disease tuberculosis in most cases, much rarer other changes of the adrenal bodies. In some cases, however, in which the typical symptom-complex of Addison's disease was observed *intra vitam*, no affection of the adrenal bodies could be demonstrated post mortem. The affection usually begins with marked *sensation of fatigue, adynamia* and *apathy*. To this are superadded, as a second cardinal symptom, *disturbances of the digestive organs*: Dyspepsia, especially vomiting, irregular stools—constipation, later diarrhœa—and *pains in the epigastrium and in the sacral region*. The third manifestation which, in objectivity, is prominent over the other symptoms, is the *pigmentation of the skin and mucous membranes*. As to the abnormal discoloration of the skin, it is concentrated upon those portions of the skin which are most exposed to the light, and to those which are physiologically more intensely pigmented (nipples, axillary folds, genitalia). Of the mucous membranes that of the mouth (lips, palate, tongue) is particularly affected, and the darkening is here rarely diffuse, but rather regularly pronounced in the shape of brown to black areas and stripes. The manner of pigmentation is not absolutely pathognomonic in Addison's disease, as similar pigmentations of the skin occur in pulmonary phthisis and cancer cachexia, in certain forms of diabetes (diabète "bronzé"), in genital affections, etc., and even pigmentations of mucous membranes have sometimes been observed (even without Addison's disease) in healthy individuals and in other patients. Outside of the three cardinal symptoms, there may super-

vene various pathological manifestations of the nervous system: psychical uneasiness, headache, delirium, etc., and progressive cachexia. The action of the heart is almost always weak and the pulse very small.

Physiology of the Adrenal Bodies.—If we may reflect at all upon connecting the above-described pathological symptoms with functional disturbances of the adrenal body, it is necessary, in each instance, that the results of physiological investigations regarding the adrenals be in accord with the clinical facts. It is established physiologically that the adrenal bodies are vital organs, the extirpation of which will, sooner or later, lead to the death of the animal; furthermore, that the administration of adrenal extract causes a considerable *increase of blood pressure*, which, according to the results of experiments, is best ascribed to a specific effect of the suprarenals upon the heart and vessels, especially to the preservation of a permanent tonic tension of the vascular walls which are under the action of the sympatheticus. *Vice versa, extirpation of the adrenal bodies* gives rise to a decrease of blood pressure, to the rapid occurrence of symptoms of fatigue, to emaciation, loss of appetite and to diarrhœa, and also, but very inconstantly or not at all, abnormal pigmentation of the skin and mucous membranes. Finally, physiological experiments have rendered it probable that the adrenal body is a gland with an "internal secretion," its task being possibly, besides the above-named specific action upon the vascular system, to bring about an adjustment of cell nutrition in general, and especially a disinfection of the body in such a manner that the toxic products of metabolism of other organs, in particular those substances which are produced by muscular and nervous activity, are neutralized or destroyed by the activity of the subrenal bodies.

Pathogenesis of Addison's Disease.—In spite of the efforts to explain the function of the adrenal bodies physiologically, our knowledge regarding them is, unquestionably, still deficient. If we assume that Addison's disease is based upon an affection of the adrenal bodies, then, according to what we have referred to in regard to symptoms occurring after extirpation of these bodies, the clinical picture of the affection should undoubtedly show adynamia, disturbances of the digestive organs, cachexia, small pulse, and nervous manifestations. It is different with pigmentation of the skin which, as we have noted, is not, or, at most, very incompletely, developed by the experimental removal of the adrenal bodies, whereas it forms the most prominent pathological manifestation in the picture of Addison's disease. Neusser, in his excellent monograph, has lately endeavoured to explain the pigmentation of the skin in this affection in such a manner that he ascribes the cause of this symptom to a disturbance of innervation in the sphere of the *sympathetic nerve*. He supports this hypothesis with good reasons, especially with facts which are taken from physiology and pathology (pigmentation in syringomyelia, neuritis, etc.). The adrenal bodies, according to his assumption, are not concerned directly in the formation of the pigment, and pigmentation occurs so often in Addison's disease or affections of the adrenals simply because the pathological processes spread to the sympathetic nerve so very usually, and because, according to Neusser, *intimate relations exist in general between the sympathetic nerve and the adrenal bodies*. Neusser tries to make it plausible that the adrenal bodies are inserted in the neuron system of the sympathetic nerve, and that the splanchnic nerve, in its connection with the solar plexus and the suprarenal plexus, acts as a secretory and trophic nerve of the adrenal body. As, furthermore, the centres of the splanchnic nerves are undoubtedly to be found in the cervical and thoracic portions of the spinal marrow, *Addison's disease should be considered as an affection of the splanchnic nerve system in general and that this affection might be caused by changes in the spinal cord, in the splanchnic nerve itself, in the cœliac ganglion, or in the terminal organ in the adrenal bodies.*

There is no question in my mind that this hypothesis of Neusser as to the character of Addison's disease rests upon a sound physiological basis, is decidedly the best at present, and is able to elucidate many dark points in the picture of this affection. But, in my opinion, it does not explain everything, although I do not

deny that Neusser tries, with great acumen, to reconcile the clinical facts and his theory, which apparently do not agree. There can be no doubt that cases exist in which the autopsy revealed total degeneration of the adrenal body, and yet, in which Addison's symptoms were in part or entirely absent, and, *vice versa*, cases of Addison's disease which showed post mortem only partial or no changes of the adrenal bodies. The latter cases could be easily explained, according to Neusser's theory, if a pathological affection can be demonstrated in another part of the splanchnic system from the spinal marrow to the terminal organ. The pigmentation of the skin is not considered at all in this connection, as it cannot at all be directly connected with the adrenal bodies. Therefore, it should not be made the starting-point in the diagnosis of affections of adrenal bodies, although it is probably connected, as a rule, with such an affection, owing to the simultaneous injury to the sympathetic.

The diagnosis of Addison's disease as a group of related pathological symptoms does not present great difficulties if they are well pronounced. But if only some of the symptoms are present, thus, with entire absence of pigmentation of the skin and mucous membranes, adynamia, gastro-intestinal disturbances, with pains in the epigastrium and sacrum, and progressive cachexia, the diagnosis is more than doubtful. If we think we are justified, in view of the completeness of the symptoms, to make the diagnosis of Addison's disease, we may, furthermore, think of the *possibility* of an affection of the adrenal bodies, because, according to statistics, such an affection has been found in 80 per cent of cases of typical Addison's disease. But, to avoid numerous disappointments, it is best absolutely to forego any "diagnosis" of affections of the adrenal bodies.

DIAGNOSIS OF DISEASES OF THE PERITONÆUM

PERITONITIS

ACUTE INFLAMMATION OF THE PERITONÆUM, ACUTE PERITONITIS, PNEUMO-PERITONITIS

The different aspects of the picture of acute peritonitis: *Intense painfulness of the abdomen, which is continuously and spontaneously pronounced, but especially so upon every movement and every touch, pain during urination,* especially towards the end of the act (when the contractions of the bladder cause a tugging of the peritonæum), *sparse quantity of urine, difficulty in voiding the same* (with secondary paralysis of the detrusor), *distention of the abdomen, vomiting, singultus, constipation, which is usually present, fever, small frequent pulse, accelerated respiration and, above all, the entire appearance of the patient, which betrays distinct collapse and anxiety,* this entire picture is so pronounced that the experienced physician is usually able at the first glance to assume the presence of peritonitis.

Results of Physical Examination.—This presumption is confirmed by the results of physical examination, in particular by the determination of an inflammatory *exudate.* Whereas a tympanitic sound can be demonstrated at the highest points of the abdomen in the neighbourhood of the umbilicus, the more dependent parts show a dull *percussion sound* owing to descent of the fluid. A change of posture, which, however, should not be made without necessity, causes a displacement of the fluid exudate and, with it, of the boundaries of the dulness. *Palpation* will give a feeling of fluctuation, with large quantities of fluid and relaxed abdominal walls; a palpable friction rub will, in rare cases, be noted in such areas in which no exudate is present.

The latter is noted especially in the hepatic region, if the parietal layer of peritonæum, which has become inflammatorily coarse, and the visceral layer of the liver rub against each other on respiration. Friction may also be heard and felt in the splenic region, whereas friction does not occur over the inflamed intestinal coils. Only if, exceptionally, stormy peristalsis takes place in the latter, a distinct friction rub can be felt, and heard with the stethoscope, even in the lower portions of the abdomen, as is clearly proved by a case of my own observation, which will be reported in the discussion of carcinoma of the peritonæum.

Absolute certainty as to the presence of an inflammatory peritoneal exudate is obtained by an *exploratory puncture,* executed with the neces-

sary precaution, which produces a sero-purulent, rarely bloody, and, still more rarely (even without the presence of open communications between the lumen of the intestine and the peritoneal cavity), a fluid with a fæcal odour. This also decides the question whether the affection is a sero-fibrinous or a purulent peritonitis, and which micro-organisms have caused the disease in the given case.

Bacteriological investigations in the last decade have demonstrated in this respect, that the most frequent and the most important generators of peritonitides are: the *bacterium coli commune* and the *streptococcus pyogenes. The latter causes especially puerperal and traumatic peritonitides, the bacterium coli commune those which originate in the intestine.* This, however, is true only in a general way, as, for instance, other bacteria, the pneumococcus, etc., were found in the last-named form of the disease. The *staphylococcus pyogenes and the typhoid bacillus* were determined only exceptionally in the peritoneal exudate. The above-named varieties of microbes occur in some of the cases as the sole generators of a peritonitis, whereas, in probably the greater portion of cases, the peritoneal exudate contains several varieties of bacteria at the same time, and so-called "mixed infections" are present.

As to the manner in which the bacteria enter the peritoneal cavity, it is obvious that a traumatism may externally, or perforation of an abdominal organ or abscess containing bacteria may internally open the way for the invasion of bacteria. But bacteria may enter the peritoneal cavity from the intestine even *without* perforation, when the resistance of the intestinal wall has become impaired for some reason or other, for instance, in strangulated hernia, invaginations, or intestinal ulcers. It may be possible, also, but only in very rare cases, that the bacteria reach the peritonæum through the blood current, as is especially the case in septicæmic processes.

If bacteria, which have entered the peritonæum in some manner, are to cause peritonitis, this depends upon the fulfilment of certain favourable conditions, to conclude from recent experimental experiences. In the first place, the *number* of bacteria which at one time enter the peritonæum is of importance, because *few* bacteria, owing to the great resorbability peculiar to the peritonæum, are quickly removed before they can display their injurious effect. Still more important is the fact that bacteria do not develop profusely and cause suppuration in the peritonæum until the latter has become injured and thus unable to absorb. This is caused by chemical irritants, especially also by bacterial toxines, which at first cause a sero-fibrinous, eventually a hæmorrhagic chemical peritonitis. Thus a favourable soil is created for the development and generation of pyogenic bacteria which have reached the peritoneal cavity (see also the theory which I have maintained for years as to the origin of endocarditis, p. 7).

A *permanent high position of the diaphragm* is caused by the dilatation of the intestines and by the accumulation of larger quantities of exudates in the peritonæum, as well as by the violent pains which are produced by every movement of the diaphragm. Such a high position of the diaphragm is easily recognised by the fact that the anterior lower boundary of the lung is high up at the fourth or fifth rib and only slight inspiratory excursions are noted; at the same time respiration becomes frequent, superficial, and costal, and the position of the heart appears to be displaced outward and upward. As the activity of the heart becomes impaired under

the influence of peritonitis, as previously stated, there occur blood stasis, *cyanosis* and decreased secretion of urine. The latter may contain *albumin*, and usually shows a great increase of *indican*, especially marked and constant in acute, diffuse, purulent peritonitis, probably due to deficient peristalsis and more marked decomposition of the intestinal contents.

Deviations from the Usual Picture and Differentio-Diagnostic Absence of Pain.—The diagnosis of the disease does not offer any difficulties if the above-named more or less typical manifestations of the disease are present. However, the various cases of peritonitis show quite remarkable deviations from the above picture, which, however, still holds good in the majority of cases. It may be possible, for instance, that the *pain*, which is always very pronounced, and which is generally considered the most important symptom of peritonitis, may be *entirely absent* in rare cases, even in the diffuse forms of the disease. I have observed this in several cases which later came to autopsy. In one case of acute, diffuse, purulent peritonitis taxis of a hernia even was performed on the last day of the disease without any perception of pain on the part of the patient! On the other hand, we must be careful not at once to conclude from an excessive painfulness of the abdomen upon the existence of a peritonitis, as in attacks of colics which affect nervous or hysterical persons, such a marked *hyperæsthesia of the abdominal walls* may be present that the slightest pressure upon the abdomen causes the most violent pain. Apart from the demonstration that the abdominal wall as such is sensitive to pressure in such cases, fever is absent, usually also vomiting and painful urination, and, above all, collapse with its symptoms as well. But all the above-named manifestations may, exceptionally, be present in attacks of colic in hysterical women or nervous men; then grave errors are avoided only by the simultaneously present, full pathological picture of hysteria: globus hystericus, spasms and the fact that deep pressure upon the abdomen is not felt more than a superficial one, etc. The absence of lateral dulness in these pathological conditions, which are not rare, in my experience, is no proof against the existence of peritonitis, as sufficient fluid accumulates in the lower portions of the abdominal cavity only upon more marked exudation, so that a dulness becomes demonstrable there.

Colics, Gastralgias.—The severity of the pains, the vomiting, and the symptoms of collapse are common to peritonitis and to various "colics"—intestinal, gall-stone, and renal colics, and also to cardialgia. However, confusions of these affections with peritonitis are scarcely possible or may occur only at the very beginning of the affection, as a concentration of the pains upon certain portions of the abdomen, the paroxysm-like occurrence of the pains, their independence from the movements of the patient, etc., cannot be mistaken and point directly against peritonitis.

Fever.—The same as the degree of pains, so may the *temperature of the body* also be quite varying in the different varieties of peritonitis. It is generally the lower, the more markedly the symptoms of collapse are developed; thus it is especially possible that any rise of temperature may be absent from the beginning in those inflammations of the peritonæum which follow upon enterostenosis. On the other hand, in acutely occurring diffuse peritonitides we may often observe a fever of 104° F., and above, to persist for some time, which probably depends upon the quantity and virulence of the absorbed bacterial toxines in the given case.

Defecation.—*Defecation* also varies in the different forms of peritonitis. Sometimes natural peristalsis ceases entirely, owing to an intestinal paresis which is associated with peritonitis, causing *ileus* (paralyticus) with its consequences, especially fæcal vomiting. In other cases profuse *diarrhœas* (ten movements and more daily) occur in place of constipation, which usually prevails.

Septic Peritonitis.—This is especially the case in "*septic*" *puerperal peritonitis* which shows still other deviations from the usual picture of

peritonitis (delirium, cutaneous hæmorrhages, etc.), and which is characterized especially by its stormy, severe course caused by a rapid absorption of the bacterial toxines, especially that of the streptococcus.

The observation of the *ætiology*, above all, determines the diagnosis of peritonitis in a given case. *It must be established as a rule, never to diagnosticate peritonitis unless we succeed positively in determining its cause.*

Primary Peritonitis.—For, although cases occur in which peritonitis represents a *primary* affection—i. e., in which its appearance is apparently "spontaneous" or, rather, is due to the invasion of pathogenic micro-organisms into the peritoneal cavity in a manner as yet unknown—I have recently seen such an extraordinary case of primary infectious peritonitis ending fatally within a few days, in a soldier who until then had been healthy and vigorous—yet such examples of cryptogenic peritonitis are extremely rare compared with the greatest majority of cases in which peritonitis is of a *secondary* nature—i. e., results from other affections. In such cases, then, it is not difficult to demonstrate the way by which the micro-organisms (bacteria coli commune, streptococci and staphylococci) reached the peritoneal cavity.

In order to determine the diagnosis of the occurrence of (secondary) peritonitides, it is best, in my experience, to adhere to the following systematic mode of examination. At first an exact inspection and palpation of the hernial ring should be made.

Course of Examination to find the Cause of Peritonitis in the given Case.—This examination should never be omitted in any case of peritonitis. If the result is negative, we must consider other causes of peritonitis, and in women an exploration of the *sexual organs* should be done primarily. The suspicion that the peritonitis originates in the latter is justified especially if the disease develops in connection with menstruation, if gonorrhœa or an affection of the uterus or its adnexa existed until then, or if a peritonitis develops during the puerperium. The personal history should also be considered, and with regard to this, eventual diseases of the various abdominal organs should also be considered in the diagnosis.

Of the affections of the various abdominal organs, those of the stomach and intestinal canal: gastric ulcer, gastric carcinoma, severe forms of gastritis (especially toxic gastritis), tuberculous, typhoid, dysenteric, carcinomatous ulcers of the intestine, and acute occlusions (axial torsions and invaginations), are to be considered as frequent causes of peritonitis, but, above all, ulcerations of the vermiform appendix which so very commonly lead to peritonitis. The affection is much rarer as a result of abscesses of the spleen, liver, and kidneys; much more frequently does it follow the formation of concretions in the last-named organs or a suppurative inflammation of the urinary tracts; inflammation of the umbilical vessels in the newborn, etc., may also lead to peritonitis. If no reason exists for referring to one of the above frequent causes of peritonitis, we must consider rarer causes: pleurisy, pericarditis, abscesses of the mesenteric glands (especially also in typhoid infiltration), retroperitoneal abscesses, and caries of the vertebral column, etc.

It is possible that in all these diseases of the abdominal organs peritonitis is the result of the simple entrance of the generators of inflamma-

tion into the peritonæum by contiguity; or perforations of pus or bacteria into the peritoneal cavity may take place. If on such an occasion an *air-containing* organ perforates, a picture of peritonitis arises which is essentially different from that of common peritonitis, and which can be diagnosticated as such. I shall later on discuss this affection as "pneumo-peritonitis," as a special form of diffuse peritonitis.

General Affection in its Relation to the Origin of Peritonitis.—The above-named direct causes of peritonitis should always be considered primarily, and, according to their presence and their influence, we should try to look for the origin of the disease. This is advisable even then, when certain general affections which are usually connected with the causation of peritonitis, are undoubtedly present besides peritonitis.

Such are various infectious diseases: scarlatina, acute rheumatism, small-pox, erysipelas and, above all, septicopyæmia. I have also observed peritonitis in scurvy and purpura rheumatica, without having been able to find another reason for their occurrence than the original disease. If we leave out septicopyæmia, the infectious diseases may, as a rule, associate themselves with peritonitis in such a manner that the chemical toxine which is produced by the various specific micro-organisms, irritates and inflames the peritonæum. This "chemical" peritonitis, which at first is not accompanied with suppuration but with formation of a sero-fibrinous or hæmorrhagic exudate, may later become associated with a bacterial peritonitis, in such a manner that the (chemically) inflamed serosa receives pyogenic bacteria secondarily (possibly through the blood current), thus causing the originally serous peritonitis to become purulent later on **(see p. 407)****. Other chemical toxines may act in the same manner as infectious toxines; this explains, in my opinion, the decided inclination of patients with Bright's disease to peritonitis, inasmuch as the soil is prepared for the infection by irritation of the serosa in consequence of the accumulating toxic urinary substances.**

From the usual picture of diffuse acute peritonitis are to be differentiated diagnostically:

Circumscribed peritonitis with its often insignificant symptoms of inflammation, and *pneumo-peritonitis* with its fulminant symptoms, which almost always rapidly leads to a fatal termination.

CIRCUMSCRIBED ACUTE PERITONITIS

Circumscribed acute peritonitis can usually be diagnosticated only with a certain degree of *probability*, from the locally circumscribed, violent pain, and from the original disease which preceded its occurrence, ulcer of the stomach, etc. Such diagnoses are of no great value; they are, almost without exception, wrong, if based solely on the symptom of pain. The diagnosis gains in certainty only when a circumscribed exudate develops, demonstrable by percussion and palpation, and, especially, if a respiratory friction sound is palpable and audible over those parts of the peritonæum which are situated above the umbilicus. Circumscribed peritonitis, in its mild form which is associated with simple vascular injection and secretion of fibrin, forms an integral part of the diagnosis of diseases of the liver, spleen, etc., and has been repeatedly mentioned in the discussion of these diseases as perihepatitis, perisplenitis, etc. If suppuration

develops in a circumscribed area of the peritonæum, this circumscribed suppurative peritonitis is designated, according to its location, as *perityphlitic, parametritic, pericholecystitic, subphrenic, etc., abscess.* The diagnosis of these peritoneal abscesses with their well-characterized pathological pictures usually does not offer any difficulties and is not to be entered upon at this place, after having been explicitly discussed in the various chapters on these morbid conditions (perityphlitis, etc.).

PERFORATIVE PERITONITIS, PNEUMOPERITONITIS

Pneumoperitonitis.—In contradistinction to the above, perforative peritonitis presents the most severe picture among the peritonitides, especially *pneumoperitonitis,* which is brought about by the perforation of an air-containing organ into the peritoneal cavity. Here, too, bacteria and their products which enter the peritoneal cavity with the perforation, not the air as such, are the cause of peritonitis. Suddenly occurring, violent pain, rapidly developing collapse of the most intense degree, icy coldness of the skin, smallness of the pulse even to its absence on palpation, distention of the abdomen at *maximum* and vomiting characterize the pathological picture.

Vomiting, as is well known, is absent in that form of peritonitis which is caused by perforation of the *stomach.* This is usually explained in such a manner that, if a large perforation is present, the stomach, upon movements of vomiting, discharges its contents in the direction of least resistance—i. e., through the perforation, and that vomiting occurs only when the laceration has been closed by peritoneal adhesions at the place of rupture.

The examination of the abdomen shows, unless the escape of air is limited—i. e., takes places into a sacculated space, owing to preceding adhesions of the peritoneal layers—that the escaped air occupies the highest points in the peritoneal cavity. This causes a disappearance of the hepatic and splenic dulness by backward displacement of these organs, and *in place of the normal dulness, we will find a tympanitic sound*; the diaphragm is abnormally high, the epigastrium bulges out markedly, often balloon-like.

Disappearance of the Liver Dulness.—The mere disappearance of the liver dulness, *even if it can absolutely no longer be demonstrated on the anterior surface of the thorax, does not,* as I particularly emphasize, *give a positive confirmation of the presence of air in the peritonæum,* inasmuch as the intestines, especially the transverse colon, enter between the thorax and liver and may force the latter completely away from the thoracic wall. In my opinion, the diagnosis in this respect is determined *only by the condition of the percussion sounds in the axillary line.* So long as the patient is in the dorsal position, lateral dulness is usually found in both cases. But if the patient is made to assume the left lateral position, there always remains, in case meteorism causes the disappearance of the liver dulness, in the axillary line a remainder of dulness (although small) in the upper parts of the area of liver dulness—i. e., at the eighth rib—while, with free movement of the gases in the abdominal cavity, tympanitic sound here takes the place of liver dulness under such circumstances. Similar rules apply to the condition, respectively disappearance. of the splenic dulness in pneumoperitonitis.

Pneumoperitonitis caused by perforation is furthermore characterized by other, very pronounced, symptoms, so that its diagnosis can always be made with certainty:

The abdomen is markedly, and even *uniformly distended,* the surface is tense and smooth; *intestinal convolutions or peristalsis cannot be seen anywhere. Percussion* will show, if a moderate amount of free gas is present in the peritoneal cavity, *the same high and low tone in all places of the abdomen,* sometimes of a metallic timbre. If, as is usually the case, an inflammatory exudate has formed shortly after the perforation, dulness occurs in the dependent portions of the abdomen; upon movements of the patient a metallic splashing sets in, because the peritoneal cavity contains air and freely moving fluid. This succussion sound can be easily distinguished from that which arises in the stomach and intestines. In pneumoperitonitis it is brought about by a jerky palpation of the entire abdomen, especially in both flanks, while in stomach succussion it is restricted to the boundaries of the stomach. The intestinal succussion sound, finally, is distinguished by the fact that, usually, it can be produced at various places of the abdomen, sometimes even at its highest part in the centre, and that it can never be heard within so wide a range as the succussion sound in pneumoperitonitis. The respiratory sound may have a metallic resonance in the large abdominal air space. The rapidly fatal course of the affection can at least serve as a support of the diagnosis; recovery from pneumoperitonitis occurs in extremely rare cases; within twenty years I observed such a termination only four times with certainty.

CHRONIC PERITONITIS

The above-described diagnostic conditions are true of acute peritonitis; another picture is present in a chronic course of the disease, and other diagnostic criteria holds good. In chronic peritonitis it is also sometimes the question of *circumscribed,* at other times of diffuse inflammatory conditions of the peritonæum.

CIRCUMSCRIBED CHRONIC PERITONITIS

Circumscribed Adhesive Chronic Peritonitis.—The former do not cause any, or very ambiguous, symptoms: Insidiously occurring thickenings, moderate pains, caused principally by tugging of the adhesions, coalescences of the abdominal organs and, with it, less movability of the same, irregular stools, etc. Usually not more than a provisional diagnosis can be made; the longer I diagnosticate, the more fearful I have become, in the individual cases, with the assumption of a chronic adhesive peritonitis—i. e., of adhesions as cause of intestinal disturbances, obstinate, inexplicable abdominal pains, of hysteria, etc. The diagnosis becomes more certain if an original affection, for instance gastric ulcer, is present which renders the development of a chronic circumscribed peritonitis probable; the diagnosis becomes certain if coarse convolutions can be palpated regularly, and if peritoneal friction sounds are audible and palpable; but, unfortunately, such cases are rare.

Diagnostic Points of Support.—I wish to enumerate especially the most important of the factors which permit, with a certain degree of probability, of diagnosticating partial adhesive peritonitis. In the first place, the *ætiology* should be considered *particularly* in each instance. If we disregard the very common changes of the female genital apparatus, which are the consequences of chronic-adhesive peritonitis, and which belong to another realm of medicine, we find that the cause of partial adhesive peritonitis is most frequently found in the *intestine*, and particularly by the effect of *mechanical* irritants. Since we know from experiments which have recently been made, especially by Wieland, in an unobjectionable manner, that a chronic adhesive peritonitis can at any time be produced experimentally by the introduction of mechanically irritating aseptic foreign bodies, we may assume that also coprostasis, especially in the flexures of the intestines, herniæ, intestinal tumours, etc., owing to the pressure and mechanical irritation which they exert, may give rise to chronic adhesive peritonitis in the respective places. Ulcerative processes in the intestines and also in the stomach are likewise able to keep up a chronic condition of irritation in the serosa, and, as is well known, to cause adhesions. The same is true of affections of the liver, especially of gall-stones, and of affections of the spleen, urinary bladder, etc.; finally, persistent external pressure and traumatism, which act from the abdominal walls upon the peritonæum, may also be considered causes of chronic-adhesive peritonitides. If their products can be felt as convolutions or solid plates, and if to the coalescences caused thereby a deficient movability of the affected organs or a demonstrable obstruction to the permeability of the intestine corresponds, the diagnosis may be made, if at the same time the ætiology of the case points to the presence of an adhesive chronic peritonitis. But even then we must be prepared, upon exploratory laparotomy, not to find changes which were expected, or, on the other hand, to find changes which do not explain the symptom-complex observed. If a diagnosis is made on the basis of distinctly palpable convolutions, *which were regularly felt upon repeated examinations*, and if this diagnosis is fortified by the result of the distention of the stomach and intestine with air, we rarely risk making a mistake. On the other hand, we will make a wrong diagnosis in most cases, if too much value is attached to the *pains* which accompany such conditions and which are sometimes continuous, at other times predominate in the morbid picture in paroxysms in the form of cardialgias, intestinal colics, gall-stone colics, etc., and if we, on the strength of them, allow the patient to force us into the performance of an exploratory laparotomy.

Circumscribed Purulent Peritonitis.—The diagnosis of chronic circumscribed *purulent* peritonitis rests upon a slightly better foundation, because in this condition irregular fever occurs with marked rises and falls of temperature, and perforation of the pus to the external skin, after preceding fluctuation of the perforating area, or perforation into the stomach, intestine, etc., may take place. Under such circumstances, then, a sacculated pneumoperitonæum may be brought about with a clear, eventually metallic, percussion sound at circumscribed areas in which formerly dulness had existed.

DIFFUSED CHRONIC PERITONITIS

This condition is very easy to diagnosticate, if it occurs as a *consequence of an acute peritonitis*, i. e., if the threatening symptoms of the latter recede, but the exudate remains, the painfulness of the abdomen gradually decreases and only flares up temporarily if recurrences of the inflammation make their appearance. In the course of the affection there occur, then, especially at the omentum, retractions, callosities and tumour-like thickenings, which, eventually, especially if the course of the given case is not

known, may give rise to grave diagnostic errors, inasmuch as tumours of the various abdominal organs are simulated. Regarding the differential diagnosis under such circumstances, I must refer to the discussion of the diagnosis of tumours of the stomach, liver, etc. Pressure of the products of chronic peritonitis upon the nerves, causes severe neuralgias of the lower extremities; compression of the vessels is the cause of thrombosis, œdema, albuminuria; locomotion of the purulent masses produces hypostatic abscesses, external and internal perforations, fistulæ, etc. If fluid can be demonstrated in the abdomen, it is less movable, owing to the numerous adhesions of the peritonæum, than in the acute form of peritonitis. Periodical symptoms of fever, asthenia, irregular stools, temporary vomiting, and ileus, complete the picture and contribute to secure the diagnosis.

Serous Chronic Peritonitis.—The formation of exudate is very profuse in some cases. Chronic peritonitis develops quite insidiously, without the precedence of an acute stage. The fever curves are only slightly indicated, the disturbances of the patient are caused principally by the mechanical action of the fluid of the exudate in the abdomen.

Differential Diagnosis between Chronic Serous Peritonitis and Ascites.—These cases of *chronic "idiopathic" serous peritonitis* are, of course, very difficult to differentiate from *ascites*, especially because in the course of the latter, particularly after preceding, repeated punctures of the abdominal cavity, inflammatory changes in the peritonæum may also occur. The differential diagnosis is to consider, above all, the absence of a certain cause of portal-vein stasis (especially cirrhosis of the liver), and the other symptoms of portal-vein stasis which can be demonstrated besides ascites: Swelling of the spleen, hæmorrhoids, etc. A diffused chronic serous peritonitis, in contradistinction to ascites, is favoured, furthermore, by the, though often slight, painfulness of the abdomen to pressure, the accelerated pulse and an eventual presence of fever. The ætiological factor that a peritonitis has developed after a traumatism affecting the abdomen, may also be applied to the assumption of a chronic serous peritonitis. Finally, the condition of the puncture fluid is of importance for the differential diagnosis.

Condition of the Puncture Fluid in Serous Peritonitis.—The fluid in serous peritonitis is usually cloudy, although sometimes it may be quite clear, in spite of marked development of inflammatory changes in the peritoneal layers. The specific gravity in ascites is usually less than 10.12, whereas the fluid in inflammatory processes of the peritonæum, even in *serous* peritonitis, shows a slightly higher specific gravity. It may be surely considered an inflammatory exudate if the specific gravity is 10.15 and above. The puncture fluid of serous peritonitis generally also contains more albumin than that of ascites. There is no sharp line of demarcation; but we may say that an amount of albumin of less than 2 per cent is surely in favour of the ascitic, and an albumin contents of over 4 per cent in favour of the peritonitic character of the peritoneal fluid. The latter, as obtained by puncture, is rarely bloody; such a condition of the fluid points, in the majority of cases, to peritonitic processes which have arisen on the basis of tuberculosis or of carcinoma of the peritonæum, to diseases of the peritonæum which we shall discuss presently.

TUBERCULOSIS OF THE PERITONÆUM—TUBERCULOUS PERITONITIS

Symptoms of Tuberculous Peritonitis which can be Utilized Diagnostically.—The diagnosis of *tuberculosis of the peritonæum* or *tuberculous peritonitis* has recently obtained particular importance, owing to the surprising therapeutic success which was obtained by laparotomy. This affection can be diagnosticated with certainty in the majority of cases, although only provisionally in some of them.

The participation of the peritonæum in the general infection is usually entirely *without symptoms* in miliary tuberculosis (in some of the cases with secretion of a fluid exudate), and quite as latent is the development of local tuberculosis of the serosa in the neighbourhood of tuberculous ulcers of the intestine.

However, some cases of tuberculosis of the peritonæum are accompanied with symptoms which form a *pronounced morbid picture* which, under favourable circumstances, is accessible to diagnosis. These are cases in which the propagation of tuberculosis takes place through the lymph channels from the bones, genital organs (especially tubes, testicles, and epididymes), intestine, lungs, or pleura, etc., so that peritoneal tuberculosis becomes predominant in opposition to these original areas. The lung is, in most cases (in over four fifths), the primary seat of tuberculosis; it is very rare that the route which was taken by the tuberculous infection of the peritonæum cannot be found ("primary" tuberculosis of the peritonæum). A profuse inflammation will not be wanting in a more marked dissemination of the tubercle bacilli and in a subacute and chronic course of the tuberculous process in the peritoneal cavity, so that, then, fibrinous and tuberculous formation of callosities and adhesions of the abdominal organs to each other, as well as exudation of fluid (free or sacculated), set in. It is especially the omentum which, under such circumstances, becomes thickened by an enormous development of tubercles, and which is transformed, partly by the latter and in part by contraction, into a coarse, cord-like plate. Such tuberculous omental tumours can be easily felt if the exudate is not too profuse, and also tumour-like masses are found at other parts of the abdomen between the intestinal coils. However, these "pseudo-tumours" are by no means constant in tuberculous peritonitis, and, *vice versa*, they are also found in chronic non-tuberculous peritonitis.

The *exudate* is either sparse or very profuse, serous, or it presents a purulent, often also a hæmorrhagic, character. The latter is surely more frequently the case in tuberculous peritonitis than in common peritonitis. It is characteristic to a certain extent that the exudate in tuberculous peritonitis appears sacculated from the beginning or, if it was freely movable at the onset of the affection, that it usually loses its movability during the further course; then confusions with abdominal cysts may occur. Thomeyer has called attention to a peculiar distribution of dulness and tympanitic sound in the abdomen in tuberculous peritonitis. Inasmuch as the mesentery of the small intestine, which is situated more to the right (the radix

mesenterii extends in an oblique direction from the lumbar vertebræ to the right sacro-iliac symphysis), similar to the omentum, suffers contraction and retraction, the coils of the small intestine are drawn all together in a bunch into the right half of the abdomen and thus cause the tympanitic sound to appear far more diffused in the right half of the abdomen than in the left. I am not able to give an opinion as to the constancy of this symptom.

Whatever else is named among the symptoms of tuberculous peritonitis, such as vomiting, hiccough, diarrhœas and meteorism, enlargement of the spleen, atypical course of the fever, cachexia, etc., is entirely too inconstant to be of any diagnostic value whatever. A greater significance for the diagnosis of tuberculous peritonitis may be claimed by a positively demonstrated, rather frequent, combination of tuberculous peritonitis and *cirrhosis of the liver.* (See p. 184.)

Differential Diagnosis.—The pathological picture of tuberculous peritonitis, accordingly, is by no means uniform, and the diagnosis, therefore, is very apt to go astray. It is possible, in the first place, that the tuberculous tumours may simulate other neoplasms in the abdominal cavity. The tuberculous character of the condition is determined by the fever which is almost always present, and by the demonstration of a simultaneous development of tuberculosis in other organs. Furthermore, in cases with profuse exudate the question suggests itself whether ascites or a peritonitic exudate is present, a question which is always easy to decide. (See Ascites.) If we arrive at the result that the fluid is of an inflammatory character, we have then to decide whether the pathological picture is due to a simple non-tuberculous or to a tuberculous peritonitis. The latter is more probable if so-called "pseudo-tumours" can be palpated through the exudate or become distinctly prominent upon evacuation of the fluid, if the exudate is sacculated and hæmorrhagic—all of which occurs considerably more frequently in tuberculous peritonitis than in the non-tuberculous form—but, especially, if tuberculosis of the lungs or of the urogenital organs or a combination with pleurisy and pericarditis is demonstrable synchronously. But only a *Koch tuberculin injection*, which is exceptionally indicated, in my opinion, in these cases, will generally rapidly and certainly clear up the individual case if no reaction takes place. If a reaction occurs, this, *vice versa*, is generally in favour of the presence of the tuberculous process in the body, and we may then usually designate such a peritonitis as tuberculous. But we should remember that in rare cases, besides pronounced pulmonary tuberculosis, simple chronic non-tuberculous peritonitides may also occur. The fever is not characteristic in tuberculous peritonitis. Of pathognomonic importance, finally, is the *finding of tubercle bacilli in the puncture fluid.* But, unfortunately, their demonstration does not succeed in every instance. However, it should be attempted at least in all cases.

TUMOURS OF THE PERITONÆUM

SARCOMA; CARCINOMA OF THE PERITONÆUM; MESENTERIC CYSTS

Primary *sarcomata* and *carcinomata of the peritonæum* (recently acknowledged as genuine epithelial cancer) are very rare. In by far the majority of cases it is a question of *secondary* tumours which originate in an adjacent or remote organ with primarily carcinomatous degeneration. They are brought about either by simple dissemination of the carcinoma by contiguity or by metastases, in which case smallest nodules which are disseminated over the entire peritonæum similar to tubercles, cover the peritonæum, or larger tumours may develop. It is obvious that a carcinosis which rapidly disseminates over the peritonæum in the manner similar to tuberculosis, is very difficult to diagnosticate, as its symptomatology is identical with that of subacute peritonitis. The diagnosis in this case is determined only by observation of the personal history of the patient, and by the simultaneous demonstration of a carcinoma of the stomach, intestine (especially rectum), uterus, etc. An examination per rectum and per vaginam must never be omitted, therefore, under such circumstances. If then carcinomata of the rectum or of the uterus or of other locations are positively diagnosticated, the demonstration of more or less freely movable fluid in the peritoneal cavity, of peritoneal friction over the liver, sensitiveness of the abdomen, transitory fever and an eventual bloody condition of the peritoneal fluid obtained by exploratory puncture, will render the presence of a carcinomatous peritonitis very plausible, and make the original assumption of cirrhosis of the liver with ascites improbable. The following case may serve as an illustration.

Case of Peritoneal Carcinoma with Friction Sounds depending upon Intestinal Movements.—The patient was a labourer, fifty-seven years of age, admitted to the hospital October 26, 1888; he suffered from abdominal disturbances for nine months. The disease commenced with darting pains in the gastric region and dyspepsia. To this were added fatigue and increasing emaciation. His stools were constipated for years; a week previous to admission a swelling of the abdomen appeared.

Examination of the abdomen showed marked distention, distinct fluctuation, percussion shows free, easily movable fluid in the peritoneal cavity. The liver was not enlarged on percussion, could not be palpated. Hæmorrhoids at the anus. The examination per rectum revealed at the anterior wall of the rectum a rather hard tumour, the surface of which was smoothly covered with mucous membrane and the palpation of which caused slight pain. The upper end of the tumour could not be reached. The urine was free from albumin and sugar. There was no œdema of the extremities, no fever, during the five weeks in the hospital, except for two days during the last week (100.5° F. to 101° F.); progressive cachexia. The *diagnosis* was made: *Malignant tumour of the rectum* and *ascites* from an unknown cause. During the course of the disease a reduction of the ascitic fluid occurred, after which a resistance, the breadth of a hand, was felt below the right costal arch and above this resistance, and also between the xiphoid process and the umbilicus, three finger-breadths over the latter, very marked friction became palpable and audible during respiration. Symptoms of enterostenosis gradually set in, against which purges and enteroclysis were used with little success. *The intestines showed marked peristalsis; in consequence there was observed, below the umbilicus, a palpable and audible friction which*

was absolutely dependent upon the peristalsis. Slight protuberances over the surface of the liver, and in the umbilical region several hard nodes could be felt. An enlargement of the liver could not be demonstrated.

The *diagnosis* was then made: *Rectal carcinoma, secondary carcinoma of the liver, diffuse carcinomatous peritonitis.* The patient died with symptoms of increasing collapse. The *autopsy* showed: *Peritoneal layers* are covered with a continuous layer of fibrin, under which *grayish-white nodules* are visible all over, especially numerous at the mesentery. The lower abdominal cavity contained an absolutely clear fluid, a *rectal carcinoma* (the size of a pigeon egg), *hepatic carcinoma*, causing several prominences over the surface of the organ, carcinoma of the peritonæum, spreading externally upon stomach and intestine, thickening of their walls, and *marked narrowing of the lumen of the intestine in several places.*

If *larger nodes* develop or if the omentum becomes transformed into a thick nodular mass, the lumen of the intestine may, as happened in the above case, by simple compression of the intestine, be narrowed to such an extent that the picture of intestinal stenosis develops; furthermore, meteorism, œdema of the lower extremities may also occur. This also renders the diagnosis easier in so far as nodular hard tumours can be felt through the skin. But it must not be forgotten that an omentum with tuberculous degeneration forms the same sort of a tumour, in fact tuberculous swellings of the gland may appear at the periphery in a similar manner as lymph gland metastases occur in the course of carcinoma. Of importance in the diagnosis of peritoneal carcinoma is surely also the *absence* of the reaction after a test injection of Koch's tuberculin, and the eventual demonstration of carcinoma in some other organ.

Primary Carcinoma of the Peritonæum.—As the latter sign is omitted, of course, in *primary* carcinoma of the peritonæum, its diagnosis is rendered extremely difficult. Mostly, only a provisional diagnosis is permissible, if certain signs in the picture of a chronic peritonitis are to a certain extent in favour of its carcinomatous nature, namely, the occurrence of a tumour which, owing to its continually growing size and hardness, does not impress us as an omentum which is simply inflammatorily retracted or affected with tuberculous degeneration, or the fact that gradually *several* nodular tumours develop at various places of the abdomen, which, however, can only be felt if the ascites is moderate. Swelling and hardness of peripheral lymph glands are always a suspicious manifestation, and, furthermore, a very rapidly progressive cachexia is *ceteris paribus* in favour of carcinoma. The latter should be thought of especially when the puncture fluid contains blood; but even this symptom is by no means pathognomonic, as the bloody condition of the peritoneal exudate occurs not only quite commonly in peritoneal tuberculosis, but is sometimes also found in simple chronic peritonitis, and, on the other hand, may be absent also in carcinoma of the peritonæum. More marked fever is generally in favour of tuberculosis; slight rises of temperature are observed also in the course of carcinomatosis of the peritonæum. The diagnosis of primary carcinoma of the peritonæum is, accordingly, difficult in all cases, and can mostly be made only provisionally unless we succeed occasionally in demonstrating cancer cells in the ascitic fluid obtained by puncture.

Even in cases in which the diagnosis of carcinoma in the abdominal cavity is certain, the *diagnostic location of the same in the peritonæum*—i. e., the exclusion of carcinoma in other abdominal organs (if we do not consider carcinomata of the liver, spleen, and ovaries)—does not always succeed with the desired certainty.

Mesenteric Cysts.—Of *benign tumours* of the peritonæum, such as lipomata, fibromata, etc., *mesenteric cysts* have recently obtained a certain diagnostic importance. They form tumours, from the size of an apple to that of a head, which, in keeping with their usual development in the mesentery of the small intestine, are situated mostly to the right of, and below, the umbilicus. The tumour is firmly elastic and fluctuating to the touch, presents a smooth surface and is very movable—i. e., it can be easily displaced in all directions in the abdomen. Pains accompanied with vomiting and constipation, occur mostly in paroxysms, possibly caused by changes of position of the heavy tumour and by the tugging connected with it at its mesenteric root. The *diagnosis* of mesenteric cysts is always difficult. Their great movability differentiates them from *retroperitoneal* and *pancreatic cysts*, and also from echinococcus of the liver and spleen, from which latter cysts they are also distinguished by the absence of respiratory locomotion. Their differentiation from *hydronephrosis* will be extremely difficult. The varying condition of the urine, the almost always determinable immobility of the hydronephrotic sac and the demonstration of symptoms which particularly characterize hydronephrosis as a renal tumour, are determining in the differentio-diagnostic question. As to the remainder I refer to the special discussion of hydronephrosis.

Finally one word more regarding *omental tumours!* In general, they, also, are distinguished by their great movability, their location in the region of the umbilicus, their respiratory immovability, and by the fact that they are, at least usually, accompanied with ascites, and that they are principally of a secondary nature—i. e., that primary neoplasms can be determined in other locations.

ASCITES

The presence of freely movable fluid in the abdominal cavity causes very characteristic symptoms diagnostically. The abdomen is more or less distended, according to the quantity of accumulated fluid; when the patient is standing it forms a hanging fold, in the recumbent position it is distinguished by its *breadth*, especially the loss of the lateral rounding; both conditions are caused by the shifting of the fluid in the various positions of the body. The skin of the distended abdomen is free from folds, smooth and glistening, and, with excessive accumulation of fluid, shows stripes which are identical with the so-called "lineæ albicantes" and, like them, are developed principally downward; the umbilicus is blurred or even tilted forward. Venous swellings also occur frequently on the abdominal walls in the region of the epigastric veins as an expression of the impaired deflux of the venous blood through the inferior vena cava which has become compressed by the fluid (œdematous swelling of the lower extremities will also be present in this instance). Disturbances in the deflux of the portal-vein blood may give rise to development of a caput Medusæ, as described in the diagnosis of hepatic cirrhosis. *Palpation* reveals, with jerky palpation against the lateral region of the abdomen, more or less undulating fluctuations which can be easily observed at the opposite side.

Percussion shows dulness at all places at which the fluid touches the abdominal wall. If change of posture causes the fluid to recede from the abdominal wall, the dulness will be replaced by a tympanitic intestinal sound. In the dorsal position there appears, in keeping with the deep location of the fluid in opposition to the air-containing lighter intestine, a tympanitic note in the centre of the abdomen in the shape of an oval which is open above. If the patient is in an upright position, the fluid sinks from the upper parts of the lateral region of the abdomen, and the upper boundary now forms a straight line or a line which is interrupted by undulation (caused by the intestinal coils which are deposited in the fluid at varying depths). If the patient assumes the lateral position, a tympanitic note can be demonstrated in the opposite, high, lateral region. In short, the fluid and, with it, the dull sound at the abdomen rapidly change place and extension, according to the desire of the examining physician, with any change of posture of the patient. If the patient is raised in the recumbent position, upon percussion a small strip of tympany will be found near the kidney towards the axillary line. This is caused by the fact that the ascending and descending cola in their posterior circumferences are not covered by peritonæum and that, consequently, no transudate can be present here.

Diagnosis of Ascites with Slight Transudation.—In order to be able to demonstrate the above symptoms of ascites, the fluid should amount to about one to two litres. If the fluid is less, ascites cannot be discovered, as the fluid sinks below the intestines towards the vertebral column and into the true pelvis. However, even in such cases we shall rarely miss, in the farthest lateral parts, corresponding to the lowest peritoneal folds which border laterally on the cola, two narrow strips of dulness which vary with the varying postures of the patient. Still better results regarding the diagnosis of ascites with scanty transudation are obtained if the patient is made to assume the knee-elbow position, which causes the fluid to flow to the lowest place, the umbilicus, and a dull sound will be demonstrable in this region instead of the tympanitic sound.

The positive proof can be furnished in the above-described manner that *freely movable fluid* is present in the peritoneal cavity. The question will then be whether the fluid is a *transudate* or *inflammatory exudate*. The remaining pathological picture is principally determining in this case, which, on the one hand, proves itself to be peritonitis by collapse, painfulness of the abdomen, fever, vomiting, desire to urinate, etc., whereas, on the other hand, it becomes diagnosticable as ascites, if these symptoms are absent, and if it can be demonstrated that the accumulation of fluid is due to an original disease causing the transudation. As to the rest, I must refer to what I have stated in the discussion of the differential diagnosis of serous chronic peritonitis.

Ætiological Diagnosis.—Ascites not being a disease, but only a symptom-complex, *the diagnosis should never be limited to the demonstration of ascites alone, but should at the same time determine the original affection causing it.* It will be sufficient to sketch the routine of the examination in this respect:

If ascites alone is found without other œdematous swellings in the body, or, with well-developed ascites, only a swelling of the lower extremities which sets in

after the occurrence of ascites, *engorgement in the portal-vein circulation* should be thought of as the cause of ascites. Then we should look for liver affections, abdominal tumours compressing the portal vein, etc., and, secondarily, for chronic diseases of the peritonæum proper: Peritoneal tuberculosis and carcinosis, especially of the omentum. The diagnosis that it is a question of ascites depending upon disturbances of the portal-vein circulation, is confirmed by an examination of the urine, if the latter is demonstrated to be free from albumin; if it contains albumin, this would, it is true, not be direct proof against portal-vein ascites, because large accumulations of fluid may eventually produce compression of the inferior vena cava and stasis in the deflux of renal-vein blood.

If the transudation of the blood serum is not restricted to the abdominal cavity—i. e., if, besides ascites, œdema of the lower extremities and, eventually, hydrothorax, hydropericardium, are present—we have to note, primarily, whether cyanosis exists at the same time and whether urine is voided scantily and contains albumin. If these factors are present, a cardiac disease or an affection of the lungs or pleuræ impairing the pulmonary blood circulation is the probable cause of œdema in general and ascites in particular.

If the above-named manifestations of general œdema exist in a patient without the presence of cyanosis, but if pallor is present, the excretion of urine not remarkably sparse, and if a marked marasmus prevails, we must think of a pathological permeability of the vascular walls and of deficient resorbability of the peritonæum as the cause of ascites and of œdema—i. e., of scarlatina, intermittent fever, amyloid disease, cancer, and other cachectic conditions, but, above all, of Bright's disease. An examination of the urine will soon give positive information whether diffuse nephritis should be considered the cause of ascites and which form of nephritis (the excretion of urine in the acute form is, of course, also considerably diminished) is to be diagnosticated.

Differential Diagnosis between Ascites and Ovarian Cysts.—Although it appears almost impossible, from the above statements, to confuse fluid which is freely movable in the peritoneal cavity with fluid in a *sac*, yet the differentiation of very large cysts, which grow upward from the pelvis, from ascites often presents considerable difficulties in practice. I have been undecided in many a case whether I had to deal with a very large ovarian cyst or with ascites in the given case; but I believe that, upon repeated, careful examination and deliberation, apart from very rare and very complicated exceptional cases, we are able always to arrive at the correct diagnosis.

The points of view which determine this differential diagnosis are as follows: The *distention of the abdomen* has grown more in breadth than height; the reverse is the case in ovarian cysts, and *one* side, eventually, bulges out more than the other; but the umbilicus never tilts forward, as in high-graded ascites. *Palpation* shows a sensation of fluctuation in ovarian cysts which is strictly limited to the dull area, whereas in ascites it surpasses the boundaries of the dulness. On *percussion*, with the patient in the dorsal posture, we find dulness in both lateral regions in ascites, changing materially with changes of posture of the patient, tympanitic sound in the form of an oval open above in the middle of the abdomen. In ovarian cysts, however, it is rare that *both* lateral surfaces of the abdomen are dull, one probably always more so than the other; dulness can be demonstrated in the middle of the abdomen; the upper boundary of the latter is almost a straight line, slightly convex (in ascites eventually undulating and concave); the change of level of the fluid is not or only little pronounced upon change of posture of the patient.

An *examination per vaginam* shows in ascites the vaginal vault forced downward, the uterus has also descended and is slightly movable, whereas in ovarian cysts the vaginal vault does not appear bulged out, the uterus is not movable, and may be displaced upward or laterally. It is usually possible, also, to determine the origin of the cysts from the uterine adnexa.

The *chemical and microscopical examinations of the puncture fluid*, finally, will reveal certain, although not always determining, differences. Paralbumin is always found oftener in the contents of ovarian cysts than in the ascitic fluid. The fluid of ovarian cysts also contains cylindrical epithelial cells, which are never found in the

ascitic fluid. The latter contains only lymph bodies and peritoneal endothelia and red blood cells. A specific gravity of over 10.20 is, in case of doubt, directly in favour of ovarian cyst; the specific gravity of the ascitic fluid is, as a rule, even less than 10.12; the amount of albumin is also small and does not exceed 21 per mille.

Chyliform Ascites and Chylous Ascites.—The evacuated ascitic fluid has, in some cases, a milky, opalescent, creamy appearance. This is caused by its contents of most finely disseminated fat which may enter the fluid in a twofold manner. Either a common transudate becomes admixed with profuse, fattily degenerated cells of various origin (as in carcinoma, tuberculosis of the peritonæum, chronic peritonitis, fatty degeneration of the peritoneal endothelium, etc.), or, upon stasis in the chyle- and lymph-ducts (compression of the thoracic duct by tumours, thrombosis of the left subclavian vein, occlusion of the mesenteric chyle-ducts by carcinoma, etc.), chylous fluid exudes directly into the peritoneal cavity, owing to a rupture of the lymph- and chyle-vessels or because they become permeable in consequence of cancer cachexia, etc. The first form of ascites lacteus is designed as ascites chyliformis (or adiposus) (Quincke), the second form, as proper ascites chylosus. These two forms of ascites can be differentiated diagnostically in so far as numerous fat granule cells can be demonstrated microscopically in the exudate of chyliform ascites, which is not the case in chylous ascites. In this latter the fat contents of the fluid also change with fat contents of the food, and sugar is found in amounts which can be determined quantitatively (Senator).

Confusions of ascites with hydronephrosis and also with an enormous gastrectasis are possible, but should not occur with a careful examination.

Differentiation from Gastrectasis.—In this respect I wish to quote an instructive case from my practice, in which the stomach was dilated so enormously that it actually occupied the *entire* length and breadth of the abdomen. The stomach was at the same time *completely* filled with fluid and contained, besides, only a few air-bubbles. The consequence was that the abdomen, with the patient in the recumbent posture, appeared, upon percussion, dull all over, and showed, upon palpation, marked fluctuation *without* succussion sound. Under such circumstances, by reason of the physical findings, a confusion with ascites was unavoidable; and, in fact, I diagnosticated ascites in this case, because such an enormous dilatation of the stomach without any succussion sound appeared so unimaginable that its presence was never taken into consideration. And yet, this grave diagnostic error (a puncture with a trocar was, fortunately, not made) could have easily been avoided—simply by the introduction of the stomach-tube! I can only advise, therefore, to perform a test-lavage of the stomach in doubtful cases before making a positive diagnosis of ascites.

DIAGNOSIS OF DISEASES OF THE NERVOUS SYSTEM

THE diagnosis of *diseases of the nervous system* has undergone a complete transformation during the last three decades; anatomy, physiology and symptomatology have an equal share in the important acquisitions which we have obtained in this field step by step. The results of exhaustive investigations as to the minute course of the nerve fibres in the central nervous system, the functions of each individual part and the relationship of definite fibre-systems in regard to their physiology, have enabled us to analyze the separate phenomena of disease of the nervous system and to correlate the clinical picture with the results of these investigations. An inestimable method in improving our diagnoses of nervous disease was brought about by the more exact manner in which our clinical methods of research were conducted; in the first place, by the introduction of the electrical test of nerve reaction for diagnostic purposes. Thus it was possible, in this realm of pathology, to obtain an extraordinarily rich material in the diagnostic field, which could be minutely differentiated, and, as a result of this, a large number of entirely *new clinical pictures* were separated from those which were already recognisable.

The justification to establish these newly obtained diseases as nosological entities, is certainly, partially at least, questionable; nevertheless, it appears to me that the endeavour of the clinician to advance in such a method to obtain a knowledge of nervous diseases, is not only permissible, but, in the present state of our anatomical and physiological knowledge, absolutely imperative, even were it necessary at a later period, with the perfection of our knowledge, to relegate a portion of what we had separated as definite disease, into the groundwork of the old affection. The knowledge of the latter will only gain in depth and clearness, and the labour, supposedly uselessly expended in the discovery of a new affection, will be amply rewarded.

Before we enter upon the special diagnosis of nervous diseases, it is absolutely necessary, in this department of pathology, to premise the discussion of the individual diseases of the nervous system with a review of the results of anatomical, physiological and general clinical investigations. This is necessary to an appreciation of special diseases and of the methods of the diagnosis. On the other hand, the details of the technique of examination will not be entered upon here; these must be known to the diagnostician, or at least he must have familiarized himself with them, if he wishes to proceed with the necessary certainty in the diagnosis of diseases of the nervous system.

DISEASES OF THE PERIPHERAL NERVES—ANATOMICAL AND PHYSIOLOGICAL

INTRODUCTION

STRUCTURE OF THE NERVOUS SYSTEM FROM NEURONS

The nervous system, according to the latest anatomico-microscopical investigations of Golgi, Ramon y Cajal, Kölliker, Waldeyer and others, is composed of many separate, individual, uniformly related elements. These are called "*nerve units*," "*neurons*" (Waldeyer) and are arranged in a chain-like order, side by side, functionally belonging together. Each of the elements consists of: (1) The *cell body* (ganglion cell, nerve body), (2) the *axis-cylinder* (nerve continuation, axon, *neurit*), which, arising from the cell body, ends in a number of free branches (arborization) and remains bare in its course, or it may be partially surrounded with neurilemma and a medullary sheath. The cell body and the axis-cylinder are the important components of the neuron. From the cell body as well as from the axis-cylinder other "secondary" processes arise—from the cell body the *dendrons* ("protoplasmic processes"), short arborescent processes, from the axis-cylinders very minute, fine lateral offshoots, the so-called *collaterals*. All these processes, without exception, appear to end *free* with their arborizations—i. e., they do not communicate with processes of other (adjacent) neurons by direct anastomosis, but only by contact, so that there is not, as was formerly supposed, a communicating nerve net, but only a mutual surrounding and interlacing of finest ramifications of the processes (end branch, or terminal arborization neuropilem).

Function of the Neurons.—The protoplasm of the cell body contains fibrillæ that partly have their origin in the axon (axis-cylinder), and partly leave them, forming compact coils. They may unquestionably be looked upon as the actual *conducting elements*, whereas the second important element of the cell protoplasm, the basophilic *granule groups* (Nissl's tigroid substance), supply the trophic function of the cell-bodies as regards the *nutrition of the neuron*. For this substance changes, in fact eventually disappears entirely if the cell bodies are overexerted or become diseased; for this reason it is most likely that they represent the nutrition of the cells. Besides the neurofibrillæ which intersperse the cell body, and the tigroid substance, a larger or smaller *remainder of basic plasma* is found, the function of which is as yet not definitely known, but the action of which is undoubtedly related to *nervous* force. Anatomical observations as well as pathologico-anatomical and clinical experiences are entirely opposed to the view recently brought forward that the cell body is only a trophic organ.

The correctness of the entire teaching in regard to the *neurons* has been very strongly questioned within the last few years; but the reasons for this belief, according to the opinions of competent investigators in this department of anatomy, are by no means conclusive, so that for the present the neuron theory may still be adhered to, all the more for the important reason that the results of pathologico-clinical investigation strongly favour this view.

The conduction of nerve activity unquestionably results through the enchained *neurons* in the manner that the axis-cylinders act *cellulifugally* [*conveying impulses* away from the cell body], whereas the dendrons act *cellulipetally*—i. e., bringing impulses to the cell body.

In the *motor area* especially the conduction is as follows: The impulse travels from the pyramidal cells of the cortex centrifugally to the neurites originating in them, and is carried from there to their terminal arborizations in the region of the cells of the anterior horn of the spinal cord, respectively of the motor nerve nuclei of the brain. Here the arborizations of the axis-cylinder are in contact with the dendrites of the cell body, through which the impulse is carried centripetally to the cell body. From here the direct impulse is conveyed to the related axon (axis cylinder)

which further conveys the impulse to the periphery to the muscles and glands. For the conduction of motor impulses, therefore, two neurons are necessary, a *central* and a *peripheral* neuron.

The *sensory* path of conduction is somewhat more complicated, in that, eventually, a larger number than two neurons is implicated, so that between the central and peripheral neurons other neurons ("parenthetic neurons") are included. If we suppose that, from the cell of the spinal ganglion, the most peripheral ganglion cell, a dendrit (peripheral sensory fibre) and a neurit (posterior root and posterior cord filament) issues, the impulse would be transmitted from the skin through the dendrites of the cell body, as always, cellulipetally, from there through the neurits centrifugally into the spinal cord to the cells of the cord or to the cells of the nucleus gracilis and nucleus cuneatus in the medulla oblongata. From here the impulse is transmitted through the neurits of the medulla oblongata cells (sensory parenthetic neuron) to new nerve cells and from these, finally, to the tract of a tertiary, the central sensory neuron of the cerebral cortex. The transmission of the impulse from one neuron to another takes place in an ascent (on principle in the same manner as in the case of motor nerves).

In order to exert their functions, all the separate parts of the *neurons* must be intact. If there is a lesion in any part of the neuron, not only will the possibility of conduction in the chain be interfered with, but anatomical changes, degenerations of the neuron chain, will occur. In regard to the last statement it must be remembered, in general, that the *secondary degeneration is limited to the affected neuron, and does not apply to others with which it is in contact.* If the cell body is injured, the neurit which springs from it will degenerate; if, however, there is a lesion in the course of the neurit, the part that no longer goes to the cell body, the peripheral part of the neurit, will degenerate whereas the central portion will remain intact (Waller's law). The latter, however, is only true to a certain extent, from the fact that in lesion of the neurit changes are noted also in the central sections of the neurit and in the cell body belonging to it. This might be explained in the following manner, that the nutrition of the cell gradually disappears and, in the absence of the usual functional impulse, it would no longer be renewed, so that the nerve nuclei, as regards their nutrition, would be permanently damaged and in time might entirely degenerate. If the latter is the case, it follows from Waller's law that also the part of the neurit communicating with the degenerated cell (and situated centrally from the point of lesion) will become secondarily affected.

The *peripheral nerves* constitute complexes of nerve fibres of peripheral neurons that represent *conduction apparatus* in the transmission between the central organs and the peripheral end organs. They possess the property of conveying a stimulus which affects them and, indeed, in such a manner that the transmission of the stimulus occurs only in isolated affected fibres—i. e., is not conveyed to contiguous fibres (law of isolated transmission).

The stimuli which are capable of exciting the nerves are: Mechanical, chemical, thermic, electric and physiological. These stimuli act either (especially the physiological) from the centre to the periphery—*centrifugal* (in the form of movement, secretion or arrest of both functions) or, inversely, *centripetal*, from the periphery to the centre, here giving rise to sensation, or finally, *centripeto-centrifugal*—i. e., by nerve irritation moving centripetally there is brought about the stimulation of fibres which convey centrifugally to the periphery (*reflex action*).

Reflex Action.—The last-named nervous reflex action, independent of the will, is dependent upon the intactness of several anatomically combined neuron chains that are designated as "*reflex arcs.*" If we consider a reflex which is transmitted through

the spinal cord, the usual process is that as "short" reflex arcs are utilized: (see Fig. 12). *Peripheral* sensory neuron (peripheral sensory nerve fibre), spinal ganglion cell, its neurit (see above), adjacent collateral of the neurit, which extends to the motor cells of the anterior horn of approximately the same spinal-cord segment (reflex collaterals). Thus the stimulus passes from the end ramifications of the reflex collaterals into the peripheral motor neuron (dendrites of the motor cell body, cell body, motor neurit—i. e., anterior root and peripheral motor-nerve fibre).

But in the reflex process it may occur that motor cells may be stimulated by means of such collaterals, which in their entire course issue from the sensory root fibres which, upon entering the spinal cord, divide and turn upward and downward. Thus it becomes obvious that a sensory stimulus may, by means of the above-

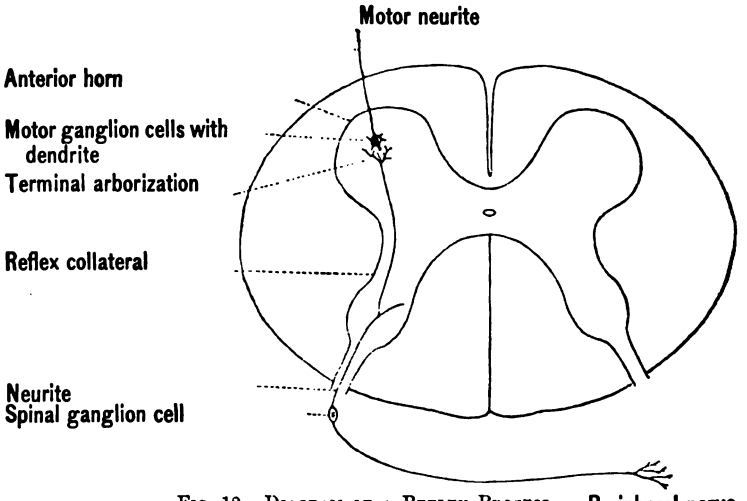


FIG. 12.—DIAGRAM OF A REFLEX PROCESS.

mentioned "*long reflex arcs*," produce reflexes in several spinal-cord segments (compare also Diseases of the Spinal Cord) and may give rise to *extensive* reflex movements (see Fig. 31). It is also obvious from the preceding remarks, that the transmission of the sensory stimulus from the posterior root fibres and their collaterals to motor cells and neurits may take place either *directly* or *indirectly* by the use of other neurons which intervene between the sensory and motor fibres, see Fig. 28, cv, ksf, kse and sc, sz, sre, and Fig. 31. *The stimulus passes ganglion cells in every reflex process*; a reflex arc without such is not conceivable according to the neuron theory. Although it has been maintained recently by reason of experiments, that reflex movements may occur without the help of ganglion cells, yet it has been admitted that the central reflex apparatus is not able constantly to functionate without ganglion cells, and that the arrested reflex action might possibly be ascribed to the nerve cell. At any rate, there exists no serious reason, for the time being, to abandon the old view that a reflex action absolutely requires ganglion cells, a view which is firmly supported by the neuron theory.

The occurrence of the reflexes may be suppressed, with complete continuity of the reflex arc, by definite mechanisms of inhibition, thus, by great stimulation of the sensory nerves, by will power, etc.; usually special nerve tracts are assumed for the arrested reflex actions. Under morbid conditions an extinction of the reflexes occurs in consequence of an interruption of any portion of the reflex arc—i. e., of the neuron chains forming it, furthermore by abnormally powerful excitation of the checking mechanisms, or, finally, by conditions in which the entire nerve activity is severely injured and has more or less lost its reaction, as in profound coma, in cerebral apoplexy, etc. *Vice versa, an abnormal exaltation of the reflex activity* occurs in patho-

logically increased irritability of the reflex arc, especially of the cell bodies of the neuron chains, or in lesion or disturbance of function of the mechanisms of inhibition. The determination of the conduct of certain reflexes is of great diagnostic significance in many cases, especially that of the so-called *tendon reflexes*, among which the patellar tendon reflex is that which is most frequently to be considered in practice.

Results of Loss of Continuity of Peripheral Nerves.—If the stimulation of the nerves is to have the normal effect, no pathological disturbance must be present of the structural and stimulation conditions in the conduction apparatus, nor under any circumstances an actual interruption in the continuity of the conduction. The interrupted continuity of the peripheral nerves is followed, not only by a suspension of its conduction, as previously stated, but also by an anatomical change of its fibres, a degeneration of the same, inasmuch as the marrow sheath in the nerve segments which are situated peripherally from the lesion, becomes fissured, disintegrates and exhibits fatty changes, the axis cylinder softens and crumbles, and the nuclei of the neurilemma swell, increase and, later, disappear.

Reaction of Degeneration.—If a motor nerve is severed or stretched, or marked pressure is brought to bear upon it, a *change of its electrical reaction* will take place besides the above-named anatomical degeneration. After the occurrence of a slight increase in the irritability of the nerve, which takes place during the first days after the action of the traumatism, a lessened response to the faradaic and galvanic currents commences on about the third day, so that, after the lapse of the first or in the middle of the second week, the electric response of the nerve disappears entirely. A different condition exists, however, with the electric response of the *muscle* which is in connection with the injured nerve. It is true, that here, too, (upon direct stimulation of the muscle) a brief increase in the irritability presents itself during the first two days, which is soon followed by a considerable diminution of the response to both currents. This muscular response to the faradaic current is entirely lost towards the middle of the second week. However, the lessened response to the galvanic current commences to increase at about this time and is replaced by a *hyperirritability*, so that noticeable muscle contractions are caused by considerably weaker currents than are required to produce a minimal contraction on the healthy side. The *qualitative* condition of these contractions changes at the same time; they do not occur, as under normal conditions, lightning-like, but in a long-drawn, weak manner. This altered form of appearance is the most constant and, therefore, the characteristic expression of degenerative processes in the muscle which are associated with the paralysis; it forms the most important characteristic of the so-called "*reaction of degeneration.*" Besides this weak course of the contractions there occurs in most, especially in severe, cases of paralysis, a change of the electric contraction formula; the intensity of the anode closure contraction (AnCC) increases, approaches the cathode CaCC, so that, gradually, it appears with the same strength of current as the latter, or even surpasses the CaCC in strength. The CaOC is also accomplished easier than normal—i. e., it is diminished by

the same current strength as the AnOC, in fact earlier than the latter in some instances. Thus it may occur that the normal contraction formula (CaCC, AnOC ≷ AnCC,CaOC) appears completely reversed, in so far as the anode reaction takes the place of the cathode reaction: AnCC,CaCC, CaOC,AnOC. But frequently an increase of the response of the muscle to *mechanical stimulation* presents itself also, besides the heightened galvanic response, so that a slight tap upon the muscle suffices to produce a distinct—sluggish—contraction.

Anatomical Changes in Nerve and Muscle.—The cause of this striking conduct of the nerve and muscle reactions is to be looked for in the ***anatomical*** changes which develop after the lesion of the motor nerve in that portion of the latter which is peripheral from the location of the lesion and in the muscle which is in connection with the injured nerve. It is obvious that the nerve, the continuity of which is interrupted and which is degenerated in the portion situated below the lesion, becomes disabled both with regard to impulses of the will as well as to electrical stimulation. More difficult to understand is the strange manner of reaction of the *muscle*. The following anatomical changes are found: An (generally not fatty) atrophy of the contractile substance, proliferation of the muscle nuclei and increase of the intramuscular connective tissue; while two weeks after the lesion of the nerve the atrophy of the embryonic fasciculi of the muscles and the proliferation of the sarcolemma nuclei are distinctly developed, at the same time the nerve endings in the muscle are still intact (Gessler). As there can be no doubt about the presence of DeR at this time, it seems that this, the galvanic, hyperirritability and the weak, *long-drawn form of contraction does depend, not upon the degeneration of the nerve, but directly upon the atrophy of the contractile substance and upon the nuclear proliferation in the muscle.* The nerve endings are also destroyed in the further course of the degeneration, while the atrophy of the muscle fibres and the nuclear increase in the sarcolemma assumes larger dimensions. If a *regeneration* of nerve and muscle occurs, it does not originate, as might be supposed, from the lesion, but, as has been demonstrated by Gessler, begins at the remotest periphery with the regeneration of the end plates; in the nerve trunk, within the old sheaths of Schwann, new nerve fibres appear which, later on, become surrounded with marrow. The interstitial connective-tissue proliferation gradually recedes during the stage of restitution in the degenerated muscle, and the entire degenerative changes may slowly reform; however, the restitution of the muscle, if it has suffered severe injury owing to the paralysis of the nerve, takes place but slowly and imperfectly. The electric and conductive conditions of nerve and muscle now also change with these regenerative processes.

The course of the reaction of degeneration varies according to the gravity of the case; in the gravest (incurable) cases the galvanic response of the paralyzed, degenerated muscle becomes continually weaker and, finally, only an insignificant weak AnCC remains as a last expression of the same, until this, too, disappears. In the curable cases, on the other hand, the CaCC again becomes prominent with the regeneration. The contractions become shorter, more lightning-like, the normal response conditions return gradually; the nerve finally again responds promptly to the faradaic and galvanic currents. It is of importance that the spontaneous mobility is usually present earlier than the electric irritability of the injured peripheral nerve; in such cases it is possible that electric stimulations which act on the nerve *above* the lesion, are able to produce muscle contractions. The normal conditions of electric reaction also recur in the muscle with the progress of the regeneration.

Modification of the Reaction of Degeneration; Atypical Forms.—It would be a mistake to believe that the above-described *typical* form of DeR is found regularly in peripheral paralyses; on the contrary, it has been found that quite numerous departures from this rule are observed, which in the main the diagnostician should understand. In contradistinction to the typical complete DeR is that form of "*partial reaction of degeneration*," in which the faradaic and galvanic responses of the nerve and also the faradaic response of the muscle remain normally developed, or nearly so, in spite of motor paralysis, whereas the *galvanic stimulation of the muscle* produces hyperexcitability, preponderance of the AnCC and sluggish contractions. Also known for a long time are those cases in which *sluggish* contractions are obtained upon *faradaic* stimulation of the nerve and muscle (faradaic reaction of degeneration). But both instances by no means represent all the possibilities of changes in the electric reaction which may be observed in peripheral paralyses. If the sluggish form of the contraction upon electric stimulation is to be considered the determining characteristic of the presence of a reaction of degeneration, ten and more variations of reaction of degeneration may be differentiated, in accordance with Stintzing, modifications which, according to his investigations, in part represent only transitory stages of the changed reaction of the diseased nerve and muscle, in keeping with the prevailing progress and retrocession of the affection. Our views regarding the significance of the various modifications of the reaction of degeneration have been considerably elucidated, and at least three principal groups of reaction of degeneration should be differentiated in future, viz.:

1. Cases in which *the nerve*, being degenerated to the end plates, *is absolutely inirritable* ("complete reaction of degeneration"), and no contraction takes place upon galvanic and faradaic stimulations, whereas the muscle responds with a sluggish contraction to the galvanic stimulation and in a varying manner to the faradaic stimulation, either not at all (the usual occurrence in complete reaction of degeneration), or sometimes promptly, at others sluggishly.

2. Cases in which the *nerve responds with prompt contraction to both currents* (constant as well as faradaic), although only upon application of strong currents (*"partial" reaction of degeneration*), whereas the muscle responds sluggishly to the constant current, and to the faradaic current either not at all or sluggishly or promptly (usual form of partial reaction of degeneration).

3. Cases which are midway between the above two groups, in which the *nerve does not possess its full ability to prompt response*, either because it responds only to one of the two currents or, although to both, to one of them only with a sluggish contraction. The muscle in these cases responds in a very varying manner, mostly sluggishly, to both currents.

Diagnostic Significance of the DeR.—In view of the results of experimental severing of peripheral motor nerves, and based upon the clinical experience that, for instance, paralyses of the facial nerve which originate from a lesion of the internal capsule (the usual seat of cerebral hæmorrhage), never show reaction of degeneration, it has been assumed for some time that the occurrence of the reaction of degeneration was the indubitable sign of a paralysis of the nerve in its *peripheral* course. But it was gradually found that affections of the spinal cord and of the medulla oblongata were also apt to cause paralyses characterized by a reaction of degeneration. As, after injury of the motor nerve, extensive degeneration takes place only in that portion of the nerve which is situated below the lesion, whereas a lesion of the motor tracts centrally from the ganglion cells of the anterior horns of the spinal column, respectively of the nuclei of the cranial nerves, is usually not followed by a degeneration of the latter and of the motor-nerve fibres situated peripherally therefrom, the assumption appears justified that the ganglion cells of the anterior horns, respectively

the nuclei of the cranial nerves, exert a conserving "trophic" action upon the peripheral nerves. Therefore, and in consideration of our modern views regarding the structure of the nervous system from neurons, the above-named ganglion cells are properly included in the peripheral tract of the motor-nerve fibres. The peripheral course of the latter, therefore, is considered to commence at the ganglion cells of the anterior horns and at the nuclei of the cranial nerves (including the same) (peripheral motor neuron), and, centrally from these, the central course of the motor-fibre tract (central motor neuron).[1] *In this sense it is permissible, upon determination of indubitable reaction of degeneration, to assume a morbid process of the peripheral motor portion of the nerve-fibre tract*—i. e., *of the motor neuron.* Then it is only a question of deciding whether the peripheral nerve trunk, including the end-organs proper, or whether the parts of the spinal cord, respectively of the middle and posterior brain and of the metencephalon which contain the cells of the motor neurons, are affected in the given case. It is obvious, therefore, that the reaction of degeneration is usually absent in affections of the spinal cord, and is but very rarely observed in cerebral maladies.

If *no* reaction of degeneration is found in the area of a motor paralysis, it does not at all follow that the paralysis may not be of a peripheral character after all. There are cases of unquestioned peripheral paralyses in which the electric contractility of the nerve and muscle remains quite unaltered or is simply reduced to both currents (without qualitative changes of the reaction). Such cases may, prognostically, be designated as paralyses which, almost without exception, run a *mild* course; but, if the irritability of the nerve to electric stimulation has been considerably affected, a rapid improvement of the paralysis cannot be counted upon, and a favourable termination can be prognosticated with less certainty, the more the signs of reaction of degeneration are developed. Paralyses of the latter kind should always be considered as severe, and a cure, if it takes place at all, usually requires many months.

Effects of Lesions of Sensory Nerves.—Materially different conditions are found in paralysis of peripheral *sensory* nerves. They arise, according to recent investigations, not in the central organ as do the motor nerves (as nerve continuations of the motor ganglion cells), but outside of the same in the cells of the spinal ganglia (respectively the ganglia of the cranial nerves). *From these, nerve continuations arise which branch off*

[1] Yet affections which concern the area of those ganglion-cell foci and of the intracentral fibres which extend from the latter to the periphery, are not regarded as affections of the peripheral nervous system, but as diseases of the spinal column, respectively of the middle and posterior brain and of the metencephalon. And correctly so! For, although the effects of maladies of the above-named portions of the central nervous system should be, and in fact are, equal to those of affections of the peripheral nerves, owing to the injury which takes place of those portions of the peripheral neurons which are situated here—i. e., intracentrally, yet the classification as affections of the peripheral nerves is, in my opinion, *not* advisable for the time being. This would be in contradiction to the anatomical classification principle generally adhered to in our present nosology; besides, sometimes the above-named portions of the central nervous system are not affected alone but in association with other parts.

to the centre (as neurits through the posterior roots) *and to the periphery* (as dendrites); *both depend upon the intactness of their ganglion cells as to their nutrition*—i. e., they degenerate as soon as they become severed from the latter.

If a sensory nerve of the spinal cord is cut centrally from the ganglion, the fibres in the peripheral nerve do *not* degenerate (except a few nerve fibres which originate in the spinal cord and which do not come in contact with the cells of the spinal ganglion, but only traverse the same), but, upward, certain portions of the spinal cord, especially the posterior columns (i. e., the main route of the fibres of the posterior roots); we shall more extensively refer to these conditions later on, when discussing the diagnosis of affections of the spinal cord. (See Fig. 30.)

The clinical effect of interrupted conductivity in the peripheral sensory nerves is *anæsthesia* as observed in the most various (traumatic, inflammatory, etc.) lesions of the sensory nerves.

DIAGNOSTIC PRELIMINARY REMARKS

Differential Diagnosis between Peripheral and Central Paralysis.—The final effect of interrupted conductivity of the motor and sensory tracts—i. e., a paralysis or anæsthesia—is the same, immaterial whether the cause of the paralysis is situated in the brain, spinal column, or peripheral nervous system. Certain difficulties are often encountered, therefore, to decide the diagnostically important question whether we are dealing with a *peripheral* or with a *central* (spinal or cerebral) *paralysis* or anæsthesia. The decision is, in part, rendered possible by the consideration of the above-mentioned conduct of the electric reaction in the paralyzed area. However, the electrico-diagnostic investigation is only *one* of the clinical expedients at our disposal to recognise the character of the paralysis in the given case. It is usually not sufficient for a positive diagnosis in this respect; in fact, quite a number of other points are to be considered at the same time, which, however, cannot be discussed until later on. But it seems advisable to me to give now at least a summary of such criteria as are generally determining regarding the diagnosis of paralysis of the peripheral nerves in contrast to the diagnosis of central paralyses:

The following phenomena are in favour of—

Peripheral Paralysis	*Central Paralysis*
Reaction of degeneration, atrophy of the paralyzed muscles.	Absence of any reaction of degeneration (except, of course, those cases in which are affected the ganglion cells of the anterior horn and the nuclei of the cranial nerve which belong principially to the peripheral motor-fibre tract, and also those fibres which extend from them to the periphery and end intracentrally). The electric contractility in the paralyzed parts is unchanged in general, nor will degenerative muscle atrophy be found in spite of long-lasting paralysis.
In paralysis of a nerve with mixed fibres simultaneous motor paralysis and	Anæsthesia and motor paralysis may exist independently, although sensory

anæsthesia in the area of distribution of the affected nerves; partial sensory paralyses are usually absent.

Precise localization of the paralytic manifestations in the area of distribution of a nerve *trunk*; all nerve branches in this area are affected.

Reflexes are entirely absent in the paralyzed or anæsthetized area if the reflex arc is totally severed by the nerve lesion. The associated movements (Mitbewegungen[1]) are also absent in complete peripheral paralysis.

Accompanying manifestations which may directly point to cerebral or spinal affection are entirely absent in the pure forms of peripheral paralysis.

and motor fibres are present in the nerve trunks of the part of the body in question; partial sensory paralyses are quite common.

Certain types of external forms of paralysis are unmistakable in extensive paralysis: Paraplegia, hemiplegia, crossed hemiplegia, etc. If the paralysis is limited, nevertheless one nerve trunk is not paralyzed alone.

Reflexes are *preserved* in the area of paralysis (or increased if inhibition tracts are effected by the disease). Associated movements (Mitbewegungen[1]) may be present.

Besides the paralysis, eventually synchronous presence of psychical disturbances, headaches, vertigo, disturbances of hearing and sight, ophthalmoscopical changes, aphasia, changes of the condition of the urine, vesical and rectal disturbances, etc.

Ætiological Supports of the Diagnosis.—The above-named general principles which are to be considered in the differential diagnosis between peripheral and central paralyses, are individually subject to certain modifications and limitations, as will be seen in the discussion of the diagnosis of the various affections of the nervous system.

But before entering upon the diagnosis of the special diseases of the peripheral nerves we wish briefly to refer to their general ætiology, in so far as the observation of such factors as are here to be considered is determining in regard to the diagnosis. The most various causes may affect the peripheral course of the nerves and injure their function, resulting in an increase or diminution of their contractility, or even in a complete suspension of nerve conduction. To be considered are: Traumatisms of various kinds, mechanical pressure and stretching (caused by new growths, inflammatory processes, etc., in the neighbourhood of the nerve), diseases affecting the nerve trunks proper (neuromata, neuritis). Furthermore, noxa of a *thermic* character should be considered as the cause of the morbid process in many cases of affections of the peripheral nerves; especially is it possible sometimes to demonstrate with certainty a *cold* to be the ætiological factor in the sudden occurrence of neuralgias and several peripheral paralyses. *Intoxications* and *infectious diseases* form another fruitful (chemical) source of peripheral nervous diseases; of the former, lead-poisoning and also, in a broader sense, as an auto-intoxication, diabetes mellitus; of the latter (the powerful effect of which upon the causation of nervous diseases has been recently determined in an increasing manner), malaria, diphtheria, and influenza are the most frequent causes of the affection.

A sufficient nutrition and an adequate blood supply are essential to the normal irritability of the nerves; the irritability decreases with advanced disturbances of nutrition. But it should be remembered that, under such conditions, *the lowering of nerve energy is usually preceded by a period during which the contractility of the nerves is increased*, a physiological fact which is of common occurrence in pathology and which accounts for the frequency of general nervousness, neuralgia, etc., in poorly nourished and anæmic individuals, or in such as are afflicted with constitutional diseases. Finally, it must be mentioned that, in a certain number of affections of the peripheral nerves, it is absolutely impossible, in spite of the most careful investigation, to find a cause for the disease.

---

[1] For full explanation of associated movements (Mitbewegungen) compare the chapter on Apoplexy.

DIAGNOSIS OF DISEASES OF THE SENSORY NERVES

Qualities of Cutaneous Sense Perceptions.—The sensory nerves cause the various qualities of sensation by stimulation of their specific end-organs. As such may be assumed in the skin: *Points of pressure, of heat and of cold*, the stimulation of which is conducted in anatomically arranged fibres, which are in connection with those points, and perceived in a varying manner in the brain at various localities which are isolated from each other (central apparatus). In the logical sequence of the law of specific energy of the nerve fibres we also assume to-day distinct *nerves of pain* with likewise isolated peripheral and central end-organs, the stimulation of which causes nothing but pain. However, this view of the character of pain perception is not generally accepted as yet, because other explanations can be given for the occurrence of this perception.

Causation of Pain.—It is well known that, under normal conditions, it requires powerful stimulation to produce pain, and the consequence of such a stimulation, immaterial upon which point of the course of the nerve it acts, is always, according to the "law of eccentric projection," the perception of a pain transferred to the periphery. The tactile nerves, on the other hand, must always be touched at the peripheral end-organs to produce a perception of pressure or of temperature by the stimulation. Stimulation of the temperature points by needle pricks is painless, the same as an irritation of the pressure points, but only when no very powerful stimulations are brought to bear or unless points are touched at which endings of sensory nerves accumulate. The general cause of *pain perception* should be looked for in the *total of stimulations* (Goldscheider). This total might be brought about in such a manner that a sum of individual stimulations accumulates in the gray substance of the spinal column (especially in its posterior part, probably in the cells), and, upon the presence of a certain quantity of stimulations, a complete discharge takes place. This hypothesis is supported by experiments of Gad and Goldscheider, according to which several separate electric opening stimuli cause secondarily, besides the individual perceptions of stimulation, a more pronounced perception, the pain. However, this conception requires, in my opinion, to be quite materially supplemented: A *weak* stimulation of sensory fibres extends centrally, probably on *one* tract of conduction or at any rate on only *a few* tracts; but, on *more marked* stimulation, several side tracts, of which quite a large number may be utilized, according to modern teachings of anatomy, are employed, besides the main tract, for conduction. These are the numerous sensory collaterals which turn into the gray substance and into the cord cells situated there, and, furthermore, most likely also the most lateral fibres of the end tree of a neurit which are in connection, not with the main cell, but with the adjacent ganglion cells. Thus the possibility exists that, upon powerful stimulation of sensory nerve endings, by means of the collaterals and of those accessory fibres of the end trees, *various ganglion cells are simultaneously* stimulated powerfully and the sum total of these stimulations is capable of causing pain, especially if the cells be charged more energetically through a number of individual stimulations. The result of this charge or stimulation of various ganglion cells is pain, which to a certain extent may still be localized in the periphery, because all the above-mentioned accessory conductions have at least *one* anatomical

starting-point. The smaller the area upon which the peripheral, powerful, pain-producing stimulation acts, the more precise will be the localization in the periphery, whereas, upon stimulation of larger peripheral areas and of the nerve trunks, an exact localization is impossible, which, according to the above statements, is quite obvious. It is quite as comprehensible that the simultaneous stimulation of a certain sum total of *central* sensory neurons, if they happen to become stimulated by morbid foci within the range of the sensory tracts in the central nervous system, may cause "spontaneous" pain (*"centrally arising pain"*).

The intensity of the pain depends upon amount and duration of the stimulation; furthermore, upon the number of nerve fibres and ganglion cells that are stimulated, and particularly upon their irritability. If the latter is pathologically increased, a slight touch of the skin may cause vivid pain. The quality of pain is quite varied; we differentiate in this respect pain of a boring, cutting, tearing, shooting, etc., character. It is obvious, from what we have stated in regard to the causation of pain in general, that severe pains "radiate"—i. e., extend to larger areas than correspond to the irritated area—also *the manner in which* this occurs.

Anæsthesia, Hyperæsthesia, Analgesia.—If the reaction of the sensory nerves for some reason or other becomes abnormal, this pathological condition becomes manifest by a diminution or increase of the irritability, as *hypæsthesia,* respectively *anæsthesia* or as *hyperæsthesia.* "Partial" sensory paralyses are also observed, so that only the pressure sense, or the temperature sense, or the pain sense alone is extinct. In contrast to the above, in conditions of *hyperalgesia* the sensory nerves react to irritants with a more marked pain perception than is in keeping with the strength of the irritant, owing to the abnormal irritability of the sensory nerves.

Neuralgia.—If in the given case it cannot be demonstrated that irritants which act from the periphery cause the pain, or if it persists even after cessation of irritation of the nerve externally, we speak of *neuralgias.* They are also distinguished by the fact that they are strictly localized to the distribution of individual nerves, and the pains occur in attacks. We may possibly be justified in assuming that, in such cases, the sensory fibres are acted upon by a number of weak irritants, the frequent repetition of which will finally be able to cause an *attack* of pain owing to unusually protracted and marked summation.

Paræsthesia means qualitatively abnormal sensations (which the patient designates as "furry," "formication," tickling, etc.), arising in such a manner that irritations which affect the sensory nerves, are not followed by the perception of pressure or pain, but by an unwonted feeling which is not in keeping with the kind and intensity of the irritation. Such qualitatively abnormal sensations may occur, as is quite comprehensible, in quantitatively abnormal reaction of the nerves—i. e., in anæsthesia and hyperæsthesia, in analgesia and hyperalgesia.

ANÆSTHESIA

The diagnosis of the various forms and degrees of anæsthesia is almost always easy. The patients themselves generally observe that one or the other portion of the surface of the body has become insensible to tactile impressions or pain irritations; this is still more the case when an organ of sense or the conduction tracts connected with the same have suffered. But

the patients do not appear to notice as distinctly the loss of muscular sense and other related sensations transmitted from the joints, bones, and intestines. An exact determination of intensity and kind of anæsthesia, however, is, under all circumstances, only possible by a thorough professional examination.

The knowledge of the details of the modes of examination is not within the limits of our purpose, and they will, therefore, only occasionally be touched upon. Only the application of the results obtained by means of such modes of examination is essential to our purpose in order to determine the diagnosis of the various forms of anæsthesia.

It is based upon the determination of anæsthesias within the limits of the organs of sense: Of sight, hearing, smell, taste and touch; furthermore of anæsthesias of the nerves which transmit the perception of common sensations. The anæsthesias in the realm of the organs of sight and hearing are treated of in the text-books on these special sciences; their discussion in this book, therefore, has been omitted.

ANÆTHESIA OF SMELL, ANOSMIA

The diagnosis of anæsthesias of the sense of smell is of a subordinate clinical significance as yet, because they occur very seldom independently and have been but little studied so far, but principally because the physician, when determining the condition, must depend upon usually very doubtful statements and upon findings which are determined by the subjective judgment of the patient. The decrease in the sense of smell is noticed in such a manner that such patients cease to observe the smell of gaseous ill-smelling or sweet-smelling substances upon inspiration or sniffing, and that all perceptions of taste in which the flavour of the substances to be tasted plays an important part, thus in the tasting of wine, are perceived in an abnormal manner. These patients complain principally of "disturbances of taste," because the absence of smell is less inconvenient to them and is therefore less observed. But a closer inspection shows at once the presence of an anosmia and not that of an anæsthesia of taste. It may then be assumed that both halves of the olfactory sphere have become anæsthetic, because only then the gustatory flavour disappears in an annoying manner. To test the sense of smell as to its functional intensity (best with an olfactometer of Zwaardemaker or of Savelieff), such substances should be selected as are exclusively scents, not such as at the same time act as powerful irritants of the sensory nerves of the nasal cavity (V, 2), (like acetic acid), because biased results of examination would be obtained upon application of the latter.

If we disregard anosmias of *central* origin, which will be referred to in the discussion of the diagnosis of affections of the brain, we must consider as peripheral anosmias only those cases in which the end-organs, the fibres of smell, and the trunk of the olfactory nerve cease to functionate as far as their roots. This can be diagnosticated best and with a degree of certainty in anosmia after fractures of the skull, in affections of the skull bones, especially also of the lamina cribrosa of the ethmoid bone, in tumours of the anterior brain and in meningitis; on the other hand, cases of *peripheral* anosmia, in which the above-named causes of injury to the olfactory nerve trunk may be excluded and in which the olfactory cells are to be considered as the probable seat of the affection, never allow of a positive diagnosis. For, as the hidden location of the olfactory region prevents an exact examination and the results of an electric investigation are too uncertain as yet, we possess too few positive points of support in such cases to venture more than a provisional diagnosis, more so because also anatomical changes of the nasal mucous membrane: Catarrhs and swellings of the same, narrowing of the olfactory canal, polypi, etc., impede or abolish the *conduction* of odours to the olfactory cells; in fact, even a certain dryness of the mucosa reduces the power of smell. Conditions of deficient receptibility of the olfactory surface for olfactory irritants will in this manner simulate nervous anosmia. At any rate, it will not be wrong to ascribe an anosmia caused by excessive

starting-point. The smaller the area upon which the peripheral, powerful, pain-producing stimulation acts, the more precise will be the localization in the periphery, whereas, upon stimulation of larger peripheral areas and of the nerve trunks, an exact localization is impossible, which, according to the above statements, is quite obvious. It is quite as comprehensible that the simultaneous stimulation of a certain sum total of *central* sensory neurons, if they happen to become stimulated by morbid foci within the range of the sensory tracts in the central nervous system, may cause "spontaneous" pain ("*centrally arising pain*").

The intensity of the pain depends upon amount and duration of the stimulation; furthermore, upon the number of nerve fibres and ganglion cells that are stimulated, and particularly upon their irritability. If the latter is pathologically increased, a slight touch of the skin may cause vivid pain. The quality of pain is quite varied; we differentiate in this respect pain of a boring, cutting, tearing, shooting, etc., character. It is obvious, from what we have stated in regard to the causation of pain in general, that severe pains "radiate"—i. e., extend to larger areas than correspond to the irritated area—also *the manner in which* this occurs.

Anæsthesia, Hyperæsthesia, Analgesia.—If the reaction of the sensory nerves for some reason or other becomes abnormal, this pathological condition becomes manifest by a diminution or increase of the irritability, as *hypæsthesia,* respectively *anæsthesia* or as *hyperæsthesia.* "Partial" sensory paralyses are also observed, so that only the pressure sense, or the temperature sense, or the pain sense alone is extinct. In contrast to the above, in conditions of *hyperalgesia* the sensory nerves react to irritants with a more marked pain perception than is in keeping with the strength of the irritant, owing to the abnormal irritability of the sensory nerves.

Neuralgia.—If in the given case it cannot be demonstrated that irritants which act from the periphery cause the pain, or if it persists even after cessation of irritation of the nerve externally, we speak of *neuralgias.* They are also distinguished by the fact that they are strictly localized to the distribution of individual nerves, and the pains occur in attacks. We may possibly be justified in assuming that, in such cases, the sensory fibres are acted upon by a number of weak irritants, the frequent repetition of which will finally be able to cause an *attack* of pain owing to unusually protracted and marked summation.

Paræsthesia means qualitatively abnormal sensations (which the patient designates as "furry," "formication," tickling, etc.), arising in such a manner that irritations which affect the sensory nerves, are not followed by the perception of pressure or pain, but by an unwonted feeling which is not in keeping with the kind and intensity of the irritation. Such qualitatively abnormal sensations may occur, as is quite comprehensible, in quantitatively abnormal reaction of the nerves—i. e., in anæsthesia and hyperæsthesia, in analgesia and hyperalgesia.

ANÆSTHESIA

The diagnosis of the various forms and degrees of anæsthesia is almost always easy. The patients themselves generally observe that one or the other portion of the surface of the body has become insensible to tactile impressions or pain irritations; this is still more the case when an organ of sense or the conduction tracts connected with the same have suffered. But

the patients do not appear to notice as distinctly the loss of muscular sense and other related sensations transmitted from the joints, bones, and intestines. An exact determination of intensity and kind of anæsthesia, however, is, under all circumstances, only possible by a thorough professional examination.

The knowledge of the details of the modes of examination is not within the limits of our purpose, and they will, therefore, only occasionally be touched upon. Only the application of the results obtained by means of such modes of examination is essential to our purpose in order to determine the diagnosis of the various forms of anæsthesia.

It is based upon the determination of anæsthesias within the limits of the organs of sense: Of sight, hearing, smell, taste and touch; furthermore of anæsthesias of the nerves which transmit the perception of common sensations. The anæsthesias in the realm of the organs of sight and hearing are treated of in the text-books on these special sciences; their discussion in this book, therefore, has been omitted.

ANÆTHESIA OF SMELL, ANOSMIA

The diagnosis of anæsthesias of the sense of smell is of a subordinate clinical significance as yet, because they occur very seldom independently and have been but little studied so far, but principally because the physician, when determining the condition, must depend upon usually very doubtful statements and upon findings which are determined by the subjective judgment of the patient. The decrease in the sense of smell is noticed in such a manner that such patients cease to observe the smell of gaseous ill-smelling or sweet-smelling substances upon inspiration or sniffing, and that all perceptions of taste in which the flavour of the substances to be tasted plays an important part, thus in the tasting of wine, are perceived in an abnormal manner. These patients complain principally of "disturbances of taste," because the absence of smell is less inconvenient to them and is therefore less observed. But a closer inspection shows at once the presence of an anosmia and not that of an anæsthesia of taste. It may then be assumed that both halves of the olfactory sphere have become anæsthetic, because only then the gustatory flavour disappears in an annoying manner. To test the sense of smell as to its functional intensity (best with an olfactometer of Zwaardemaker or of Savelieff), such substances should be selected as are exclusively scents, not such as at the same time act as powerful irritants of the sensory nerves of the nasal cavity (V, 2), (like acetic acid), because biased results of examination would be obtained upon application of the latter.

If we disregard anosmias of *central* origin, which will be referred to in the discussion of the diagnosis of affections of the brain, we must consider as peripheral anosmias only those cases in which the end-organs, the fibres of smell, and the trunk of the olfactory nerve cease to functionate as far as their roots. This can be diagnosticated best and with a degree of certainty in anosmia after fractures of the skull, in affections of the skull bones, especially also of the lamina cribrosa of the ethmoid bone, in tumours of the anterior brain and in meningitis; on the other hand, cases of *peripheral* anosmia, in which the above-named causes of injury to the olfactory nerve trunk may be excluded and in which the olfactory cells are to be considered as the probable seat of the affection, never allow of a positive diagnosis. For, as the hidden location of the olfactory region prevents an exact examination and the results of an electric investigation are too uncertain as yet, we possess too few positive points of support in such cases to venture more than a provisional diagnosis, more so because also anatomical changes of the nasal mucous membrane: Catarrhs and swellings of the same, narrowing of the olfactory canal, polypi, etc., impede or abolish the *conduction* of odours to the olfactory cells; in fact, even a certain dryness of the mucosa reduces the power of smell. Conditions of deficient receptibility of the olfactory surface for olfactory irritants will in this manner simulate nervous anosmia. At any rate, it will not be wrong to ascribe an anosmia caused by excessive

odours, or one which remains after severe catarrhs of the nasal mucous membrane, to an anæsthesia of the end-organs of the olfactory nerve.

ANÆSTHESIA OF TASTE, AGEUSIA

Gustatory anæsthesia offers a decidedly greater clinical interest than anosmia. Before entering upon the diagnosis of the various forms of ageusia, it is absolutely necessary to refer briefly to the anatomical and physiological conduct of those nerve tracts which are to be considered in tasting.

Anatomical and Physiological Conduct of the Gustatory Nerves.—To begin with, it must be stated that the question of that participation in the perception of taste which belongs to the various nerve fibres, is, of course, very difficult to solve by animal experiments; the decision of certain questionable points in this chapter is most likely to be expected from *clinical* experience. ***It has been positively demonstrated that the glossopharyngeal nerve is a gustatory nerve.*** It is distributed as such in the *root of the tongue*, the soft palate and its surroundings; but the ***anterior two-thirds of the tongue*** taste by stimulation of fibres which extend from the ***lingual nerve*** and to the greatest part pass to the *chorda tympani* and from here into the facial nerve (see Fig. 13). The stimulation of taste is then transmitted, from the tract of the facial nerve ***through the small superficial petrosus nerve, which branches off from the geniculate ganglion of the facial nerve, into the third branch of the fifth nerve and its spinal root to the nucleus of the glossopharyngeal nerve.*** But, besides the main route leading to the brain, other accessory tracts are at the disposal of the conduction of the perception of taste, namely:

In the tract of the glossopharyngeal nerve, through the connection of the stylohyoid (VII) outside of the stylomastoid foramen with the IX cranial nerve, furthermore through the anastomosis between the facial nerve—the small superficial petrosus, Jacobson's nerve—petrous ganglion (IX); it is very probable that another communication exists between the VII and IX nerves through the *Portio intermedia Wrisbergii*, inasmuch as the latter grows as a fine root out of the geniculate ganglion of the facial nerve, to enter into the medulla oblongata along the facial nerve trunk, ending in the nucleus of the glossopharyngeal nerve (see Fig. 13).

In the tract of the trigeminus through the anastomosis of the geniculate ganglion (VII) with the large superficial petrosus nerve—Vidianus sphenopalatine [Meckel's] ganglion (V, 2), furthermore through the direct connection between the otic [Arnold's] ganglion (V, 3) with the chorda tympani. The facial nerve anastomoses, besides, in its peripheral course *below the stylomastoid foramen,* with the fifth nerve, especially with the auriculotemporal nerve (V, 3).

It has become likely, according to the most recent anatomical investigations, that the gustatory fibres of the lingual nerve, or chorda, enter into the same end nucleus as the gustatory fibres of the glossopharyngeal nerve, namely into the long nuclear column adjacent to the solitary fasciculus, the "nucleus of the glossopharyngeal nerve," so that the latter may be considered the collective point of the various gustatory fibres (see the red tracts in Fig. 13).

Disturbances of taste, as they occur according to clinical experiences under the most varying conditions, are explainable upon this basis. Thus it is quite obvious that the perception of taste is lost in the posterior third of the tongue upon isolated *paralyses of the glossopharyngeal nerve*, also that, in cases of isolated anæsthesia of the trigeminus caused by *affection of the trunk of the fifth nerve* at the base of the skull, ageusia has been positively determined in the anterior two thirds of the tongue, and also positively in *lesions of the facial nerve in the petrous bone* between the entrance of the chorda tympani and geniculate ganglion. On the other hand, *disturbance of taste may be absent*, if only the above-named *accessory tracts* are affected, and this also holds good for the persistence of perception of taste in ***basal paralyses of the trunk of the facial nerve,*** obviously because here the gusta-

tory fibres of the chorda tympani, which disappear in the intermediate nerve, represent an inferior gustatory tract in comparison to the majority of gustatory fibres of the chorda tympani in the tract of the small superficial petrosal nerve and in the third branch of the trigeminus.

Examination of the Gustatory Disturbance.—To determine an impediment or suspension of taste sensation, various places of the tongue are painted with solutions which contain one each of the substances of the known four qualities of taste (salty, sour, sweet, bitter). Of the greatest

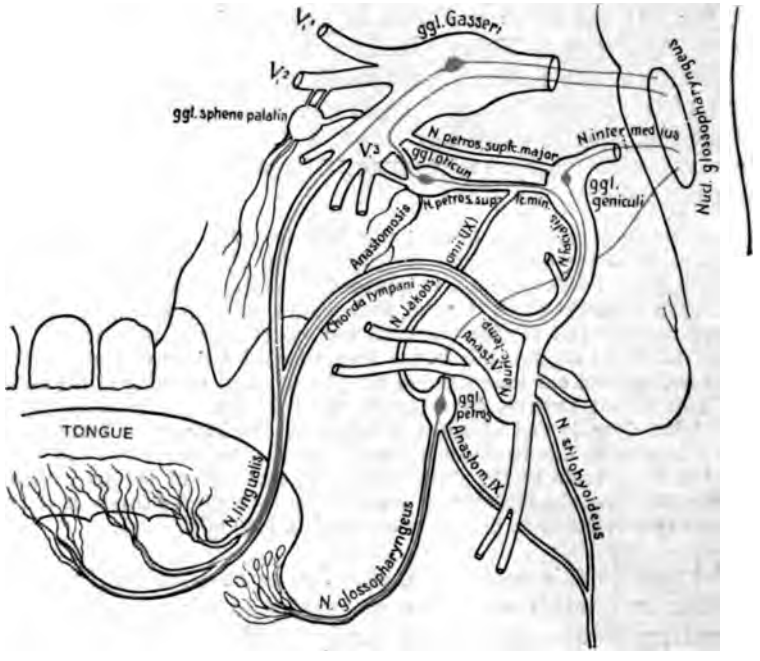


FIG. 13.—COURSE OF THE FACIAL NERVE AND ITS COMMUNICATIONS WITH THE TRIGEMINUS AND GLOSSOPHARYNGEAL NERVES. COURSE OF THE MAIN ROUTES OF THE GUSTATORY FIBRES MARKED RED.

importance, above all, is the determination of a deficient perception of the taste of sweet and bitter substances, because they only irritate the gustatory fibres, whereas sour and salty substances incidentally also irritate the tactile nerves of the tongue, and their application to the investigation of a doubtful ageusia can, therefore, but furnish questionable results. It is understood that the examination should be made with the necessary precautions on the protruded tongue, etc. The examination of the reaction of the gustatory nerves to the electric current has as yet not been sufficiently applied diagnostically, although the constant current always causes perceptions of taste (an acid taste at the positive, alkaline-burning at the negative pole).

If we do not consider conditions in which the conduction of irritations of taste to the terminal organs of the gustatory nerves is impaired

by a thick coating of the tongue, abnormal dryness of the lingual mucous membrane, and if we also leave out of question those central ageusias, the localization of which in the brain, in view of our lack of knowledge as to the cerebral course of the gustatory fibres, we can, at best, not even surmise, the diagnostic analysis of ageusia is, in general, to proceed along the following lines:

Mode of Diagnosis in the Special Forms of Ageusia.—At first it is to be determined in what part of the tongue a disturbance of taste is marked, whether unilateral or bilateral, whether in the anterior or posterior half of the tongue. If the latter condition is present we think of an affection of the *glossopharyngeal* nerve. This eventuality, however, has been constructed theoretically rather than actually observed, as cases of isolated paralysis of the glossopharyngeal nerve are, under all circumstances, rarities of the first order, and such affections of the glossopharyngeal nerve as are associated with other nerve paralyses represent very complicated objects of diagnosis, difficult to explain.

The perception of taste was found intact in progressive bulbar paralysis with its typical affection of the nerve nuclei in the medulla oblongata, among which also the glossopharyngeal nerve nucleus was proved to be degenerated in some instances. However, such findings which, at the first glance, seem remarkable, will not be appreciated to their full significance until our investigations regarding the degree and the extent of the degeneration in the long-stretched column of the glossopharyngeal nerve nucleus and our knowledge of the physiological meaning of the individual ganglion cells of the nucleus, have become more precise. If, occasionally, in the course of tabes dorsalis and of multiple sclerosis, disturbances of taste have been observed, this may be due to degeneration of the nucleus, respectively of the nerve fibres of the nerve roots of the glossopharyngeal nerve or of the trigeminus (the spinal root), but, possibly, it may also be caused by anatomical changes of the peripheral nervous system (such as have been observed in both affections).

In contrast to this occurrence, anæsthesias of taste in the region of the anterior two thirds of the tongue are quite common events. They point, contrary to what has been so far discussed, to an inhibition of conduction in the *chorda tympani* or in the complicated centripetal course of gustatory fibres of the chorda. If impairment of taste in this region has been determined, we must ascertain further whether, besides, paralysis of the facial nerve or anæsthesia of the trigeminus is present, or whether ageusia exists without this complication.

Ageusia without Symptoms of Paralysis of the Facialis or Trigeminus Anæsthesia—Ageusia complicated with Paralysis of the Facialis.—In the latter instance we must think of an *isolated affection of the chorda*; *but then the sensibility of the tongue may also be disturbed*, because sensory nerve fibres originating in the mucous membrane of the tongue may also extend into the chorda.

If pronounced paralysis of the facial nerve is present besides ageusia, we must decide in regard to the seat of the facial nerve paralysis. In most cases it is probable, at the outset, that the paralysis originates in that portion of the facial nerve which is situated between the entrance of the chorda tympani and the geniculate ganglion. It will be well to take this diagnostic principle into consideration and not until a morbid condition of this

portion of the course of the facialis can be excluded, to assume an affection of this nerve at another place to be the cause of gustatory paralysis. The diagnosis of paralysis of the facialis at the above-named locality of its course is easy, since it is characterized by very marked additional symptoms (decreased salivation and paresis of the velum palati), as will be fully explained in the discussion of paralysis of the facial nerve.

If a positive reason exists for believing that another part of the course of the facialis is affected, we may, in case the paralysis of this nerve is *not* accompanied with disturbances of hearing, of salivation and of the position of the soft palate, think of the possibility that an ageusia is exceptionally caused by inhibition of conduction in the gustatory fibres which extend from the chorda to the periphery through the facialis nerve to the auriculotemporal nerve (V, 3). However, quite as probable would be that the inhibition of nerve transmission affects another anastomosis of the facialis, viz., the communication between the stylohyoid and glossopharyngeal nerves. The interruption of that accessory tract of taste transmission ought to be sufficient in such cases to produce at least partial ageusia.

Ageusia complicated with Anæsthesia of the Trigeminus.—*Anæsthesia in the region of the trigeminus with disturbance of taste in the anterior half of the tongue* means that it is a question of an inhibition of conduction in the trunk, in the second or in the third branch of the fifth nerve, or, again, in the lingual nerve alone. *For this nerve, also, conveys gustatory fibres* (although only a small portion) to the brain (compare Fig. 13). A differential diagnosis between affection of the chorda and affection of the lingual nerve over the origin of the chorda presents difficulties, especially because, as previously mentioned, besides gustatory fibres, sensory fibres are likewise contained in the chorda. The diagnostic conclusion which is usually drawn, that a lingual affection over the origin of the chorda causes insensibility of the tongue to tactile irritations without disturbance of taste, whereas an affection of the chorda, on the contrary, produces only disturbance of taste without anæsthesia and symptoms of facial paralysis, is, therefore, not correct in this precise meaning. It is advisable, rather, to define the diagnosis according to the preponderance of one of these two symptoms, so that pronounced impairment of taste with slight sensory disturbances of the tongue points to affection of the chorda, whereas marked anæsthesia with an indication of disturbance of taste is in favour of interruption of continuity in the lingual nerve over the origin of chorda; the simultaneous presence, finally, of total ageusia and total anæsthesia of the lingual mucous membrane would mean an affection of the lingual nerve below the origin of the chorda. Of course, it is presumed in the last-named cases that the anæsthesia only refers to the mucous membranes of the tongue and of the oral cavity. If, besides ageusia, symptoms of anæsthesia are present in the remaining region of the trigeminus, we would have to decide, according to the distribution of the anæsthesia, whether the (second or the) third branch of this nerve, together with its communications with the tracts of the gustatory fibres, is injured, or whether, owing to anæsthesia in the entire distribution of the fifth nerve, an affection of the entire trunk of the trigeminus should be diagnosticated.

CUTANEOUS ANÆSTHESIA

Partial Paralyses of Sensation.—As previously stated, this category of nervous affections embraces conditions of decreased perceptibility of the nerves which transmit the various qualities of sensation: Sensations of pressure, temperature and pain. It may occur, in such an instance, that the disturbance affects all of these sensations or only some of them (partial sensory paralyses), so that, in one case, only the pressure sense or only the temperature sense is disturbed, while in others both are intact and only the sensation of pain is lost. Such *partial sensory paralyses* are *not* to be looked for in *peripheral anæsthesia,* not considering quite unusual conditions (partial degeneration of the fibres in neuritis).

The various fibres being closely crowded in the sensory nerve trunks, the function, therefore, of all of them will be impaired. But, according to what we know physiologically in regard to the anatomical distribution of the terminal organs, it is not probable from this point of view that, in the region of these terminal organs in the skin in which an isolated disturbance of the various sensory qualities is most likely to be expected, isolated partial sensory paralyses will occur. This is different in *central* anæsthesias. Partial sensory paralysis can be quite usually observed in cases of anæsthesia which originate in the brain and, especially, in the spinal cord. Whereas the posterior roots that enter the spinal cord still contain the entire mass of sensory fibres, their destruction, therefore, would be conducive to an arrest of any kind of sensation, with the entering of the sensory nerve fibres into the spinal cord there occurs a locally separate distribution of the nerve fibres conducting the different sensory qualities, so that now, according to the special seat of the spinal-cord affection, partial sensory paralyses are very liable to occur. This, in particular, also holds good for that sensation of pain which, as we have seen, is probably dependent upon transmission through the ganglion cells and upon their being intact.

Differential Diagnosis between Central and Peripheral Anæsthesias.—Besides the absence, which is usually complete, of partial sensory paralyses we must, furthermore, consider in the diagnosis of peripheral anæsthesias that the *skin reflexes* and, according to the extent and cause of the anæsthesia, also the *tendon reflexes* appear diminished or are entirely *absent.* Also the observation of the exact localization of anæsthesia upon the distribution of an individual nerve trunk or even nerve twig and the demonstration of a combination of motor disturbances with anæsthesia (if the nerve conducts mixed fibres) contributes to the establishment of a correct diagnosis—i. e., to differentiate peripheral from central anæsthesias.

Symptoms of Anæsthesias according to the Various Localizations.—*The symptoms of anæsthesias* which can be utilized diagnostically are partly of a subjective, partly of an objective kind. In more pronounced degrees of anæsthesia the patients will at once notice the numbness of skin sensation, often in the most disagreeable manner; this is especially the case if *the hands* have become *anæsthetic* and useless for more delicate work, or if *the soles have lost their tactile sense,* the patient thus losing the correct estimation of his bodily posture. In the latter case, if the eyes are closed and thus the important regulating influence upon co-ordination, which is transmitted from the organ of sight, is removed, the equilibration of the body suffers and the body commences to sway; however, to be troublesome to the patient, plantar anæsthesia must be present in both feet. Anæsthesia of the soles is rarely of a peripheral character, it is usually the expression of a central, especially a spinal, affection.

Trigeminus Anæsthesia.—Less troublesome, but very marked in their symptoms, are *anæsthesias in the distribution of the trigeminus nerve*: The skin of the face and the mucous membranes of the adjacent cavities of the body are insensible to mechanical, thermic and chemical irritations; thus the patients do not feel the entrance of a foreign body into the eye, if sharp irritations strike the nasal mucous membrane, if the teeth come in contact with the tongue, if morsels of food enter between teeth and buccal mucous membrane, etc. In consequence, injuries of the

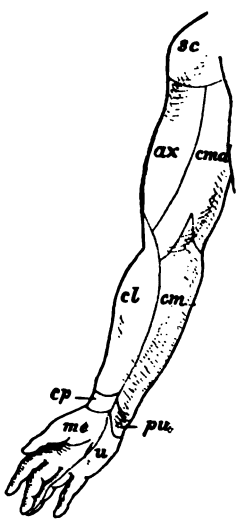


Fig. 14.

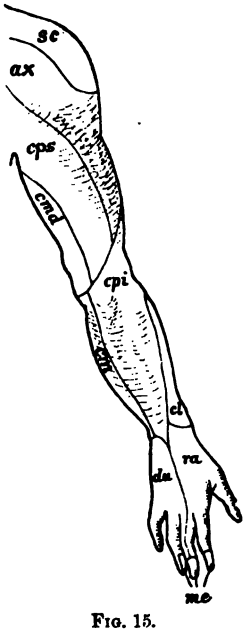


Fig. 15.

Fig. 14.—Distribution of the Sensory Cutaneous Nerves at the Anterior Surface of the Upper Extremity. (After Henle.)

sc, Supraclavicular nerves (from the cervical plexus); *ax*, Cutaneous branch of the axillary nerve; *cmd*, *cm*, and *cl*, Median cutaneous, median, and lateral nerves; *cp*, Cutaneous palmar nerve, branch of the median nerve; *pu*, Ulnar palmar nerve, branch of the ulnar nerve; *me*, Median nerve; *u*, Ulnar nerve.

Fig. 15.—Distribution of the Sensory Cutaneous Nerves at the Posterior Surface of the Upper Extremity. (After Henle.)

sc, Supraclavicular nerves (from the cervical plexus); *ax*, Cutaneous branch of the axillary nerve; *cps* and *cpi*, Superior and inferior posterior cutaneous nerves from the radial (ra); *cmd*, *cm*, and *cl*, Median cutaneous, median, and lateral nerves; *du*, Dorsal ramifications of the ulnar nerve; *ra*, Radial nerve; *me*, Median nerve.

tongue, ulcers of the mouth, conjunctiva and cornea may form, in fact a complete panophthalmitis may occur. To accomplish this, however, various other factors contribute, besides the effect of the trauma which, without hindrance, acts externally upon the insensitive eye, namely, deficient secretion of tears (caused either by paralysis of genuine secretory fibres of the V or by impairment of reflex stimulation of tear secretion) and probably also irritative processes in the paralyzed nerves which, to conclude from the experience gained up to the present time, are followed by general trophic disturbances in the periphery. Furthermore, according to what we know of the occurrence of salivation in a reflex manner by irritation of trigeminus fibres in the oral cavity, salivation may decrease (I have no personal experience in this respect); and the perception of taste will also suffer, as we have seen previously. If the disease affects the trunk of the trigeminus or the entire third branch, there exists also, besides anæsthesia, a paralysis of the *muscles of*

mastication with secondary atrophy of the latter. **A deviation in the positions of** the soft palate and of the uvula was also observed in **trigeminus paralysis:**

Slightly low position of the posterior soft palate on the side of the paralysis and oblique position of the uvula towards the affected side, probably caused by paralysis of the sphenostaphylinus muscle which is innervated from V, 3. (C. W. Müller.)

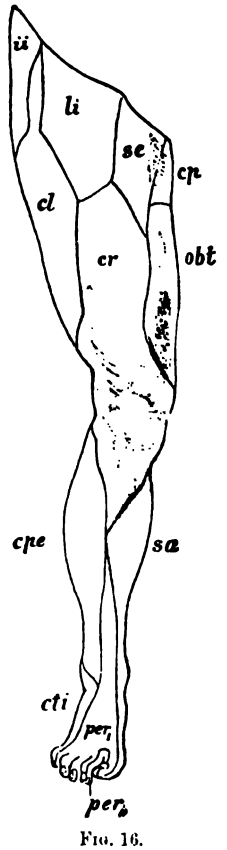


Fig. 16.

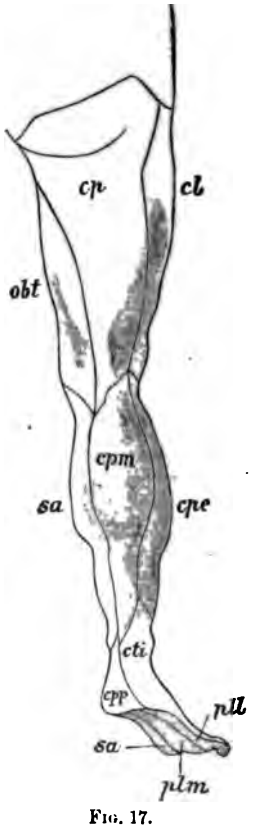


Fig. 17.

Fig. 16.—Distribution of the Sensory Cutaneous Nerves to the Anterior Surface of the Lower Extremity. (After Henle.)

ii, Ilio-inguinal nerve; *li*, Lumbo-inguinal nerve; *se*, External spermatic; *cp*, Posterior cutaneous; *cl*, Lateral cutaneous; *cr*, Crural; *obt*, Obturator; *sa*, Saphenous; *cpe*, Peroneal communicating nerve; *cti*, Tibial communicating nerve; *per'*, Superficial branch of the peroneal nerve; *per''*, Deep peroneal nerve.

Fig. 17.—Distribution of the Sensory Cutaneous Nerves to the Posterior Surface of the Lower Extremity. (After Henle.)

cp, Posterior cutaneous; *cl*, Lateral cutaneous; *obt*, Obturator; *sa*, Saphenous; *cpm*, Posterior median cutaneous nerve; *cpe*, Peroneal communicating nerve; *cti*, Tibial communicating nerve; *cpp*, Cutaneous plantar nerve; *pll*, External plantar nerve; *plm*, Internal plantar nerve.

After it has been determined beyond a doubt, by an examination with the different means of testing the intensity of sensation (touching of the skin with cold and warm objects, with differently shaped objects, placing of weights, etc., or, to determine the degree of sensibility to pain, with

the faradaic current, the point of a needle, etc.), that the sensibility of the skin has become impaired, it is then necessary to find the extent and limitation of the anæsthetic district in that, in examining, we advance from the latter to the healthy parts and *vice versa*. Then follows the deliberation *which nervous tract is affected.* Determining in the latter respect is solely the knowledge of the anatomical distribution of the individual peripheral nerves; it will be fully sufficient, therefore, instead of discussing in particular anæsthesia of the different nerve districts (especially as the most important facts regarding paralysis of the respective cutaneous branches will be stated in the discussion of symptoms which are peculiar to paralysis of the various motor nerves), to give some diagrams of the distribution of the sensory nerves as guides (see Figs. 14 to 17, inclusive).

Anæsthesia Dolorosa.—Incidentally it may be mentioned that sometimes violent pains occur in the anæsthetized areas of the skin (anæsthesia dolorosa). The diagnosis of this special form of anæsthesia as such is easy; neither does the explanation of the manifestations which at first glance appear remarkable, offer any difficulties if the painful area corresponds exactly to the area of the cutaneous nerves which behave anæsthetically. It is then the question of an anæsthesia *in which the conduction in the peripheral nerve trunk is interrupted* by severing of the same, neuritis, etc., whereas *in the central portion of the nerve irritations occur* the effect of which is, according to the law of "eccentric projection," a sensation of pain at the periphery. Anæsthesia dolorosa is most frequently observed in severe organic affections of the trigeminus.

Peripheral Analgesia, Muscular Anæsthesia.—*Subjective sensory anæsthesias* [*Gemeingefühlsanästhesien*"]. This category embraces analgesias, muscular anæsthesias, loss of the subjective sensations transmitted by the joints and entrails. It is only in the rarest cases a question of *peripheral* anæsthesia; subjective sensory anæsthesias are, rather, of a central, especially spinal, character and play an important *rôle* in the diagnosis of cerebral and spinal-cord diseases. The most reliable factor for the diagnosis of *peripheral subjective sensory anæsthesias* is, by all means, the ætiology of the given case, in that the peripheral character of the same is to be assumed in practice only when a certain nerve twig, a circumscribed cutaneous area, a muscle, etc., have become subject to a deep lesion. According to what we have discussed in regard to the origin of pain, it may be presumed that analgesias originating in the periphery are also associated with cutaneous anæsthesia.

HYPERÆSTHESIA—NEURALGIA

The same as anæsthesia so, also, does *hyperæsthesia*—increased irritability of the sensory nerves—concern partly the higher sensory organs, partly those of the skin.

Hyperæsthesias of Smell and Taste.—*The hyperæsthesias of the olfactory and gustatory nerves* is of little practical significance and scarcely occurs as a peripheral affection, whereas peripheral paræsthesias of smell and taste are sometimes observed as consequences of lesions of the olfactory and facial nerves. The above-named deviations in the reaction of both sensory nerves are, usually, of a central character and occur as frequent symptoms in the clinical picture of hysteria and neurasthenia, in epilepsy and psychoses.

Cutaneous Hyperæsthesia.—In contrast to these generally rare hyper- and paræsthesias of the sensory nerves, *hyperæsthesia of the cutaneous nerves due to peripheral causes* is a symptom which can be observed rela-

tively often in inflammations of the skin, neuritis, etc. The diagnosis is based upon the fact that the reaction of the tactile nerves is increased upon application of irritations to the skin. The increased irritability may be so considerable that even the slightest touch of the skin (not, as normally, only increasing pressure) causes painful sensations instead of pressure sensation. It is determining for the diagnosis that external irritations are to act upon the hyperæsthetic nerve districts to express the morbid hyperirritability of the nerves.

Neuralgias.—Different conditions prevail in *neuralgia,* in which, although mild external irritations may also produce pain, the characteristic of the affection consists in the fact that the, *mostly very violent, pain persists after cessation of the irritation, furthermore, also occurs, without demonstrable external irritation, in certain nerve trunks or twigs and recurs in periodical attacks.*

Attention should also be paid in the diagnosis of neuralgias and in the differentiation of this condition from others which are likewise accompanied with paroxysms of pain, to *painful areas* (points douloureux), sensitive to pressure, which are nearly regularly found in the course of the neuralgically affected nerves, and which are mostly attacked unilaterally, especially at such points at which they pass through bones, muscles, etc. It is easy to demonstrate these areas, not only during the attacks, but also during the painless intervals, if we follow the course of the nerve, examining by making deep pressure. Painful areas are *entirely* absent only in rare cases, according to my observations, and their presence should correctly be considered as an important symptom which supports the diagnosis materially.

Accompanying Sensory Symptoms.—Another subsequent symptom of neuralgia, which may likewise be utilized in the diagnosis, is that the reaction of the sensory nerves in the neuralgic area is changed during the time of the paroxysms and also during the painless period. In this respect either paræsthesias (formication, furry sensation), or hyperæsthesia, respectively *hyperalgia* and *anæsthesia* manifest themselves. The latter sets in especially in long-lasting neuralgia which, then, appears in the type of anæsthesia dolorosa. The pain, at the height of the paroxysm, often radiates from neuralgically affected nerves to adjacent, rarely to remote, nerve areas.

The motor and vaso-motor nerve spheres also show changes in the course of neuralgia. The motor disturbances: Spasms, direct and, particularly, reflex, later paralytic, symptoms, are less constant than the *vaso-motor* disturbances.

Vaso-motor Phenomena.—The symptoms of vaso-motor spasm at the onset of the paroxysm (pallor and coldness of the skin) are more or less pronounced, but very soon the characteristic phenomena of vascular dilatation appear with reddening of the skin and mucous membranes. Probably it is here not so much a question of mainly paralytic effects, but of an irritation of the vaso-dilator nerves. In connection with these symptoms are secretory disturbances of various kinds, such as are quite commonly associated with neuralgia: Marked lacrymation and perspiration with reddened skin, salivation, etc. Some of these secretory alterations are surely due to disturbances of innervation in the vaso-motors, especially in the vaso-dilator nerves, others probably to direct ir[illegible]retory nerves.

Trophic Accompaniments.—We also observe *trophic* disturbances of various kinds in the affected nerve district in the course of neuralgia: Thinning, in other cases thickening, and roughness of the skin, increase or decrease of the panniculus adiposus, discoloration, loss or even increased growth of hair, various exanthems (erythema, urticaria and, above all, *herpes*). The cause of such phenomena is to be looked for, primarily, in the above-mentioned effect upon the vaso-motor nervous system.

The diagnosis of neuralgia can, according to the above statements, usually be made with certainty. However, cases may occur in practice which are liable to confusion of neuralgia with other morbid conditions occurring with violent pains. It remains, therefore, to discuss the *differential diagnosis in detail.*

Differential Diagnosis.—Pains which are produced by *inflammatory affections* can easily be differentiated. They do not occur purely paroxysmally, are not localized strictly upon the course of a single nerve and increase in the same ratio as does the inflammatory pain which, sooner or later, becomes unmistakable by unerring signs (swelling, reddening, exudation, etc.). The differential diagnosis between neuralgia and *rheumatic muscle pains* is more difficult at times. Muscular rheumatism is generally characterized as such by the fact that pains depending upon it are produced by movements—i. e., by contraction of the affected muscle, also by touch. The diagnosis becomes slightly more complicated in muscles the position of which coincides with the distribution of some nerves, as the intercostal nerves, for instance when several intercostal nerves are neuralgically affected. The facts, that the pains in muscular rheumatism are dependent upon movement, especially respiration, that typical painful areas and also the well-known accompanying phenomena of neuralgia are absent, and that the pain is not of a pronouncedly paroxysmal character, aid us to overcome the difficulties of the diagnosis. Similar points are true regarding the differentiation between neuralgia and *articular pains.* Even if the joint affection is associated with radiations of the pain, the pains are concentrated essentially upon the affected joint and are regularly aggravated by active and passive movements of the same. Pains originating in periosteum and bones will scarcely give rise to confusion with neuralgia if an exact local inspection is made and if the marked characteristics of neuralgic pains are remembered in the differential diagnosis. We are most liable to be in doubt in some cases whether neuralgic or *hysterical* pains (hyperalgesia, hyperæsthesia of the skin, myodynia, etc.) prevail in the given case, especially because genuine neuralgias are quite commonly associated with hysteria; it often depends upon the subjective judgment of the physician whether he will designate pains in hysterical patients as actual neuralgia or not. Painfulness of the vertebræ is almost always found in hysteria, as is well known; but sensibility to pressure of the spinous processes of the spinal column is quite a common occurrence in neuralgia of the trunk and of the extremities, so that vertebral pain does not represent an exclusive differentio-diagnostic criterion. It is necessary, by all means, that the often-stated characteristics of neuralgic pains should be well developed so that we should be justified in speaking of neuralgia in hysteria and to forego the diagnosis of hysterical hyperæsthesia, articu-

lar neurosis, etc. The differentiation between neuralgia of the trigeminus from migraine will be exhaustively treated in the discussion of prosopalgia.

Diagnosis of the Seat of a Neuralgia.—After we have become convinced, by observation of the typical symptoms of the affection and by means of the above-described differentio-diagnostic method, that a neuralgia is present in the given case, it remains to ascertain its *seat*. Of course, to determine which nerve or branch of a nerve has been attacked by neuralgia does not present any difficulty to one familiar with the anatomy of the nerves. Another question, much more difficult to decide, is whether a purely *peripheral* or a *central* neuralgia is present. Unfortunately, we are not in possession of such precise characteristics as are at our disposal for the differentiation between peripheral and central paralyses of the motor and, partly, of the sensory nerves. If it is a question of neuralgia of mixed nerves, it may be presumed, generally, if the affection implicates the peripheral course of the nerve, that, besides sensory phenomena of irritation, motor and vaso-motor symptoms may be present accompanying the same. For, as most peripheral nerves contain all three kinds of fibres, sensory, motor and vaso-motor, at the same time, their common irritation usually coincides. The sensory and motor fibres in the central organ occupy tracts more widely separated, hence a separate affection of the sensory nerves is more apt to occur. It is true that here, too, motor-irritative phenomena may occur reflexly, besides those relating to sensation. Still more than by localized symptoms of motor irritation is a peripheral seat of neuralgia indicated by phenomena of motor paralysis which are limited to the distribution of the motor fibres extending in the nerve trunk that is affected neuralgically. In such an instance we must premise, which has become plausible according to other pathological experience, that the motor fibres, which are acknowledged to be less resistant to injurious effects, have already become unable to conduct at the same stage at which the sensory fibres of the same nerve trunk still react to neuralgic irritation. The combination of the affection with vaso-motor and trophic disturbances is less applicable to the differential diagnosis between peripheral and central neuralgias. It is permissible, *in general*, to infer a peripheral seat of neuralgia from a strict limitation of the vaso-motor and trophic disturbances to the neuralgic area, whereas, inversely, it is not permissible to exclude, if extensive vaso-motor and secretory symptoms accompany the affection, the peripheral character of a neuralgia, because *extensive* vaso-motor and secretory accessory symptoms may be present, also, in peripheral neuralgias, as the expression of an irritation of vaso-motor and secretory nerve tracts which takes place reflexly from the periphery.

It follows, from these remarks, that the above-described characteristics are only of a limited value differentio-diagnostically. However, they may serve to give a certain direction to diagnostic calculations. The diagnosis acquires a firm support only upon consideration of other symptoms which accompany neuralgia and which point to a central affection: Phenomena of paralysis and irritation in motor and sensory nerve tracts which are remote from the neuralgic nerve district, simultaneous insufficiency of ves-

ical and rectal functions, ocular findings which may be referred to a central seat of the nervous affection, increasing curvature of the vertebral column, disturbances of the function of some cerebral nerves and of the psychical activity, etc. One of the most frequent forms of neuralgia with a central seat, except the hysterical, is that which accompanies or, as is often the case, introduces tabes dorsalis. The diagnosis of the same as a symptom of locomotor ataxia is, fortunately, facilitated in that, not considering rare exceptions, they are accompanied with another phenomena characteristic of the initial stage of tabes (reflex immobility of the pupil or absence of the patellar reflex), so that the recognition of tabetic neuralgias as such is almost always successfully and easily accomplished. If a neuralgia is only associated with absence of the patellar reflex, it should be remembered that one disease which is also often accompanied with this symptom, viz., diabetes mellitus, is the cause of neuralgia. An examination of the urine for sugar, therefore, is a *conditio sine qua non* for the final diagnosis of the special character of a neuralgia.

Ætiological Diagnosis.—It probably does not require any further explanation, in consideration of the above statements, that the diagnosis of neuralgia is not complete until the *cause* of the affection has also been found in the given case. This ætiological part of the diagnosis is, apart from everything else, of the greatest importance especially because the therapy of the special form of neuralgia present is successfully guided by it. I recommend the following practical method in this respect:

After the character of the nervous affection has been determined to be a neuralgia, we must consider in the first place whether or not it is only a more pronounced symptom of an affection of the central nervous system. If we conclude, in consideration of the above postulates, that this is not the case, the task of the diagnostician is now to look for a noxious influence directly acting upon the nerve which is neuralgically affected. In this respect we have to consider primarily *traumatically* or *mechanically* acting effects, especially as they are objectively demonstrable, contrary to other causes of neuralgia: Foreign bodies which have entered the nerve or its surroundings, traumatisms and their consequences (constricting cicatrices, etc.), affections of the periosteum or of the bones (periostitis, dental caries, etc.), which give rise to pressure upon the passing nerves or upon nerves which pass through the osseous canals, furthermore, inflammatory conditions and neoplasms affecting the nerve itself (neuritis, neuroma), tumours (neoplasms, herniæ, pregnancy, etc.) which exert pressure upon the nerves from the neighbourhood. It is necessary, therefore, never to omit, especially in neuralgia of the lower extremity, exploration *per vaginam* and *per rectum* and examination of the hernial rings.

If nothing is found in this respect to explain the origin of a neuralgia, attention is to be paid to the possibility of an effect of *chemical noxa*, and especially of *infectious substances*, in the first place to the existence of a *malarial infection* which most frequently leads to neuralgia. It is indicated, in a diagnostic respect, so soon as a neuralgia occurs in regularly recurring paroxysms or if the spleen proves to be enlarged, to administer quinine experimentally, to render the diagnosis more positive *ex juvantibus*. It is true, diagnostic conclusions based upon this procedure are not quite reliable, even if all the above-mentioned factors which render the presence of a malarial neuralgia probable, are jointly present in a case, as is proved by the following remarkable instance in my practice:

A woman, thirty years of age, after she had sojourned for several weeks in a district which was suspected to be malarial, became afflicted with supra-orbital neuralgia which manifested itself in paroxysms of violent pain. Her spleen was large, palpable. Quinine acted very favourably at first and promptly aborted the attacks every time, but, in the later course of the affection, ceased to act as well.

Electric treatment was unsatisfactory. Upon examination of the fillings of her teeth by a dentist it was discovered that, in a tooth with ill-fitting plug, dental caries was progressing. The proper dental operation promptly removed the supra-orbital neuralgia permanently, which had been refractory for months!

[It is also good practice in suspected cases of neuralgia supposed to be due to malarial intoxication to examine the blood for the plasmodium.]

Other infectious diseases are incomparably less often the cause of neuralgia, thus small-pox, enteric fever in the first stage, or syphilis. The latter may become an indirect cause of neuralgia in that periostitides, gummata, etc., mechanically injure the nerve. It is another question whether the syphilitic contagion as such may give rise to neuralgia—i. e., whether a neuralgia which is observed in the early stage of syphilis, may be ascribed to syphilitic infection. It is very doubtful to me whether this is ever the case, as I have never seen a single instance of genuine syphiliticoinfectious neuralgia, in spite of the large material in the syphilis clinics which I have directed. Not much better is the certainty of the demonstration that *intoxications* with alcohol or nicotine are in any direct causal connection with neuralgias; this fact is certain only, in my opinion, in intoxications with lead, capper and, possibly, with mercury and iodine. The altered composition of the blood may possibly be connected with the occurrence of neuralgia in nephritis and in individuals who suffer from certain constitutional diseases, especially *gout* and *diabetes*; it is necessary, therefore, in searching for the cause of neuralgia to consider these original affections in the given case.

The relation of "*colds*" to the origin of neuralgia (*rheumatic neuralgias*) is, in my opinion, established beyond any doubt. Even if we are absolutely sceptical regarding the significance of "refrigerations" as an ætiological principle, it would, as I believe, offer violence to the facts if we would simply deny the action of cold as the cause of neuralgia. There are cases, in my experience, in which the sudden effect of icy coldness upon a circumscribed area of the skin undoubtedly places the nerve that passes there, into a neuralgic condition. An example that concerns my own person and which may claim the distinction of an experiment, may serve as a proof of the above contention.

Instance of the Causation of Neuralgia by the Effect of Cold.—No case of neuralgia has ever occurred, as far as I can remember, among the numerous members of my family; I myself suffered, about twenty-three years ago, from a long-lasting neuralgia of the supra-orbital nerve, but neither previously nor afterward have I ever been afflicted with neuralgia. I acquired the affection in the following manner: When setting out on a journey, while in perfect health, I was compelled to drive for about an hour against an unusually strong, icy north wind, in an open carriage. I protected myself in such a manner that I pulled a cape completely over my face, exposing only the smallest portion of the forehead over the left eye, and the latter. During the trip I felt the sharp, cold wind very disagreeably at the exposed portion of the skin; when stepping from the carriage about an hour later, I felt violent pains over the left eye and, from then on, was afflicted with a regular supra-orbital neuralgia which disappeared only gradually after several months.

Finally, it may be mentioned briefly that in some cases no cause can be found, whereas in others it appears that affections of the uterus, ovaries, intestines, etc., may give rise to neuralgias in remote parts of the body ("*reflex neuralgia*"). It is also necessary, in the diagnosis of neuralgia, to consider predisposing factors, especially synchronous *anæmia*, the significance of which in the origin of neuralgia has already been mentioned, and also the constitution and occupation of the patient, as well as preceding bodily and mental overexertions, hereditary conditions, a "neuropathic predisposition" (epilepsy, neurasthenia, psychoses). Simultaneous, marked symptoms of *hysteria* should be especially taken into consideration.

Aided by the above rules, it is, almost without exception, easy to make the diagnosis of neuralgia; and it is no more difficult for a physician who nows the anatomical distribution of the various nerves to determine in ıe given case which nerve is affected by the neuralgia. We might, espe-

cially with reference to the diagrams 14 to 17, inclusive, omit, therefore, to discuss specially neuralgia of the individual nerves, if it were not that, in the course of the same, certain details become predominant in the morbid picture which could not be mentioned in the discussion of the diagnosis of neuralgias in general. But brief diagnostic remarks will suffice to furnish the supplements which are necessary to a diagnosis of neuralgia of an individual nerve.

NEURALGIA OF INDIVIDUAL NERVES

NEURALGIA OF THE TRIGEMINUS NERVE, PROSOPALGIA

Those principles which have been thoroughly expounded in the diagnosis of neuralgias in general, are true in all the details in the most frequent, practically most important neuralgia—*prosopalgia*. Especially the various accompanying symptoms become particularly markedly and characteristically prominent in neuralgias of the trigeminus, viz., radiation of pain, hyperæsthesia and anæsthesia, spasm of the muscles of the face and, especially, also vaso-motor and secretory symptoms: Pallor or usually marked reddening of the skin of the face and of the mucous membranes (of the orinasal mucous membrane and, especially, that of the conjunctiva), changes in the colour of the hair, thickening of the facial skin, etc. But neuralgias of the trigeminus are complicated, above all, in keeping with the course of genuine and reflexly acting secretory fibres in the fifth nerve, by secretory disturbances, such as lacrymation, salivation and, in rare cases, increased secretion of the nasal mucous membrane.

Of course, the clinical picture will vary, according to the trunk or only some branches or twigs of the trigeminus being affected, and the diagnosis should consider these facts primarily. The cardinal points to differentiate between neuralgias of the individual branches of the trifacial nerve, are as follows:

Ophthalmic Neuralgia.—*Neuralgia of the first branch (ophthalmic neuralgia)*: Diffusion of the pain over the entire region of the ophthalmic branch or in some ramifications of the same, most frequently in the supra-orbital nerve, in the region of which the pain is principally located in the forehead up to the vertex, in the adjacent skin of the temples and in the upper eyelid. At the same time, there exists redness of the conjunctiva, increased lacrymation and, which is particularly important diagnostically, an area which is almost constantly painful upon pressure, at the supra-orbital foramen, rarer also in the inner angle of the eye, at the nose, etc. By these symptoms and, eventually, by the sensibility of the nerve in its entire course to external pressure is supra-orbital neuralgia differentiated from paroxysms of pain in migraine and other forms of cephalalgia, which otherwise are liable to be confused with it. Neuralgia is very often due to malarial infection which almost always, if it gives rise to neuralgia, affects in particular the supra-orbital nerve. The effect of cold also manifests itself in the causation of neuralgia particularly of this nerve. Furthermore, affections of the nasal and frontal cavities, indirectly also dental caries, which generally lead to neuralgias of the second and third branches

of the trifacial nerve, may cause supra-orbital neuralgia, which should be well remembered in the diagnosis in consideration of the therapeutic indication (see the above-mentioned case on p. 447).

As to the remainder, the general diagnostic criteria, the presence of accompanying symptoms, etc., are determining especially for the diagnosis of supra-orbital neuralgia.

Neuralgia of the Second Branch (Supramaxillary Neuralgia).—The infra-orbital nerve is that branch of the supramaxillary nerve (the regions of distribution of which it is unnecessary to mention here) which is most frequently attacked by localization of the pain in the lower eyelid, in the cheeks, the lateral region of the nose, etc. The principal area of pain is in the infra-orbital foramen. Of the other neuralgically affected nerve twigs of the second branch I wish to mention especially the superior alveolar nerve, owing to its relation to diseases of the teeth and of the alveolar process of the upper maxillary bone; the sclerotic processes in the toothless alveolar processes of the aged are, among others, also to be considered ætiologico-diagnostically.

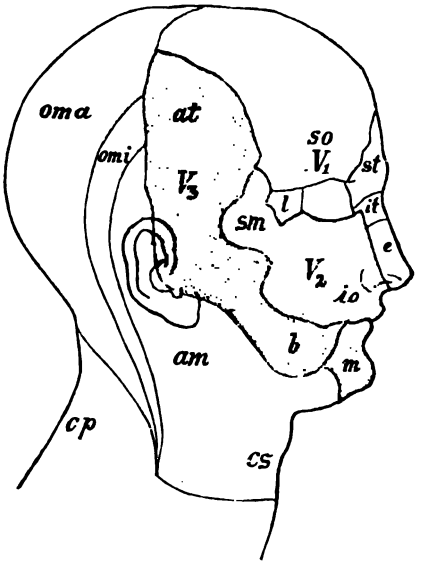


FIG. 18.—DISTRIBUTION OF THE SENSORY CUTANEOUS NERVES IN THE HEAD.

oma and *omi*, Occipitalis major and minor; *am*, Auricularis magnus; *cs*, Superficial cervical; V_1, V_2, V_3, First, second, and third branches of the fifth (V_r); *so*, Supra-orbital; *st*, Supra-trochlear; *it*, Infra-trochlear; *e*, Ethmoidal; *l*, Lacrymal; *sm*, Subcutaneous malæ, or zygomatic; *at*, Auriculo-temporal; *b*, Buccinator; *io*, Infra-orbital nerve; *m*, Mental; *cp*, Posterior branches of the third cervical.

Neuralgia of the Third Branch (Inframaxillary Nerve).—This neuralgia is most frequently restricted to the inferior alveolar nerve, with the painful area at the mental foramen. Isolated neuralgias in the regions of the auriculo-temporal nerve and of the lingual nerve (with pain in one half of the tongue, unilateral coating of the tongue, and salivation) are also observed occasionally. The causes of neuralgias in the third branch are, above all, affections of the teeth of the lower maxillary bone, sometimes also inflammatory processes in the middle ear (minor superficial petrosal nerve which communicates with the tympanic plexus), as well as affections of the base of the skull.

The diagnosis of localization of neuralgia upon individual branches of the trifacial nerve is never difficult, if we recall the distribution of the various nerves (see Fig. 18).

The diagnosis becomes more difficult if a recurrent nerve of the three branches of the trifacial nerve is the seat of neuralgia. It is well known that the recurrent nerves supply the dura mater, respectively the tentorium, with sensory fibres and cause, if neuralgically affected, violently boring, deep-seated pains in the interior of the head.

The question suggests itself in such cases whether we are dealing with the *neuralgia* in question *or with an affection of the brain with symptomatic cephalalgia.*

Differential Diagnosis in Neuralgic Forms of Headache.—Determining in favour of the former condition in such a case is, above all, the demonstration of a synchronous neuralgia in other twigs of the trigeminus with its accompanying phenomena, unilateral headache and paroxysmal occurrence of the same, although the latter symptom can claim only a very subordinate differentio-diagnostic significance in that the absence of any paroxysmal characteristic points against neuralgia. On the other hand, the assumption that cephalalgia in the given case is the expression of an anatomical cerebral affection, is based upon the presence of sensory and motor disturbances, of convulsions and phenomena of paralysis in the extremities (usually of one half of the body) and in the distribution of some cerebral nerves, furthermore upon synchronous alterations of speech and intelligence, upon vertigo, delirium, vomiting, etc. The result of an ophthalmological examination may also give valuable information regarding the differential diagnosis.

Migraine.—*Paroxysms of migraine* also bear a certain similarity to trigeminus neuralgia in that here, also, vaso-motor and secretory symptoms accompany the affection, the pain is unilateral and concentrated upon certain portions of the roof of the skull. But this concentration is usually not restricted to the distribution of a single nerve; pronounced areas painful to pressure are also absent and the intervals between the individual paroxysms of migraine last longer, as do the attacks, than in actual neuralgias, in which vomiting, the usual accompaniment of hemicrania, occurs only exceptionally. However, theoretically we may well assume, in hemicrania, a neuralgic affection of the just-mentioned recurrent nerves of the trigeminus and of other branches of the first branch of the trigeminus, in rare cases also of other nerves of the head, for instance, of the great occipital nerve. This would represent a neuralgia brought about in that, owing to a neurosis of the cervical sympathetic, the filling of the vessels which accompany the sensory nerves would be changed more or less suddenly, thus causing an intense irritation of the respective nerves. If this irritative condition of the nerves differs in its course from that of an ordinary neuralgia, this might be due, in my opinion, to the peculiarity of the ætiological factor, inasmuch as that form of vascular neurosis lasts longer, it is true, yet, with its remission, in contrast to neuralgias of other origin, the reaction of the nerve rapidly becomes normal again, to reassume its neuralgic character only after the lapse of some length of time.

Migraine Ophthalmoplégique.—A special form of migraine is to be mentioned in particular, which is accompanied with paralytic phenomena in the distribution of the oculomotor nerve (*recurring oculomotor paralysis, migraine ophthalmoplégique*). The affection is characterized by unilateral paralysis of the oculomotor nerve occurring in paroxysms, which is ushered in by headache and vomiting. The paralysis sometimes affects all, at other times only some, of the branches of the oculomotor nerve; the internal branches especially may be exempt. The paralysis, the condition which follows migraine, may, in typical cases, fully recede in the course of a few days or weeks, to return with a new attack of headache and vomiting after a shorter or longer interval; remnants of paralysis rarely persist during the free intervals. The diagnosis of the affection is not difficult; it is necessary only to differentiate it from certain cases of paralysis of the oculomotor nerve which occur, in the course of progressive, organic disease of the brain, as a symptom of the latter, and which take place, exceptionally, not continuously-progressively, but in intervals.

NEURALGIA OF THE CERVICAL NERVES

In keeping with the variation in the anatomical condition of the four superior and four inferior cervical nerves, we distinguish in neuralgias which affect the cervical nerves, a cervico-occipital and a cervico-brachial neuralgia.

Occipital Neuralgia.—*Cervico-occipital neuralgia.*—Of the various branches which originate in the superior cervical nerves, it is most frequently the major occipital nerve which is neuralgically affected, whereas isolated neuralgias of the superficial

nerves which originate in the cervical plexus (of the minor occipital nerve, of the major auricular nerve, the supraclavicular nerves, etc.) are observed only rarely. The place of exit of the major occipital nerve is the principal painful area of occipital neuralgia, which, in my experience, is not of a very rare occurrence; the pain radiates from the most superior region of the neck along the posterior part of the head up to the vertex. The neck is kept stiff, partly on account of a simultaneous spasm of the cervical muscles, partly on account of a more voluntary contraction of the same to straighten the vertebral column, thus avoiding attacks of pain which take place due to movements of the cervical vertebral column. The latter should, under all circumstances, be examined for caries, syphilitic affections of the bones, tumours, etc., and the presence of spinal meningitis, affections of the ear, etc., should be taken into consideration. A regular migraine, with intervals of weeks, may, as I have recently seen, regularly occur in the form of a headache restricted to the occipital region, a condition which may be explained in the manner already mentioned.

Phrenic Neuralgia.—Neuralgia of a branch of the cervical ramifications, which is not enumerated as yet, that of the *phrenic nerve*, should be especially mentioned. It has been assumed that this nerve, besides its motor fibres, also conveys sensory fibres, and that the latter, eventually, especially in the left half of the body, may become neuralgically affected. Violent pains at the lower aperture of the thorax, radiating to shoulder, neck and extremities, increase of the pains by pressure upon the diaphragm from the cardiac region and by pressure upon the trunk of the phrenic nerve, furthermore disturbances of respiration with painful sneezing, cough, etc., are said to characterize *phrenic neuralgia*. I do not mean to dispute the possibility of such neuralgias affecting the distribution of the phrenic nerve, especially as I have seen cases which admit of such an explanation. However, a satisfactory certainty of diagnosis regarding this form of neuralgia will scarcely ever be attained, and it is preferable, therefore, to leave it in suspense, in the meanwhile, whether the presence of a phrenic neuralgia may actually be assumed in the given case.

Cervico-brachial Neuralgia.—It is rare that a single brachial nerve is the seat of a neuralgia; usually several branches of the brachial plexus are attacked simultaneously. Likewise does this neuralgia but very rarely occur bilaterally, unless it is brought about by a compression of both plexuses by a tumour occupying the median line of the neck or that it forms a symptom of a central affection of the spinal cord or of a general intoxication, as I have seen in case of lead-poisoning, for instance. Of course, traumatism often plays an important part in the ætiology of this form of neuralgia in particular. The pain is concentrated upon the distributionary region sometimes of one, at other times of another, nerve; the painful area, therefore, varies accordingly: In the brachial plexus proper, at the turning point of the radial nerve in the upper arm, at the exposed area of the ulnar nerve between the internal condyle and olecranon, etc. Cutaneous affections such as herpes, pemphigus, urticaria, etc., are quite usual as accompanying symptoms, and also, owing to the mixed character of the brachial nerves, as a matter of course, an altered reaction of the motor nerves: Spasms, paralyses, muscular atrophy. A radiating variety of brachial neuralgia are the neuralgic pains which occur in the left arm in the course of angina pectoris.

INTERCOSTAL NEURALGIA

The diagnosis of this frequent neuralgia is easy in most cases, especially so long as it affects the middle intercostal nerves, as is usually the case. However, the affection sometimes also concerns the lower intercostal nerves which are distributed in the skin of the lateral and anterior abdominal wall. The diagnosis is slightly more difficult in the latter case, because several abdominal affections that are accompanied with pains may be confused with it. Characteristic of intercostal neuralgia is, above all, the direction of the course of the pains and the situation of the painful areas

near the vertebral column, in the axillary line and in the median line of the thoracic and abdominal walls. Important as accompaniment of the affection is also an eruption of herpes zoster, which, however, supervenes upon neuralgia much rarer than is usually supposed.

Differential Diagnosis.—The differential diagnosis is to consider, principally, rheumatic affections of the thoracic and abdominal muscles, peritonitis and dry pleurisy, gastralgia and sometimes also gastric ulcer. *Muscular rheumatism* differs from intercostal neuralgia principally in that, in the former, not only more intense respiratory movements, which may increase the pains in intercostal neuralgia also, but, essentially, movements of the trunk and arms call forth the pains, that pressure upon the muscular fasciculi of the pectoralis, rectus abdominis, etc., which are grasped between the fingers, is all over painfully felt by the patient, and that, after the action of a powerful induction current—i. e., after the affected muscle has remained in marked contraction for some time, the pains almost always improve considerably. *Peritonitis* and *dry pleurisy* more rarely give rise to mistakes upon closer inspection. The best manner to arrive at a correct conclusion in the differential diagnosis is to bear in mind the presence of areas painful to pressure, which are characteristic of neuralgia, the independence of the paroxysm of pain to respiration, and the often prompt action of the anode upon the intensity of the pain. As usually also the skin of neuralgically affected areas is painful to pressure, the demonstration of an intense sensibility of a portion of skin grasped in a fold may aid to overcome the difficulties of the diagnosis. Of course, all doubts disappear if, on the other hand, a distinct friction sound, the gradual occurrence of an exudate and other symptoms of pleuritis or peritonitis secure the presence of the latter affections. The differential diagnosis between *gastralgia* and intercostal neuralgia is more difficult if the pains which are caused by the latter disease are limited to the epigastrium. If, in such intercostal neuralgias, the pains are independent of eating, if, on palpation of the inferior intercostal spaces, one of the latter is sensitive to pressure at one of the usual painful areas, and if, upon electrization of the affected intercostal space, the pain in the epigastrium disappears, the presence of an intercostal neuralgia is certain. The differentiation of the latter from *gastric ulcer* is easier, as the paroxysms of pain in this affection are most markedly dependent upon the posture of the patient, upon commotion of the stomach and upon ingestion of food, whereas these factors are of minor consideration in the morbid picture of intercostal neuralgia. A confusion of intercostal neuralgia with gall-stone colic is not very apt to occur. The pain in the latter is much more violent, recurs less often and regularly, ceases more suddenly and is often associated with vomiting or singultus; jaundice is usually present, and palpation of the liver, especially of its border, causes pain. The diagnosis of intercostal neuralgia may be supported by an observation of the *ætiology*; affections of the vertebræ and of the ribs, aortic aneurysms and, above all, also spinal-cord diseases (especially meningitic processes, tabes, etc.) are the most frequent causes of intercostal neuralgia, unless it occurs idiopathically, which happens quite often.

Mastodynia.—As a variety of intercostal neuralgia may be mentioned *mastodynia*, a neuralgic affection of the nipples, especially of the female, a neuralgia which is sometimes connected with the development of neuromata, small, hard, mostly very painful nodules in the mammary tissue. The diagnosis may present some difficulties in such a case, because it is often scarcely possible under such circumstances to differentiate a benign mastodynia from incipient malignant neoplasms with irritation of the sensory nerves of the nipple and the skin over the same. The steady growth of the malignant tumours, the secondary infiltration of the adjacent lymph glands, the severe impairment of the constitution, the concentration of the pains upon that area of the mamma in which the tumour is located, are in favour of the development of a neoplasm in the mamma, while, on the other hand, the paroxysmal character of the pains, the change in the painfulness of the tubercula tuberosa, the sensibility of the respective spinous processes and the occurrence of herpes zoster secure the diagnosis of mastodynia and also permit of an easy differentiation from mastitis.

NEURALGIA OF THE LUMBAR NERVES

The lumbar plexus is the origin of six branches, three of which, the ilio-hypogastric, the ilio-inguinal and the genito-crural nerves (external spermatic and lumbo-inguinal), supply principally the lower portion of the abdominal wall and the pubic region, while the other three, the external femoral, the obturator and the crural nerves, mainly supply the anterior surface of the lower extremity with cutaneous nerves. They may, therefore, become seats of neuralgias the precise diagnosis of which presupposes the knowledge of the special mode of distribution of these nerves. The two figures, numbers 19 and 20, which depict the distribution of the lumbar nerves, may facilitate the diagnosis in the given case.

Some details which concern the diagnosis of neuralgia of the lumbar nerves, may be especially emphasized: A neuralgic affection of the ilio-hypogastric, ilio-inguinal and genito-crural nerves is usually comprised in the common designation: *Lumbo-abdominal neuralgia.* The pain in this affection is concentrated upon the lumbar and gluteal and hypogastric regions and within reach of the genitalia. A small portion of the skin of the anterior surface of the femur is also affected in this form of neuralgia, viz., the distributionary region of the lumbo-inguinal nerve. There are severe painful areas in lumbo-abdominal neuralgia. Near the lumbar portion of the vertebral column, in the centre of the crest of the ileum, in the hypogastric region over the symphysis and at the scrotum, respectively at the labium majus and at the vaginal vault.

In *neuralgia of the external cutaneous or of the cutaneous (internal or middle)* nerves the pain is localized in the external lateral (anterior and principally posterior) surface of the femur down to the knee. The district of distribution of the nerve borders, at the posterior surface of the femur, immediately upon that of the posterior cutaneous nerve, a branch of the sciatic plexus, so that neuralgias of the latter are very liable to be confused with the neuralgias in question. The principal painful area is immediately near the anterior superior spine of the ilium at which the nerve trunk passes over the border of the pelvis.

Neuralgia of the crural nerve (respectively of its branches: Cutaneous (median and internal) and the saphenous or major saphenous nerves) is characterized by pains in the central portion of the anterior surface of the femur and the anterior region of the knee, but, above all, also by a diffusion of the pain, which corresponds to the course of the saphenous nerve. The pain remains at the inner side of the knee joint and of the calf and extends along the inner anterior surface of the leg past the interior ankle to the inner border of the dorsal surface of the foot as far as to the big toe. The pains are intensified by movements of the leg; some areas in the course of the nerve are especially sensitive; the area below Poupart's ligament, at which the nerve passes the border of the pelvis, furthermore the place at which the saphenous nerve penetrates the fascia at the inner surface of the knee joint and, finally, areas in front of the internal malleolus and at the inner ankle and at the inner border of the big toe (see Fig. 19).

Obturator Neuralgia.—Finally, the sixth nerve branch of the lumbar plexus, the obturator nerve, is also sometimes attacked by an isolated neuralgia (obturator neuralgia). The cutaneous nerve to be considered in this affection extends along the inner surface of the lower two thirds of the femur down to the knee. This very rare

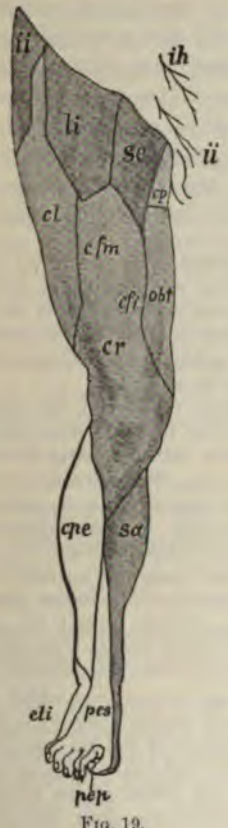


Fig. 19.

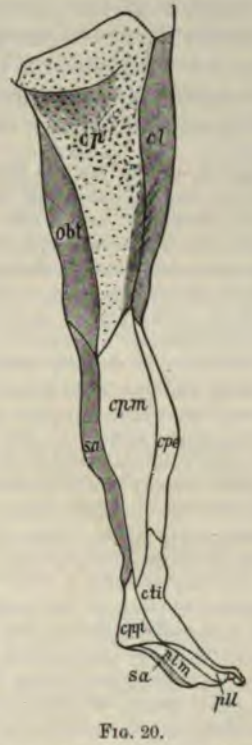


Fig. 20.

Figs. 19 and 20.—Distribution of the Sensory Cutaneous Nerves of the Anterior and Posterior Surfaces of the Lower Extremity.

Lumbar Nerves: 1, *ih*, Ileo-hypogastric nerve; 2, *ii*, Ileo-inguinal nerve; 3, Genito-crural nerve; *sc*, External spermatic nerve; *li*, Lumbo-inguinal nerve; 4, *cl*, External cutaneous nerve of the thigh; 5, *cr*, Crural nerve; *cfm*, Middle cutaneous nerve of the thigh; *cfi*, Internal cutaneous nerve of the thigh; *sa*, Saphenous nerve; 6, *obt*, Obturator nerve.

Sacral Nerves: 1, *cp*, Posterior cutaneous femoral nerve; 2, Sciatic nerve.

Peroneus: *cpm*, Medial posterior cutaneous nerve of the thigh; *cpe*, Peroneal communicating nerve; *pep*, Anterior tibial nerve; *pes*, Musculo-cutaneous nerve of the leg.

Tibialis: *cti*, Tibial communicating nerve; *cpp*, Cutaneus plantaris proprius; *plm*, Internal plantar nerve; *pll*, External plantar nerve.

form of neuralgia is of diagnostic importance in so far as, upon incarceration of an obturator hernia pains and formication occur in the region of the cutaneous nerve of the obturator nerve; at the same time it may be that a paresis of the adductors of the femur (which are supplied with motor fibres by the obturator nerve before it penetrates the fascia lata) becomes manifest in this condition.

NEURALGIA OF THE SACRAL NERVES—NEURALGIA OF THE SCIATIC PLEXUS—SCIATICA

The sciatic plexus, the principal part of the sacral plexus (formed of the fifth lumbar and the I–V sacral nerves is the point of origin, besides of the superior and inferior gluteal nerves which enter the gluteus muscle, of two nerves which, to the greatest extent, supply the skin of the posterior surface of the femur: The posterior cutaneous femoral nerve and the sciatic nerve. Neuralgias which occur in the course of the last two nerves are comprised in the general designation of *sciatica.* Their diagnosis requires a more detailed discussion, because sciatica represents, next to trigeminal neuralgia, the most frequent form of neuralgia.

Distribution of the Nerves from the Sciatic Plexus.—The posterior cutaneous femoral nerve, passing out below the gluteus maximus, supplies nerve twigs to the gluteal skin; anteriorly, nerves into the perineal skin, the posterior parts of the external genitalia and the most superior part of the inner surface of the femur; inferiorly, cutaneous branches to the posterior surface of the femur down to the knee, respectively to the middle part of the calf (see Figs. 19 and 20).

The *sciatic nerve* proper passes between the tuber ischii and the greater trochanter to the posterior surface of the femur, descending in its median line. In the centre of the femur or slightly above, the nerve divides into two large branches, the *peroneal nerve* and the internally situated *tibial nerve.* The peroneal nerve turns outward and downward and then sends forth three branches, the n. articul. genu to the capsule of the knee joint, the n. cut. cruris post. ext. or communicans peroneus to the skin of the lateral border of the leg, and the n. cutaneus cruris post. medius, which supplies sensory fibres to the median part of the posterior surface of the leg. Both nerves (n. cpe and cpm, Figs. 19 and 20) usually end in the region of the ankle. After sending forth these twigs, the peroneal nerve extends behind the capitulum of the fibula downward and forward and divides into a deep branch (nervus peroneus profundus, which, preferably a motor nerve, only supplies a few small sensory branches to the posterior surface of the borders of the first and second toes which face each other) and a superficial branch, the superficial peroneal nerve. The latter perforates the fascia cruris in the lower third of the anterior surface of the leg and descends to the dorsum of the foot, innervating the skin of the same and that of the dorsum of the toes. The *tibial nerve* continues in the direction of the trunk of the sciatic nerve; in the popliteal space it sends forth the nervus articulares genu and the nervus communicans tibialis or suralis, which extends downward on the posterior surface of the gastrocnemius and perforates the fascia in the lower third of the leg; it is here situated at the outer border of the tendo Achillis, communicates with the communicans peroneus and extends at the outer border of the foot to the end phalanx of the fifth toe. After the tibial nerve has extended under the muscles of the calf on the posterior surface of the leg to the inner surface of the ankle, it passes behind the latter, sending forth a few thin twigs to the skin of the heel and of the posterior end of the *sole of the foot* (nervus cutaneus plantaris proprius or cutaneus plantæ), and now divides into its two final twigs, the *nervus plantaris int.* (or medialis) and *ext.* (or lateralis). Of the latter two, the internus supplies the plantar surface of the first to fourth toes, and the externus that of the fourth and fifth toes. Figs. 19 and 20 depict the distribution of the sciatic nerve in such a manner that the not shaded portions of the lower extremity correspond to the same.

Symptoms of Sciatica.—Attention is to be paid to the above-described distribution of the sciatic nerve, in the diagnosis of sciatica, especially to the fact that the sciatic nerve (including the nervus cutaneus fem. post.) supplies the skin of the ***posterior surface of the femur*** (except its lateral

borders which are supplied by lumbar nerves, viz., the cut. lateralis and obturatorius) and of *the entire leg and foot, with the sole exception of the distributionary region of the saphenous nerve* (from the crural nerve), which embraces the inner surface of the leg (anterior and posterior), and of the foot the inner border of the big toe (see Figs. 19 and 20). The skin is, usually, the only seat of the pain, rarely the muscles which are supplied by the sciatic branches; it is remarkable that actual paroxysms are not as marked as in other neuralgias, especially in trigeminal neuralgia. The pains are intensified principally at night and upon movements of the lower extremities, especially *upon lifting of the stretched-out leg,* furthermore upon bending, straining during defecation, etc. Sometimes the entire distributionary region of the sciatic nerve is neuralgically affected, at other times only the district of the nervous cutan. fem. posterior, at other times again, that of one of the two main branches of the sciatic nerve, most frequently that of the peroneal nerve. Special painful areas are: At the exit of the nerve from the pelvis, corresponding to the greater sciatic incisure, at the lower border of the gluteus maximus, in the bend of the knee (tibial muscle), behind the capitulum fibulæ (peroneal nerve), furthermore, behind the external (nervus communicans tibialis-peroneus) and internal ankle (tibial nerve), and, finally, some areas on the dorsum of the foot (nervus peroneous superfic.). It is important diagnostically that the pain often radiates into the region of the sciatic nerve of the opposite side, and that motor disturbances, spasmodic muscle contractions (during the paroxysm), on the one hand, and slight paresis and stiffness in the affected leg, on the other, usually accompany this form of neuralgia.

In some cases a characteristic, abnormal attitude of the trunk develops in patients suffering from sciatica, especially noticeable when walking (*sciatic scoliosis*).

Differential Diagnosis.—The body is bent towards the healthy side and the convexity of the vertebral column bent out towards this side (heterologous form of scoliosis), an inclination of the trunk towards the affected side is rarer (homologous form), still more so a change in the attitude of the trunk so that the homologous and the heterologous forms of scoliosis alternate ("alternating" sciatic scoliosis). The cause of these curvatures is probably due to unilateral reflex contractures of the muscles of the back, especially of the sacro-lumbar muscle. There are also other attitudes of the body observed at times, which are caused by sciatica, thus an extreme bending forward of the upper part of the body, etc. The pain and the altered attitude of the body cause the walk of patients with sciatica to become altered, it is shorter, halting, asymmetrical. Placed on the floor, the patients are able to raise themselves only with the aid of the hands, and this is done, in unilateral sciatica in such a manner that the patient "at first places the hands behind his back, then pushes the pelvis and the flexed legs backward between the arms, and now slowly raises the trunk, straightening the knees and pushing himself off the floor with one hand, while the other is balanced in the air" (Minor). This manner of rising from a sitting posture, if found constantly in sciatic patients, would be important not only in the diagnosis of sciatica, but also to detect simulation of the condition.

A wrong diagnosis is prevented best if we bear in mind that the localization of the pain in sciatic neuralgia (outside of radiations) must correspond strictly to the anatomical course of the nerve. Mistakes are not exactly rare, as sciatica is confused with other painful affections in the lower

extremities, thus especially *coxitis* and, above all, ***nervous coxalgia.*** ***Coxitis*** can create the impression of sciatica only upon superficial examination. But the pains in coxitis, even if they radiate, are principally concentrated upon the hip joint; special attention should be paid to the swelling of the joint region, to the intense pain upon percussion of the trochanter, to the abnormal posture, eventually shortening, of the leg, to the regular increase of the pains upon movement of the ends of the joint and to the simultaneous presence of fever, although the latter is by no means constant in coxitis. The differential diagnosis is sometimes more difficult in ***nervous coxalgia*** which occurs principally in hysteric patients as a joint neurosis. Here, also, the same as in coxitis, in contrast to sciatica, the pains in the region of the joint are especially marked, but often more intense than upon approximation of the ends of the joint; it is also possible to determine that the principal seat of the pain is rather in the soft parts surrounding the joint than in the latter. The rapid change in the intensity of the pain, the determination of synchronous phenomena of hysteria leads to the diagnosis of the latter as a basis of the affection; the concentration of the pains upon the region of the joint serves to differentiate hysteric coxalgia from sciatica, which, generally, does not occur often in hysteria. *Inflammatory*, especially *rheumatic muscular affections* may also have to be considered in the diagnosis of sciatica. The differentiation, even if radiating pains complicate the clinical picture in these muscular affections, is not difficult if we recall that, in rheumatism, usually only isolated muscles are affected, that, therefore, only a certain kind of movement causes pain; nor is the course of the nerves sensitive to the touch, but especially the spanning and pressing of the muscle masses. Inflammation and abscess formation in the muscles is, besides, accompanied with swelling of the inflamed parts, fever, etc.

If the above affections can be excluded in the given case, the diagnosis is limited to sciatica; it becomes the more certain, the more exact the affection follows the tract of the nerve and, furthermore, the more we are able to ascertain the painful areas, previously mentioned, and, also, if accompanying symptoms, such as spasms and vaso-motor disturbances, are present.

Ætiological Diagnosis.—But in no other neuralgia is it less permissible to be satisfied with the simple diagnosis of neuralgia without having made the attempt, at the same time, to ascertain the *cause* of the affection. It is possible, in the majority of cases, to discover the ætiological basis of sciatica. Predisposition plays a decidedly smaller part in this neuralgia than in others; anæmia, hysteria and nervousness are only subordinate factors in the origin of sciatica, whereas locally acting, harmful effects, in most cases, solely determine the causation of the neuralgia, owing to the exposed position of the sciatic plexus and of the nerves arising therefrom. The *rule should be strictly adhered to, not to make a diagnosis of sciatica at all until an exploration of the rectum has been made.* On this occasion we should satisfy ourselves whether tumours of the pelvic bones, carcinomata of the rectum, a gravid uterus, displacement and tumours of the uterus, ovarian tumours, etc., are present. According to size and position, such tumours, by pressure upon the plexus, may be the direct cause of sciatica or, at least, give rise to it indirectly in that hard fæces have accumulated over a tumour, which give way posteriorly and cause a permanent compression of the nerves. A remarkable instance of my practice may illustrate this:

The patient, in the full vigour of life, of robust constitution, presenting no signs of cachexia, was attacked by a violent sciatica, apparently without cause. A digital examination of the rectum revealed a carcinoma, the high seat of which eventually explained the origin of the neuralgic affection. As the patient, at the same time, suffered from constipation, and as it was probable that the fæces which had accumulated over the stenosis, increased the pressure upon the sciatic plexus, systematic lavage of the rectum was ordered by means of a rectal tube passed through the stricture. The success of this therapy was instantaneous. The application of irrigations caused the sciatica to disappear at once without leaving a trace of pain; the patient felt entirely well and able to work, and did not understand that he had not entirely recovered. The sciatic affection did not recur, although the inoperable carcinoma grew and cachexia which had been absent so far, appeared, to its fullest extent, shortly afterward.

The time-honoured rule to commence the treatment of sciatica with a purge, was dictated by experience, in that not only in stenosing pelvic tumours, but also in other locally acting disturbances, the neuralgic affection of the nerves may suffer an increase if firm fæcal masses press against the promontory and against the sacrum. Even a congestion of the venous sacral plexus and of the common pudic vein as such seems to suffice to irritate the sacral nerves to such an extent that either sciatica is caused by it or, in some cases, at least a predisposition to this affection is created. For this reason, in examining an individual case of sciatica, the possibility of venous stasis in the pelvis and, as an expression of this, the occurrence of *hæmorrhoids* must be noted. The proof of the dependence of sciatica upon direct action of the nerves is even simpler, if transitory pressure upon the sacral plexus in the course of a severe labour can be shown, or fracture of bones of the sacrum takes place, or if in the course of the nerves of the leg trauma, cicatrices, etc., may be noted. Those cases of sciatica are also of mechanical origin in which *bending of bone, neoplasm, periostitis* or *caries*, particularly of the lumbar vertebræ, or of the sacrum have occurred and irritate the sacral plexus, or in which the nerve roots which find exit here are implicated and irritated as the result of a *spinal meningitis*.

Only if these direct proofs of the cause of sciatica are absent and on the other hand the anamnesis shows that a marked *action of cold* has influenced the nerves, or a strong *compression* of the nerve has taken place, for example due to long sitting upon a sharp edge, by a fall upon the buttock, by a prolonged horseback ride, etc., the cause of the origin may be referred to this. In long-continued sciatica the examination of the urine should never be neglected, as *diabetes mellitus* is known to give rise to sciatica and on the other hand a neuralgic affection of the sciatic nerve may cause the appearance of sugar in the urine. The last-mentioned connection between sciatica and glycosuria is on the whole rare, but has unquestionably been noted (by myself) and finds its explanation in the physiologico-experimental fact that as a result of cutting of the sciatic nerve in the animal, melituria arises. Sciatica may also develop as the result of *gout, syphilis, gonorrhœa*, etc.

Central Varieties of Sciatica.—Besides these peripheral varieties of sciatica, *central* forms also occur—i. e., cases in which sciatica is a symptom of *disease of the central nervous system*.

This is especially the case in *tabes dorsalis*, which, as is well known, very frequently begins with attacks of sciatic pain. If there are simultaneously present the other well-known initial symptoms of tabes: The reflex small pupil and above all the absence of the patella tendon reflex, the neuralgic pains especially if they affect both lower extremities, are to be looked upon with certainty as symptoms of an incipient tabes. That sciatica may also occur as a part phenomenon of a cerebral affection, I will not deny; an example which would prove this, has as yet not occurred to me.

Neuralgiæ Pudendo-coccygeæ.—*Neuralgias in the region of the genital organs of the breech and their nerve branches.* In describing the mode of distribution of the lumbar nerves (especially of the ilio-inguinal and of the external spermatic nerves) as well as of the nerves originating in the sciatic plexus (especially of the posterior cutaneous femoral nerve), the supply of the external genitals and the bowel with branches of these nerves was already mentioned; in keeping with this in the course of lumbar abdominal neuralgias and sciatica, genital neuralgias may occur as an accompanying affection. The latter are, however, occasionally the result of an isolated neuralgia of the pudendal plexus and its branches. As is well known the sacral plexus has three divisions: The sciatic plexus, the pudendal plexus, and the coccygeal plexus. From the latter cutaneous nerves arise which cover the skin of the coccyx and the region of the anus, distributing themselves from this point; from the *pudendal plexus* the *hæmorrhoidal inferior nerves* with their branches originate, supplying the skin and immediate vicinity of the anus and the common pudendal nerve with an upper and lower branch from which the former (perineal nerve) supplies the skin of the perinæum, the posterior part of the scrotum or the vulva, the latter (the dorsal nerve of the penis, respectively clitoris) the surface of the penis or the clitoris. In neuralgic affections of these nervous branches there arise anal, perineal and scrotal neuralgia, neuralgias of the glans penis, etc., which may be anatomically localized to certain nervous branches in individual cases (under other circumstances also by the proof of painful points upon the perinæum, etc.).

Coccygodynia.—If the coccygeal plexus is the seat of an isolated neuralgia the designation *coccygodynia* is chosen. It is characterized by severe pain in the region of the coccyx, especially marked in rising from a sitting posture, by pressure, upon urination and especially in defecation as well as by being exaggerated in consequence of coitus. Causes are trauma, inflammatory processes, etc., which may affect the coccyx, severe cold and as it appears in the male sex (in whom this affection has lately also been observed) abnormal irritations in the sexual sphere. Pressure upon the coccyx and passive movements of the same produce the attacks; the diagnosis in cases in which the pains are exactly limited to the coccygeal region offers no difficulties.

In connection with neuralgias of the peripheral nerve, *arthritic neuralgia* and *cephalalgia* shall be briefly mentioned.

Neuralgia of the Joints.—One of the most frequent of *arthritic neuralgias*, nervous coxalgia, has already been considered in a differentio-diagnostic respect in the diagnosis of sciatica. The general diagnostic points of view which were especially emphasized are also *ceteris paribus* prominent in the affection of neuralgias of other joints. The knee joint is most frequently affected, after this the hip joint and much more rarely are the other joints implicated. In general we must confine ourselves to the rule only to diagnosticate arthritic neuralgias by exclusion—i. e., only to suspect them provided the arthritic pains can with certainty be determined not to be due to a demonstrable anatomical change of the joint, and especially if their intensity is in an unmistakable disproportion to the local changes in the joint and if the character of the pain has the type of neuralgia (paroxysmal exacerbations, etc.). The diagnosis becomes the more certain if there are present besides the pain, muscular spasm, hyperæsthesia or anæsthesia of the skin and vaso-motor disturbances and if the affection occurs in individuals who are unquestionably predisposed to nervous hysterical attacks.

In individual cases the neuralgically affected joint appears to be permanently painful, but neither swollen nor continuously reddened; the skin over the joint shows itself painful to the touch, usually more so than the joint itself, even if deep pressure be made upon it. Besides this, some individual points in the region of the joint are conspicuous by being especially painful, as for instance in the case of neuralgia of the hip joint, a point between the trochanter and the tuberosity of the ischium; in neuralgia of the knee joint, a point between the internal condyle of the femur and the head of the fibula, etc. In testing the movability of the joint it may occur that the concomitant reflex contraction of the latter allows the joint to appear as if ankylosis had taken place; an examination however under the influence of an anæsthetic will very rapidly clear the situation in reference to the normal anatomical and func-

tional condition of the joint. As characteristic, there must be still especially mentioned the dependence of the arthritic neuralgia upon psychical influences, under the action of which they often suddenly come and go. Likewise the appearance of another disease frequently causes the neuralgia to disappear rapidly; I saw in a patient suffering from neuralgia of the knee joint a spontaneous disappearance and an immediate reappearance before and after an attack of angina.

Cephalalgia.—*Cephalalgia, cephalœa, headache.* As in the diagnosis of arthritic neuralgia the observance of the method of exclusion is the most certain to guard us from error, so is this also true in the diagnosis of *cephalalgia.* Do not if at all possible content yourself with a diagnosis of "headache" in which the origin cannot be explained. Primarily there must be decided in the individual case, whether we are dealing with a *trigeminus* or *occipital neuralgia* or with *migraine* (whether it be the usual well-characterized variety or one arising as a prosopalgia or as an occipital neuralgia) the diagnosis of which has already been referred to (see p. 451). If these affections can be excluded, search should be made whether or not pathological processes in the *bones of the skull* or their immediate vicinity are present and by mechanical irritation of the nerve or from changed circulatory conditions headache arises. In this connection we must regard: Catarrh of the cavities of the frontal bone, inflammation of the skin of the head, caries and periostitis, syphilitic affections of the bones of the skull, etc. If these conditions are absent, *diseases of the brain and meninges* are to be considered, which as is well known give rise to severe headache. Conclusive, above all, is that besides the latter also other symptoms of cerebral affection are present: Vomiting, delirium, headache paralysis, convulsions, rigidity of the neck, etc. It must be especially emphasized that headache in the majority of cerebral tumours is the most prominent and frequently for a long time the only symptom of the affection. The ophthalmoscopic examination which in a case of headache which is not ætiologically clear dare never be neglected, frequently gives the most certain proof of the dependence of headache upon cerebral diseases, especially if a choked disk can be determined, due to a tumour of the brain. In passing, it may be mentioned that glaucoma as well as anomalies of refraction and insufficiency of the internal rectus muscle not rarely are accompanied by decided *frontal headache.*

Only after this briefly outlined foundation of the diagnosis of the origin of headache as a symptom of the previously mentioned affection is absent, dare a more *individual* importance be placed upon headache. It is then usually due to transitory *circulatory* or *blood-pressure changes in the brain,* or to *poisoning* or *intoxication* (nicotine, alcohol, lead-poisoning, uræmia, carbonic acid, etc.) or to *infections* (typhoid, etc.) in which case the various toxines produced during an infectious disease act as irritants upon the nerves. As from the various organs in general, pains may radiate to distant parts of the body, so also "reflex" headache may occur, for example in the course of diseases of the stomach and intestines and also as a resulting condition ("sympathetic" headache). Head has lately attempted to determine whether the "reflex" headaches occurring in disease of the organs showed distinct areas of localization upon the skull.

This in fact appears to be true to a certain extent. However, very many clinical observations are still necessary to determine precise diagnostic points of support in this respect. Finally, in individuals of an *hysterical* and *anæmic* temperament and in *neurasthenics* headache is a very common symptom. Almost always the diagnosis "idiopathic" "sympathetic" "hysterical" or similar varieties of headache (the ætiology of which is determined by experience, but the nature of which is by no means certain) indicates nothing more than silently admitting that in the special instance nothing certain is known of the origin and the course of the cephalalgia. The headaches complained of by patients very frequently constitute a vera crucis for the diagnostician, in that their origin, in spite of the most exact analysis of the affection, remains an insolvable diagnostic enigma.

DIAGNOSIS OF DISEASES OF THE MOTOR NERVES

THE pathological reaction of motor nerves shows itself, in keeping with those of the sensory nerves, in an increase or diminution of irritability, as *spasm* or as *paralysis*. The diagnosis of these disturbances of the reaction of the peripheral motor nerve has, in contrast to the diagnosis of diseases of the sensory nerve, which has just been considered, a definite foundation, as the former is supported by phenomena which are of a more objective nature. On the other hand, the details often develop marked difficulties, so that an explicit consideration of the individual affections of the motor nerve, especially of paralysis, is necessary.

PARALYSES OF THE PERIPHERAL MOTOR NERVES

The phenomena which are to guide us in the diagnosis of paralysis of the motor nerves have already been mentioned in general, so that we may proceed at once to a diagnosis of the special varieties of paralysis.

Among the motor cranial nerves, the *nerves supplying the muscles of the eye,* the *motor portions of the trigeminus, the facial, the spinal accessory* and the *hypoglossal* nerves are the ones in which the paralysis shows well-characterized pathological conditions. Of these paralyses, those affecting the *muscles of the eye* belong to the realm of ophthalmology. However, they are so frequently connected with the various diseases, belonging to internal medicine, that at least a brief description of the main points which are of importance in the diagnosis of paralyses of the individual nerves supplying the muscles of the eye, seems advisable.

Paralysis of the Oculo-Motor Nerve.—As the oculo-motor nerve supplies the levator palpebræ superioris, the superior, inferior and internal rectus muscles, further the inferior oblique, and at the same time contains fibres for accommodation and for the sphincter of the iris, the picture of a complete paralysis is as follows: The upper lid hangs down over the eye without showing folds (ptosis) and cannot be raised; the movements of the eye internally and upward are absent and the between positions cannot be assumed. The movement of the eye downward is markedly impeded and only occurs due to the action of the superior oblique, whereas the movement of the eye outward is naturally retained. The position of the affected eye is directed outward and downward (strabismus paralyticus divergens et deorsum vergens). If the upper lid is raised, double pictures are noted. The pupil appears to be medium wide and rigid; accommodation is sus-

pended. Modifications of this described finding occur if the oculo-motor in its individual branches is affected or is incompletely paralyzed: Therefore, in paralysis of the levator palpebræ superioris, ptosis occurs; in paralysis of the internal rectus insufficient or absent movement of the eye inwardly takes place.

Paralysis of the Trochlearis.—This results in disturbance of function of the superior oblique muscle. In complete paralysis there are noted limited movements of the eye, downwardly and outwardly as well as slight inward and upward squint; in the lower half of the field of vision diplopia occurs which is homonymous, and the false image is lower than the true one.

Paralysis of the Abducens.—The action of the external rectus has ceased —i. e., movements are impossible directly outward (convergent paralytic strabismus). The diplopia is homonymous and the pictures stand side by side, the distance increasing towards the side of the diseased muscle. The pathological picture described under the name of ophthalmoplegia progressiva will be considered later on.

Seat of the Cause of the Paralysis of the Muscles of the Eye; Peripheral and Central Paralyses.—A further word as to the diagnosis of the localization of the cause of paralysis shall be mentioned in deciding the question *whether in an individual instance the paralysis is due to a central or a peripheral lesion*; for the details regarding paralysis of the muscles of the eye ophthalmological text-books should be consulted. In general the diagnostic rules which have already been indicated are of use here. An important differentio-diagnostic method naturally cannot be utilized here, viz., the results of the electrical test, on account of the position of the individual muscles and nerves of the eye, as they cannot be approached. If the paralysis affecting an individual nerve be *complete* and *unilateral*, this is in favour of a *peripheral* character of the paralysis, whereas in *central* paralyses *individual fibres are paralyzed* while other fibres of the same nerves may be intact. Especially in disease of the corpora quadrigemina, these partial fibre paralyses are noted in the course of the oculo-motor; their origin may be easily explained, after anatomical investigations have proved that the oculo-motor fibres going to the different muscles arise separately in a distinct order of sequence from the nuclear ganglion cells. If it be a question of a *bilateral* oculo-motor paralysis, in general a *centrally* acting cause should be thought of (in so far as it may be supposed that the lesion represents *one* focus), the seat of the cause then may be assigned to the region of the oculo-motor nucleus.

If *hemiplegia be present besides the paralysis of the muscles of the eyes*, the question arises whether the former *alternates* with the paralysis of the muscles of the eyes or not. If the paralysis alternates, i. e., if for example in a case there exists paralysis of the extremity on one side of the body, and ptosis, dilatation of the pupil and divergent strabismus of the other side of the body, it is to be assumed that the cause of paralysis affects the [illegible] and the oculo-motor fibres of the same side both in the [illegible] brain ("central"), and the latter act *after* [illegible] tract, however, above its decussation, which

as is well known is much farther down, or that the cause of paralysis is especially a tumour at the base of the brain, in the region of a crus cerebri, for example, of the right side. As a result there is: Interruption of conduction in the right pyramidal tract above its decussation, consequently left-sided hemiplegia and a lesion of the right oculo-motor *trunk* in its exit, especially as it lies upon the inner surface of the right crus cerebri: Therefore right-sided oculo-motor paralysis. In the latter case paralysis of the extremities and oculo-motor paralysis never occur simultaneously, instead, one gradually follows the other. If the paralyses of the oculo-motor nerve and of the extremities are *simultaneous*, a focus above the corpora quadrigemina is to be thought of, which implicates *both* tracts, the pyramidal tract and the fibres of the oculo-motor in their central course *before* their decussation.

The absence of the pupillary reflex, i. e., the continuance of the dilatation of the pupil under the influence of light, is in favour of a *peripheral* oculo-motor paralysis, be it of the nerve trunk or of the intracerebral iris fibres of the oculo-motor nerve peripherally from the centre of the reflex arc transmitting the reflex.

The observation of the just-described differentio-diagnostic signs will be sufficient in most cases to decide the question whether an oculo-motor paralysis is of central or of peripheral nature, and at the same time to allow points of support for the more accurate determination of the *seat* of the cause of paralysis. In the latter respect the diagnosis will be still further complemented by the simultaneous presence of symptoms of an affection of the corpora quadrigemina or an affection of the base of the brain, (successive affection of other cranial nerves, besides alternating [crossed] hemiplegia, etc.); further by the previous action of a trauma to the cranial bones, etc. Similar points of support are present in fixing the seat of the cause of paralysis of the trochlearis and abducens. Thus in a bilateral paralysis of the trochlearis, the seat of the disease would be found in the region of the corpora quadrigemina or in the anterior medullary velum. In a bilateral paralysis of the abducens the seat will be found in the middle of the pons. A unilateral paralysis of the abducens may also be referred to a diseased focus in the corresponding half of the pons, if paralysis occurs in the opposite extremities.

PARALYSIS OF THE MOTOR PORTION OF THE FIFTH NERVE—PARALYSIS OF THE MUSCLES OF MASTICATION

The diagnosis of this rather rare paralysis of the motor portion of the trigeminus is easy, as the symptoms of this variety of paralysis are very characteristic and may be readily discerned. The act of chewing is incomplete or impossible; the hardness on contraction of the masseter and temporal muscles, which may easily be demonstrated, is absent in spite of energetic efforts made in chewing, and in cases of paralysis of long duration the sunken-in appearance of the region of the muscles of mastication is readily noticeable. A piece of cloth placed between the rows of teeth cannot be held in place; the lateral movements of the inferior maxilla are impossible, on account of paralysis of the pterygoid muscles. If there be bilateral paralysis, the lower jaw droops.

Seat of the Paralysis.—The paralysis may affect the trunk of the III branch of the fifth nerve, in morbid processes which affect the base of the skull or are exter-

nal to the skull beneath the foramen ovale, thus injuring the nerves. Accordingly as the trunk of the entire trigeminus or only the trunk of the III branch is affected, the symptoms will naturally vary. In the latter case, besides paralysis of the muscles of mastication and atrophic anæsthesia which is distributed to the temporal region, to the lower portions of the cheek and the mucous membrane of the cheek, the chin and the lower lip as well as the mucous membrane of the tongue, and also affecting the teeth and the mucous membranes of the lower jaw, it may also be complicated by ageusia (see Ageusia). Prior to the occurrence of anæsthesia the result of paralysis of the trunk, usually neuralgia occurs in the affected areas of the fifth nerve.

In other cases, the paralysis affects the *root fibres and nucleus* of the motor portion of the trigeminus in the pons, especially in cases of bulbar paralysis, or finally centrally above the crossing of the motor fibres of the fifth nerve in their cerebral course. Lately, it has also become likely, by several findings at autopsy, that also *cortical lesions* may result in paralysis of the muscles of mastication (without muscular atrophy and changes in electrical reaction), disease having been found in the lower third of the anterior central convolution and at the base of the third and second temporal convolutions, so that these cerebral areas are looked upon by some investigators as cortical fields for the innervation of the muscles of mastication. If paralysis of the muscles of mastication be due to a cortical lesion, it must be presumed that these cortical areas are *bilaterally* affected, as unilateral focal affections of the brain in general do not result in paralysis of the muscles of mastication—being explainable by the fact, that the muscles of mastication are almost universally moved bilaterally and are in connection with both hemispheres. However, it appears, according to the post-mortem findings of Hirt, that exceptionally also a unilateral (left-sided) cortical lesion in the previously mentioned areas is sufficient to produce a bilateral paralysis of the muscles of mastication. If paralysis of the muscles of mastication occurs in the course of *bulbar paralysis*, the paralysis is accompanied, as it is the result of a degenerative atrophy of the motor nucleus of the trigeminus, with a decrease in the size of the muscles, absence of the masseter reflex and changes in the electrical reaction. On the other hand, in keeping with the usually entirely intact anatomical condition of the sensory nucleus of the trigeminus, in this affection almost exclusively sensory disturbances in the course of the trigeminus are absent—i. e., anæsthesia of the skin of the face and of the tongue, disturbances of taste, etc.

The diagnosis of paralysis of the facial nerve is of much greater interest. It is the most frequent and most important paralysis of the individual cranial nerves and therefore requires a more extended discussion.

PARALYSIS OF THE FACIAL NERVE—MIMICAL FACIAL PARALYSIS

Symptoms of Facial Paralysis.—The symptoms of facial paralysis upon which the diagnosis is based are well known, and of such an unquestioned nature that, apart from those instances in which only slightest traces of the paralysis are present, there can never be any doubt as to whether the facial nerve is in a condition of paralysis or not. The vain efforts to cause contraction of the muscles of the face innervated by the facial nerve, the immobility and distorted position of the face, respectively of one half of the face, in laughing, speaking, showing the teeth, wrinkling the skin, etc., especially the disappearance of the naso-labial fold, the drooping position of the corner of the mouth upon the side of the paralysis, the open condition of the eyelid (lagophthalmus) on account of the paralysis of the orbicularis palpebrarum, the difficulties in eating (on account of paralysis of the buccinator) allow us, at first sight, easily to recognise the paralysis

in the region of the facial nerve. Upon closer examination, in some cases there are found, besides: Paralysis of the velum of the palate, and a deflection of the uvula (especially a noticeable drooping of the velum of the palate upon the affected, and a deviation of the uvula towards the healthy, side, on account of paralysis of the palatine nerves issuing from the sphenopalatine ganglion, to which the large superficial petrosal nerve conveys fibres from the geniculate ganglion), further disturbances of taste, diminished secretion of saliva upon the side of the paralysis (paralysis of the chorda), etc.

As has already been mentioned, although there are rarely real difficulties to diagnosticate a paralysis of the facial nerve from the symptoms just mentioned, and that moreover the recognition of the same belongs to the easiest problems of diagnosis in the realm of nervous pathology, on the other hand, it is often very difficult to determine the *seat* of the paralysis. If this is to occur with the necessary certainty, an exact analysis of the symptoms must be undertaken in every individual case, and the result of numerous clinical observations as well as anatomical and physiological investigations regarding the direction and course and function of the facial fibres, must be considered.

Anatomico-physiological Conditions—Central Course of the Facial Nerve.—The course of the fibres of the facial nerve, thanks to the latest investigations, has been determined in all of its parts from the cortex of the brain to its furthermost periphery. We shall first follow its *central* course and we will thus be forced to the conclusion, by our experience at the bedside as also by the result of experiments, that the *lower end of the anterior central convolution* must be looked upon as the point in the central nervous system from which, most distant from the peripheral distribution of the facial nerve, fibres of the same can be stimulated. From this region the muscles innervated by the facial nerve, especially the lower muscles of the face, can be caused to contract,[1] and of the cortical area affecting this innervation of facial fibres it is very probable that direct corona radiata fibres from this region find their way deeply into the brain. In their course downward they converge with corona radiata fibres which are in connection with the production of motor impulses in the extremities; both varieties of fibres lie in the corpus striatum, especially in the posterior peduncle of the internal capsule (near the genu) lying closely jammed together and side by side. This much is certain that morbid foci in this region, even if they have a very limited distribution, result in paralysis of the extremities and of the muscles of the face innervated by the facial nerve and upon the opposite side. From here the tract of the facial joins the tract going to the extremity through the foot of the cerebral peduncle. Farther down, the facial fibres leave the chief motor innervation tract—the pyramidal tract—so that in the pons they appear more separated and are

[1] The origin of the "upper facial," i. e., the fibres for the temporal muscles and for the orbicularis palpebrarum, is not definitely known as yet. Perhaps they are to be found in the inferior parietal gyrus, in the neighbourhood of the oculo-motor centre, also in the middle brain the upper facial seems to be in connection with the tracts of the oculo-motor. In complete atrophy of the nucleus of the facial nerve, the facial genu has been found degenerated, but a part of the facial fibres in their exit were found intact. For the latter fibres there must therefore be another origin than from the facial nucleus; it is therefore believed that the beginning of the peripheral neuron of the upper facial fibres is higher up in individual ganglion cells of the oculo-motor nucleus, and the anatomical fact is in favour of this, that a plentiful bundle of fibres originating in the posterior longitudinal fasciculus, is situated distally from the nucleus of the intramedullary facialis root.

placed more dorsally. In the lower part of the pons after the fibres have partly crossed with those on the opposite side, they enter the nucleus of the facial (at the caudal extremity of the pons) upon the side opposite to the course of the fibres in the brain, from which finally the peripheral facial nerve originates.

Peripheral Course of the Facial Nerve.—After its origin from the nucleus, the facial root first turns upward, makes a characteristic bend by two loops and then finds exit at the base of the brain, at the posterior border of the pons close to the olivary body as the facial trunk. This nerve together with the acusticus enters the internal auditory canal and separating from it in the Fallopian canal, at the hiatus it bends forming almost a right angle (genu of the facial with the geniculate

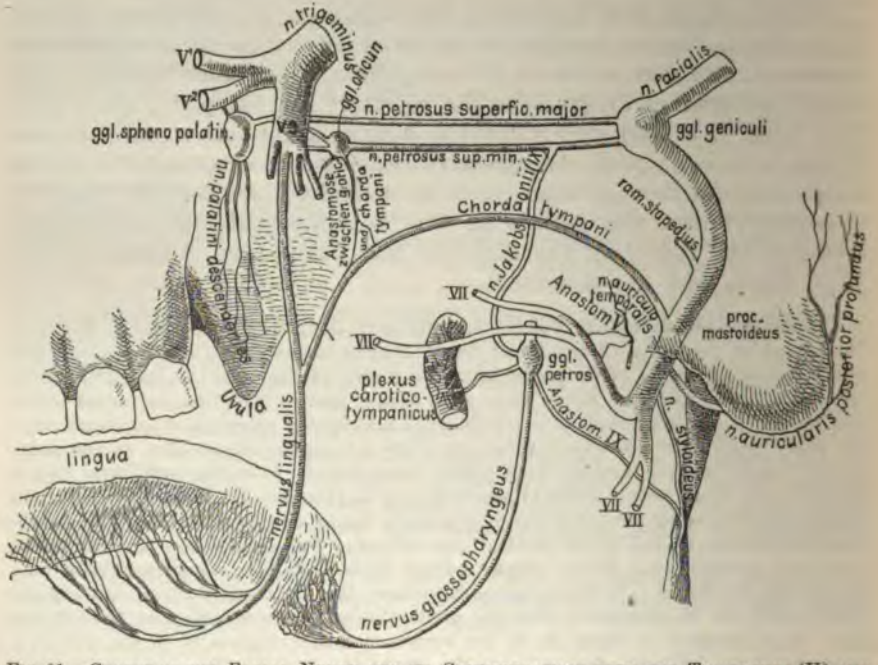


Fig. 21.—Course of the Facial Nerve and its Communications with the Trigeminus (V) and the Glossopharyngeal Nerves.

ganglion) and running downward to the mouth of the Fallopian canal to the stylomastoid foramen, where it finds exit dividing into its two main branches and their twigs which supply the muscles of the face.

During its course through the Fallopian canal the nerve after forming its genu gives off three branches from above downward; first the *large superficial petrosal nerve* whose fibres reach the spheno-palatine ganglion (with the sympathetic and the petrosal prof. major coming from the carotid plexus forming the Vidian nerve). From here motor facial elements are distributed through the palatine nerves (posterior) to the elevators of the palate, and secretory fibres for the tears by the orbital nerve (V, 2) to the tear glands. In the further course of the facial nerve in the Fallopian canal the *stapedius nerve* is given off, going to the muscle of the like name, and about one half a centimetre above the stylomastoid foramen the *chorda tympani*, which nerve joins the lingual nerve on its convex side forming a direct connection with the otic ganglion. The chorda tympani contains fibres for the secretion of saliva and centripetally running taste fibres; the latter go to the brain, after having reached the facial branch in the petrous portion of the temporal bone, but unquestionably again leave the facial nerve in the genu of the nerve partly by the large

superficial petrosal nerve, which runs to the spheno-palatine ganglion (V, 2) and partly by the superficial petrosal minor (V, 3) or to the tympanic plexus (IX). The main tract of the taste fibres originating in the chorda is by way of the small petrosal—otic ganglion—the third branch of the trigeminus. (Compare pp. 436 and 437.)

After the facial nerve has left the stylomastoid foramen, the undivided branch gives off close below the foramen: The *posterior auricular* for the muscles of motion of the concha auris, for the retrahens aurem and the transverse auricular, and for the occipitalis, further the styloid nerve for the stylohyoid muscle and to the posterior belly of the biventer mandible. The facial trunk after giving off these two branches further divides into two principal end twigs, a superior and inferior ramification, which by entwining and anastomosing form the pes anserinus; the latter as well as the dividing trunk of the facial traverse the parenchyma of the parotid gland. The *upper twig* gives off muscular fibres to the frontal muscle, the corrugator supercilii and the orbicularis palpebrarum, zygomaticus major and minor, the levator anguli oris, alæ nasi et labii superior., orbicularis oris and the buccinator. The *lower branch* supplies the motor twigs for the triangularis and quadratus menti, as well as the levator menti, finally with its lowest branch the subcutaneous coli superficialis, the lower part of the subcutaneous coli muscle and the fibres of this muscle which are distributed to the face, especially also the so-called risorius muscle.

In the main, the facial nerve is a pure motor nerve; it also carries, as we have seen, *secretory* salivary fibres perhaps also for the tears and direct fibres for the secretion of sweat, further certainly *taste fibres* in its accurately described extension and finally also, on account of its manifold anastomoses with the trigeminus, the vagus and the auricularis magnus, *sensory fibres*. In keeping with this fact, in paralysis of the facial nerve, there occurs diminution of the secretion of tears, ageusia and disturbances of the secretion of sweat, upon the paralyzed side; further, *sensory disturbances*, which in contrast to the symptoms of the motor paralysis disappear rapidly. These disturbances of sensation show themselves partly (at the onset of the paralysis) in *painful sensation* affecting the face and the vicinity of the ear, partly in slight *anæsthesia* of the affected half of the face and of the mucous membrane of the cheeks and tongue. Also, *vaso-motor symptoms*: bloated appearance of the face, herpes, etc., have occasionally been noted in the course of paralysis of the facial nerve which are said to be due to an accompanying affection of the sympathetic elements of the geniculate ganglion (v. Lenhossek)..

Upon close observation of the anatomical origin and distribution of the facial nerve, the diagnosis of paralysis of the nerve and its branches, especially the diagnosis of the division of the facial course in which the lesion giving rise to the paralysis of the nerve is found, can be determined with certainty. Real interest is only thus imparted to the analysis of the finer details of the paralysis of the facial nerve, and the significance for the entire conception of the special case is obtained in a diagnostic, prognostic and therapeutic respect.

Symptoms in General.—To diagnosticate a *facial paralysis* is very easy. It can only be overlooked, if it be developed to a very slight extent and by the inexcusable faultiness of observation on the part of the physician. The open eyelids, the continuous flow of tears, the smoothness of the skin over the frontal bone, the drooping of the corners of the mouth, the difficulty in speech, the entire absence of expression of the face in the paralyzed part are apparent at once. The paralysis of the facial muscles is more marked if the patient is asked to close the eyes, to wrinkle the forehead, to show the upper row of teeth, to laugh, to whistle, etc., the picture is espe-

cially conspicuous, if, as occurs in by far the majority of cases, a *unilateral* affection is present, and especially if all muscles innervated by the facial nerve are paralyzed, whereas in a *bilateral* facial paralysis this very characteristic distortion of the face (the point of the nose, the lip and the chin towards the healthy side) is obviously absent. But the observation that both eyes remain open and tears flow, that speech is especially difficult, and above all, that expression in the face is entirely lost, and that the face during speech, laughter, etc., remains in mask-like quiet, point to the correct diagnosis without any difficulty.

Bilateral Facial Paralysis—Individual Symptoms.—Individually the paralysis of the various branches of the facial nerve shows itself in the following phenomena: As a result of paralysis of the *frontalis*, it occurs that the forehead of the corresponding side can no longer be wrinkled. The region of the *corrugator supercilii* no longer shows longitudinal folds. The result of the paralysis upon the *orbicularis palpebrarum* is lagophthalmus, the insufficient closure of the lids upon the paralyzed side (whereby, if the patient is asked tightly to close the eyes, the so-called Bell's phenomenon appears, i. e., the physiological turning of the bulbi upward and inward and then outward in the eye in which the lids cannot close), further the overflow of tears, and as a further result the so-called ectropium paralyticum and disease of the conjunctivæ. Very conspicuous is the effect of the paralysis upon the muscles which produce movement in the nose and lips. The nasal opening appears, on account of the paralysis of the dilators (the majority of the nasal muscles) upon the affected side, narrower and smaller; the faculty of sniffing is entirely lost. On account of the disappearance of this important aid in assisting the finer smell, it is easy to understand, that patients with paralysis of the facial complain of *anosmia*, which is markedly increased by the fact that the nasal mucous membrane, on account of insufficient flow of tears into the nose, remains dry in facial paralysis. The paralysis of the *muscles of the mouth, respectively of the lips*, causes many important changes in the external appearance and in the disturbance of the functions of the mouth. The naso-labial fold has disappeared upon the paralyzed side, the angle of the mouth upon the affected side droops (on account of the paralysis of the *levator anguli oris*), the mouth space cannot be tightly closed upon the paralyzed side (on account of paralysis of the *orbicularis oris* and *buccinator*) so that fluid or saliva flows from the corners of the mouth. From the fact that during chewing, the cheek is no longer tightly pressed against the row of teeth (*buccinator*) there accumulate, between the latter and the mucous membrane of the cheeks, the foods, so that the patients in eating are compelled to make pressure upon the outer surface of the cheeks, to force the food back into the cavity of the mouth. It is also due to paralysis of the buccinator, that the patients are no longer able forcibly to eject air and fluid from the half-closed mouth; in blowing up the cheek with air upon the side of paralysis, it appears as if a loose sail were being shoved forward [flapping of the cheek]; on account of paralysis of the *sphincter oris*, the mouth can no longer be puckered and whistling is impossible. The formation of lingual letters is difficult and speech is disturbed in this respect.

The paralysis of the *zygomaticus major, levator menti* and *risorius* gives rise to incomplete laughter, and the dimple which occurs during laughing (action of the risorius) is absent upon the paralyzed side. Less conspicuous is the absence of the action of other muscles innervated by the facial nerve: The zygomaticus major, the muscles of the chin, etc. Their paralysis however can be determined without difficulty by testing them. The *tongue*, in paralysis of the facial nerve, is usually protruded straight; if it deviates, this occurs not as is the case in paralysis of the hypoglossus, towards the affected, but towards the *normal* side. In determining this condition, it must not be forgotten, that a deviation of the protruded tongue may be simulated by the crooked position of the open mouth; in rare cases, the base of the tongue upon the affected side may be deeper than upon the unaffected side (F. Schultze). A satisfactory explanation of this condition has not yet been offered.

Paralysis of the "Internal" Facial Branches.—Only in a small number of cases are the muscles innervated by the facial, those of the *velum of the palate* and *uvula* paralyzed. The velum of the palate hangs flaccidly upon the paralyzed side and moves incompletely in phonation, rarely the speech may also be nasal, and fluids upon being swallowed, enter the posterior nares; the uvula will then slant (by no means constantly) towards the unaffected side. As the cause of these rather rare phenomena, a paralysis of the palatine nerves must be supposed which are given off from the spheno-palatine ganglion through the large superficial petrosal nerve, being in connection with the genu of the facial nerve.

Taste is certainly altered in some cases, so that the anterior two thirds of the tongue, upon the paralyzed side, are not susceptible to taste impression. Obviously, paralysis of the chorda fibres is the cause of this form of ageusia; after what has previously been minutely explained, this disturbance of taste should be expected, if the cause of the facial paralysis is situated downward, from the geniculate ganglion to the point at which the chorda is given off; under some circumstances even farther down than this, to the point of communication of the facial with the auricular temporal (V, 3), or below the stylomastoid foramen, the exit of the glossopharyngeus.

Disturbance of the Salivary Secretion.—A diminution also of the *secretion of saliva* is plainly noted in some cases. The secretory fibres for the submaxillary and lingual glands are supplied by the facial nerve, whereas the secretory fibres for the parotid gland do not come from the facial but from the glosso-pharyngeal nerve (tympanic nerve—petrosal superficial minor—auriculo-temporal). The salivary secretory fibres from the facial nerve leave the trunk of the nerve by the chorda tympani; therefore, in paralysis of the facial up to the point where this nerve is given off, diminution in the secretion of saliva and complaints of the patient regarding dryness of the mouth upon the paralyzed side are to be expected. As this has not been noted in all cases it may be understood that, in a simultaneous preservation of the tympanic nerve and the course of the tract of its secretory fibres, the secretion of saliva in the parotid is not affected, yes, even may be vicariously increased, making up for the salivary secretion of the submaxillary gland, which has ceased.

For this reason, in the majority of cases, we cannot determine the *diminution in the secretion of tears*, which was to be expected (see p. 471), as the overflow of the

already secreted tears, as the result of the paralysis of the orbicularis palpebrarum, is hidden.

Disturbances of Hearing.—*Disturbance of hearing* is very frequent in patients with facial paralysis, so soon as the lesion of the nerve has its seat in the petrous portion of the temporal bone, in that herewith pathological changes in the apparatus of hearing or in the acoustic nerve occur, and difficulty of hearing results. Apart from this more accidental complication, there is another, in my experience quite rare disturbance of hearing, as a direct result of the paralysis of the facial nerve, namely, an abnormal acuteness of hearing. This is explained—whether correctly or not I cannot decide—that in paralysis of the stapedius muscle innervated by the stapedius nerve (VII) the tensor tympani, innervated by the third branch of the trigeminus, gains the upper hand and the tympanic membrane is more tensely stretched.

Sensory Disturbances.—The *sensibility* of the paralyzed half of the face is frequently entirely intact; in other cases anæsthesia is present (see p. 471). It must be especially mentioned here that occasionally a diminution of the tactile sense in the anterior half of the tongue in paralysis of the chorda is noted besides the loss of taste, which may be explained from the fact that the chorda tympani also contains, besides fibres of taste, tactile fibres. (Compare Ageusia.)

Determination of the Seat of the Facial Lesion.—If after the observation of the symptoms referred to, we have determined the presence of a paralysis of the facial nerve, the important object for the diagnostician now consists in discovering which division in the long course of the nerve is interrupted and does not conduct. In general, we should adhere to the following course in the examination and reflection. The first question to decide, is whether the special form of facial paralysis is of a central or peripheral nature.

Diagnosis of Central Facial Paralysis.—In favour of facial paralysis of *central* origin are: 1. *Partial paralysis of the middle and lower branches,* which according to experience, in lesions of the facial tract occurring in the brain, are much more markedly developed than the upper facial branches. The paralysis of the latter is not very prominent, as the normal hemisphere is usually also affected by the resulting symmetrical muscular contraction innervated by the upper facial and in case of paralysis of these branches acts by compensation. 2. That while in peripheral facial paralysis the paralysis of the muscles of the face appears to be unaffected upon strong mimical action, in cases of *central* affection *the phenomena of paralysis in mimical movements* (crying) are decidedly less strongly developed or may be *suspended entirely.* 3. *That in central paralysis, the reflex irritability of the facial branches* whose voluntary innervation has disappeared, *remains intact.*

Condition of the Reflexes in Central Facial Paralysis—Psycho-Reflexes.—This intact condition of the reflex movements does not relate only to an impulse of the sensory nerve of the face, that is to the usual reflexes, but as a rule also to those reflex movements in the tract of the facial which are independent of the will, arising from psychical emotions (psychical reflexes). For both varieties of reflexes, different reflex tracts must be assumed, according to the latest investigations: For the usual reflexes, transmission of the irritation of the sensory nerves by ganglion cells from the pons to the peripheral facial tract (which in a case of central interruption of the facial nerve is naturally intact), for the effective reflex movements of expression, most likely tracts in the corona radiata of the optic thalamus, the fibres of which carry the psychical impulses produced by irritation from the surface of the brain cen-

trifugally to the thalamus opticus. From these centres the tracts for the release of these effective movements of expression to the periphery, but *not* in the facial tract in which the voluntary irritations travel (internal capsule, peduncle of the cerebrum—"pyramidal tract"). According to Bechterew's investigations, movements of expression arise by irritation of the optic thalamus, even if the motor zone has been previously destroyed and secondary degeneration of the pyramidal tract has taken place. Moreover there is every reason for the assumption that these obliteration tracts are separated from those of the voluntary innervation facial tract in the peduncles of the cerebrum, in the nerve fibres in the tegmentum of the crus cerebrum and in the region of the tegmentum of the pons. This assumption based upon physiological experience is also in keeping with *clinical* facts. In central facial paralysis, various forms are noted in regard to the condition of these effective movements of expression. The by far most frequent variety is this, that the voluntary movement of the muscles of the face is impossible, while the affected reflex movements of expression are unimpaired (see above). In other cases of central facial paralysis there is noted loss of the voluntary and effective movements; here there must be assumed, besides an interruption of the usual voluntary tract of the facial nerve, the effect of a morbid focus upon the optic thalamus, adjacent to the internal capsule or upon the optic thalamus tracts going towards the periphery in the nerve fibres in the tegmentum. Finally, also cases exist in which the voluntary innervation of the muscles supplied by the facial is intact, but the affective movements of expression have disappeared, also in which a central facial paralysis in the ordinary sense does not exist but only a disturbance of the innervation of the facial tract shows itself by psychical phenomena, for example, during *involuntary* laughter the zygomaticus major remains immovable. That this latter psycho-reflex facial paralysis is due to disease of the (contralateral) optic thalami and the tract going out from thence, a larger number of positive clinical observations prove. In diseases of the pons as well also especially in focal disease of the thalamus, the extinction of affective movements of expression in one half of the face with retention of the voluntary facial muscles has been noted, and various observers, among others I have succeeded especially upon the basis of the symptoms mentioned correctly to diagnosticate disease of the optic thalamus *intra vitam*.

Further, 4, characteristic of the central seat of a facial lesion is the condition of the *electro-contractility* of the paralyzed nerves and muscles. This is retained in central paralysis, occasionally even increased, certainly not lost or qualitatively changed in the sense of a reaction of degeneration. 5. The *accompanying symptoms* which lead to a diagnosis of central facial paralysis vary, according to the seat of the lesion in the facial tract. In general, in favour of the origin of the lesion in the brain are: Headache, vertigo, delirium, disturbances of sight, paralysis of the extremities, hemiplegia, increase of the tendon reflexes upon the paralyzed side, etc.

Situation of Central Facial Paralysis.—Especially in favour of a lesion in the internal capsule in the neighbourhood of the knee are, besides facial paralysis, all paralyses of the extremities on the same side and of the tongue with hemianæsthesia and aphasia. Modifications of this, the usual picture, lead us to suspect a rarer location such as in the brain proper; monoplegias, epileptiform attacks, aphasia, complete retention of the function of the upper facial branches, are to be referred to a cortical lesion of the cerebrum, whereas hemiplegia, hemianæsthesia, increased tendon reflexes upon the paralyzed side and conjugate paralysis of the oculomotor (which lies adjacent at the crus cerebrum) or of the trochlearis (which leaves the peduncle at the external border close to the pons) point to an *affection of the crus cerebrum*. In favour of the seat of the cause of a

facial paralysis in the pons are: Besides hemianæsthesia, which is occasionally noted, anarthria, disturbances in deglutition, etc. (for details, see Diseases of the Pons). There are noted also, besides affection of the facial nerve, simultaneous paralytic phenomena on the part of the nucleus of the fifth nerve and abducens nucleus which lie in the pons, (paralysis of the muscles of mastication and abducens paralysis); further, cerebral nerve paralysis alternating with hemiplegia of the extremities, so-called "*crossed paralysis*" (paralysis of the facial on the side of the lesion, paralysis of the extremity upon the opposite side). The latter, as a rule, is the most important symptom of affections of the pons (see Fig. 46), but this may only be expected if the lesion be in the lower half of the pons. If the lesion is present higher up in the pons previous to the decussation of the facial fibres, the pyramidal and facialis tracts of the same side will be affected, therefore not a crossed paralysis but a contralateral paralysis of the facial nerve and the extremities of the same side will result, i. e., total hemiplegia, as in lesions of the internal capsule. The taste fibres are not affected in paralysis due to a lesion in the pons, which as has been previously explained, leave the facial tract more peripherally to enter into other centripetal tracts. The previously mentioned fact relates only to lesions of the facial nerve in the pons (intrapontal); but crossed paralysis of the facial nerve and of the extremities may also be due to *extrapontal* lesions, especially tumours, which by pressure upon the pyramidal tract at the caudal margin of the pons externally, may produce a paralysis of the extremity, upon the opposite side, and by pressure upon the trunk of the facial at the point of its exit, give rise to a total facial paralysis upon the same side. But necessarily, the abducens and the acusticus must also be affected by pressure at the base of the brain besides the facial and must also show alternate paralysis of the face with paralysis of the extremity.

Whether the crossed facial paralysis is due to paralysis of the trunk from extrapontal lesions, or occurs from intrapontal affections as a paralysis of the nucleus and root, in both cases the *electric contractility* of the paralyzed facial branches will show the characters of a peripheral paralysis (diminution of the electric contractility, with more or less well-developed reaction of degeneration), whereas a lesion of the facial fibres above the nucleus will never show reactions of degeneration or secondary atrophy of the muscles supplied by the facial nerve. With the aid of these points the *condition of the reflexes* in individual cases of pontine facial paralysis is also to be determined; these will either be normal (in an interruption of the facial tract above the nucleus), or absent, or as the transference of the sensory irritation of the facial fibres may be supposed to occur in the pons, they will be irregular, and eventually crossed. In some of the cases on account of the close approximation of the fibres in the pons *bilateral facial paralysis* may occur as the result of one or more lesions: Bilateral facial paralysis (and nuclear paralysis at that) is found in progressive bulbar paralysis.

We have now touched regions, the lesions of which strictly taken result in *peripheral facial paralysis.* The diagnosis of the latter—i. e., the determination of the special region in which the facial nerve has been injured

in its peripheral course, is much simpler and nearly always may be precisely determined.

Peripheral Facial Paralysis.—The *peripheral character of facial paralysis is noted in general* by the following characteristics: Almost always, both of the peripheral main branches of the nerve are uniformly markedly affected, i. e., paralysis of the entire trunk occurs. Very rarely is but one of the two branches injured by trauma (in one of my cases a cow with its horn only tore the lower facial branch in the affected patient). In keeping with this, *all of the external branches are incapable of conduction.* Lagophthalmus, smoothness of the forehead, etc., are well pronounced; a definite sign of peripheral facial paralysis, however, can only be obtained from the condition of the reflexes and the electric contractility in the individual case. Regarding the former, they have disappeared in complete interruption of conduction in the peripheral facial trunk; and in the latter case the electric irritability is also changed showing reaction of degeneration, and atrophy of the paralyzed muscles as a later result of a severe paralysis, may be expected with certainty.

Electrical Action in Peripheral Facial Paralysis.—As previously remarked (see p. 429) complete DeR by no means occurs regularly in peripheral facial paralysis; the electrical conditions moreover may vary very much in individual cases. Three varieties may be differentiated, which in regard to the prognosis may be designated as mild, medium severe, and severe forms, and to-day it is negligence in diagnosis, which cannot be justified, if the electrical reactions are not determined in every special case of facial paralysis. The *mild form* is characterized by the fact that the electrical contractility of the paralyzed nerve and muscle for the faradaic as well as for the constant current, remains *normal* during the entire duration of the affection, or that it may be simply diminished, without showing qualitative changes of the reaction in individual cases. Such cases recover, according to experience, in a comparatively brief time, in a few (about three) weeks. The other extreme shows the *severe* variety of peripheral facial paralysis, with symptoms of degeneration of the nerve and muscle: Loss of contractility of the nerve to both currents, complete reaction of degeneration. The prognosis in such cases is always dubious; if regeneration and recovery occurs at all, this will take place only after many months. Besides the mild and severe form, also a *medium severe* variety occurs, which as far as electrical reaction and course are concerned, remains midway between the two first-mentioned varieties. This "midform," first described by Erb, runs its course without complete DeR, moreover, with the symptoms of the previously described atypical so-called "partial" DeR, in which the nerve reacts to electrical currents (usually strong ones) and promptly to the galvanic and faradaic, whereas the muscle only shows sluggish contraction. Recovery in these medium severe varieties takes from one to two months, therefore twice as long as in the mild forms.

Seat of the Peripheral Paralysis.—If the peripheral character of the paralysis has been determined, the question occurs, *In what portion of the peripheral course of the facial nerve has the lesion occurred?* This decision has no real difficulties as soon as the individual anatomical characters of the facial course have been considered. (Compare Fig. 21.)

Paralysis of the Facial Trunk at the Base of the Brain.—The paralysis of the *facial trunk in the cavity of the skull up to its entrance into the auditory meatus*, occurs in basal affections (basal tumours, etc.) and is characterized by paralysis of all of the facial branches *excepting the fibres of taste* which leave the facial tract at the genu of the nerve and under

such circumstances as is obvious are not subject to secondary degeneration. The seat of the lesion is diagnosticated principally from the simultaneous phenomena of compression of other cranial nerves at the base of the brain, above all of the acusticus and abducens, as well as of individual parts of the brain, primarily the pons and the medulla oblongata.

The same paralytic phenomena in the facial tract, but without the last-named accompanying phenomena, occur if the facial nerve is injured in its course *in the internal auditory meatus*. In such cases we may expect that the acusticus is also affected as it runs alongside of the facial in this narrow passage and that symptoms of disease of the petrous bone and meningeal phenomena occur as complications.

Paralysis in the Fallopian Canal.—If the facial be affected in the *Fallopian canal*, the paralytic phenomena will depend upon, whether the lesion is situated in the genu or further peripherally. In the former case, besides the paralysis of all the external facial branches: Diminished secretion of saliva, vaso-motor disturbance, *disturbances of taste*, (on account of interruption of the tract of taste fibres which leave the facial tract in the superficial minor petrosal nerve) and paralysis of the *velum of the palate* (on account of the transmitted innervation by the large superficial petrosal nerve from the spheno-palatine ganglion and the palatine nerves leaving there, whose connection with the muscles of the palate has been very much questioned lately from various observations), eventually, also, abnormal acuteness of hearing. If the lesion has its seat between the exit of the stapedius nerve and the chorda tympani, there is besides paralysis of the branches going to the face only disturbance of taste and diminution of salivary secretion, whereas the velum of the palate is naturally not paralyzed and the alterations in hearing due to paralysis of the stapedius nerve can also not be determined.

Paralysis External to the Fallopian Canal.—As soon as the facial is paralyzed outside of the stylomastoid foramen or in the last portions of the Fallopian canal below the exit of the chorda tympani, naturally all internal branches are capable of action, i. e., taste, secretion of saliva, hearing, position of the palate, are normal, all muscles of the face, however, are paralyzed, and according to the seat of the lesion also the muscles of the concha (posterior auricular nerve) and the muscles supplied by the styloideus.

Finally, if the lesion be still more peripheral, therefore in a position which is *beyond the division of the nerve into its upper and lower branch*, the distribution of the paralysis under all circumstances will be very limited, and will affect at one time a greater at another time a lesser number of the muscles.

Diagnosis.—The analysis of the individual case in facial paralysis as described involves in itself the question of the special *cause* of the lesion of which is an integral part of the diagnosis. The method followed in this respect in this examination therefore must be

facial nerve, in which the seat of the lesion can be diagnosti-
facial tract, *below the origin of the chorda tympani*, may be
changes in the region of the ear: Tumours of the parotid gland,

deep ulcers of the face, etc. These are to be considered primarily; further trauma which has occurred in this region should be noted, etc., in the facial paralysis in the new-born also pressure from the pelvis or from the forceps during birth, pressure scars, etc., should be noted. If no such direct cause can be determined, it is most likely that neuritis is the cause of the paralysis or that severe *cold* may have produced the affection. The latter cause is only too readily admitted by the patient, but must always be accepted by the physician with caution and only when no other plausible reason for the lesion can be determined. But beyond all doubt, it is nevertheless a fact that many paralyses of the facial nerve are of a rheumatic nature; on the contrary, if we do not desire to force the facts, in one quarter of the cases nothing remains but to accept the action of cold as the cause of facial paralysis. In one half of the cases the reason of the peripheral paralysis remains entirely unknown; perhaps an intoxication or an infection plays the principal *rôle* in these.

If there are symptoms which point to a lesion of the facial *inside of the petrous bone*, injury to the ear by trauma, fracture of the petrous portion of the temporal bone and especially disease of the ear, must be determined. Most frequently, in these cases, there will be found, caries of the petrous bone with an internal otitis, more rarely simple catarrh of the middle ear or neoplasms of the internal ear. In all cases a flow from the ear must be investigated in this connection.

Basal paralyses of the nerve are due to fractures of the skull, exudates, aneurysms, tumours, acute and chronic meningitis; above all in facial basal paralysis, syphilis should be suspected; the products of this disease at the base of the skull (periostitis, osteomyelitis, gummatous nodules, etc.) may produce pressure upon the nerve.

Peripheral facial paralysis may also, as it appears, similar to other forms of paralysis, occur in enteric fever, scarlatina, diphtheria, etc., also in the course of constitutional diseases as the result of gout, leucæmic new formations or from diabetes mellitus.

If we are dealing with a *central facial paralysis*, if the form of the paralysis points to the pons, we should primarily think of bulbar paralysis or multiple sclerosis; if the cause of the paralysis be seated higher up in the brain, hæmorrhages, emboli, abscesses, and eventually also cortical affections should be considered. The differentio-diagnostic analysis of the last-mentioned ætiological conditions will be discussed under diseases of the central nervous system.

PARALYSIS OF THE VAGUS

PARALYSIS OF THE SPINAL ACCESSORY

Paralytic Phenomena in the Course of the Pneumogastric.—*The paralytic phenomena in the course of the vagus*, have been to a great extent discussed previously in other chapters; I refer in this connection to the diagnosis of *paralysis of the muscles of the larynx* (laryngeus superior and recurrent laryngeal), the diagnosis of *paralytic dysphagia* (partly also due to paralysis of the glossopharyngeal nerve which gives off sensory and motor fibres to the top of the œsophagus), the *cardiac neuroses*, in which latter disease conditions, the question whether branches of the vagus are paralyzed or not can never be decided with certainty. In the cases observed by me, in which a paralysis of the *cardiac inhibitory fibres*, respectively of the heart vagus centre in the medulla oblongata could be assumed with great probability, *the pulse frequency increased to more than double*, and this was followed by a pulse which was in general not slow, the beats following in a very uniform manner. This change was especially conspicuous in a case of multiple spinal sclerosis, in which as the autopsy showed, the morbid areas reached the medulla oblongata and unquestionably in the last days of life affected the pneumogastric centre. Other observations at the bedside, however, have shown that paralysis of the pneumogastric nerve may also develop arrhythmic cardiac activity, and that, with this, difficulty in full respiration may occur, which also takes place in experimental severing of the vagus.

Besides, certain paralytic conditions of the vagus have been described occur-

ring in *diseases of the stomach and intestines*, especially in the clinical picture of the nervous form of these affections. (See these diseases.)

Paralysis of the External Branch of the Spinal Accessory.—Much more certain is the diagnosis of *paralysis of the spinal accessory*, at least, there can be no doubt of the presence of paralysis of the *external branches* of this nerve which supply the motor filaments, for the sternocleidomastoid and trapezius. Both branches may be affected together or each may be paralyzed individually. The paralysis of the *sternocleidomastoid muscle* which is *exclusively* innervated by the spinal accessory, is characterized by an oblique position of the head, with an upward position of the chin towards the affected side (on account of the action of the unaffected sternocleidomastoid); at the same time the active turning of the head towards the opposite side gives rise to difficulties, whereas passive turning, in contrast to that occurring due to spasm giving rise to the caput obstipum, may be easily carried out. In the later course, a contracture of the healthy muscle may develop which makes the diagnosis more difficult; however, during this time the well-developed atrophy of the paralyzed muscle will scarcely ever be absent and this will set the diagnosis aright. The innervation of the *trapezius* is largely due to the accessorius; it is still a question of discussion, whether the middle portion of this muscle is supplied by spinal branches from the cervical plexus (third and fourth cervical nerves) and that, due to this secondary innervation in paralysis of the accessorius, the dislocation of the scapula is prevented. *A total paralysis of the trapezius* shows itself in an abnormal position of the scapula, which Duchenne was the first to analyze, and who characterized it as a "mouvement de bascule" (swinging position). The scapula moves laterally from the vertebral column, the shoulder falls forward and at the same time downward, in that the scapula is turned upon an axis which is horizontal from before backward so that the external angle of the scapula is moved downward, the lower upward and inward, the upper internal angle upward and laterally. The shoulder can be raised with difficulty, and this occurs by the action of the levator anguli scapulæ, which is innervated by the upper cervical nerves and especially raises the upper inner angle to which it is attached. With some trouble there is still possible an approximation of the scapula somewhat upward and against the vertebral column, by the action of the rhomboideus which draws the shoulder-blade, especially also its lower angle inward and upward, and assists the middle portion of the trapezius in the retraction of the shoulder. Elevating the arm beyond the horizontal [illegible] is influenced, as an elevation of the acromion by the trapezius [illegible] (Compare also p. 185.)

[illegible] especially of the sternocleidomastoid the lower part of the [illegible] drawn towards the neck, especially if both trapezii are also [illegible] the head under these circumstances falls slightly forward. [illegible] of the trapezii causes, besides, as the shoulder-blades [illegible] towards the median line of the back, an increase in the [illegible] transversal rounding of the back. The diagnosis [illegible] the observation of the atrophy of the affected

Paralysis of the Internal Branch of the Spinal Accessory.—If besides the external branch of the accessorius, also the *internal* branch is paralyzed, there is found with paralysis of the affected sternocleidomastoid and trapezius muscles, symptoms of paralysis of the vagus, as the greatest portion of the motor fibres of the vagus and also the inhibitory nerves going to the heart are supplied by the entrance of the internal branch of the spinal accessory into the plexus ganglioformis. In keeping with this, there are noted, disturbances in deglutition (on account of paralysis of the constrictors of the œsophagus) and symptoms of paralysis of the muscles of the larynx, whereas the sensibility of the mucous membrane of the larynx and pharynx is maintained (in contrast to paralysis of the pneumogastric). Besides, increase in the pulse rate, at least in a bilateral paralysis of the spinal accessory, has certainly been noted.

PARALYSIS OF THE HYPOGLOSSAL NERVE—GLOSSOPLEGIA

Signs of Paralysis of the Hypoglossal Nerve.—Paralysis of the hypoglossal nerve is comparatively frequently the subject of diagnosis, as it is a very usual accompanying phenomenon in the picture of various diseases of the central nervous system. The recognition of a well-developed paralysis of the hypoglossal nerve is easy. *In a unilateral paralysis, the tongue deviates in being protruded towards the affected side*, simultaneously with an arching of the raphe, so that it is directly towards the paralyzed side concavely; the deviation of the tongue is the result of the action of the normal genioglossus muscle. In a *bilateral* affection, the tongue can only be protruded with great difficulty (in complete paralysis) or it may not be protruded at all and may lie immovable at the bottom of the oral cavity, chewing and swallowing are impeded, as the bolus can no longer be forced back into the œsophagus; saliva also is insufficiently swallowed and collects in the mouth. Especially conspicuous is the *disturbance of speech*, varying according to the grade of paralysis; there will be great difficulty in the formation of lingual sounds (the consonants, l, t, s, sch, r, n, k, ch, the vowels u, e, o, and especially i, and in German the modified vowels, ä, ü, etc.); this may increase so that only unarticulated muttering occurs.

Central Hypoglossal Paralysis.—The hypoglossal nerve may be paralyzed peripherally or centrally. If the diagnosis of a peripheral hypoglossal paralysis is to be recognised with certainty, the presence of a central paralysis must first be excluded. The latter rarely offers great difficulties as *central* paralysis may be easily distinguished by the intactness of the electrical contractility and the absence of atrophy of the muscles of the tongue. As a rule central paralyses are unilateral; but exceptions may occur, as the innervation of the tongue, obviously for both sides is supplied by one hemipshere even if to an unequal degree. For this reason there may occur, as has been variously noted, a bilateral paralysis of the hypoglossal nerve by a small lesion of *one* hemisphere, or also—the more frequent case—a unilateral paralysis may be but slightly developed, as in this instance the unaffected hemisphere with its secondary innervation supplies the absent innervation of the same half of the tongue. The clinical picture of paralysis of the hypoglossal nerve varies, according to the situation of the paralysis in the central tract of the hypoglossal fibres, from the cortex up to its nucleus.

Cortical paralysis of the hypoglossus is characterized by a *simultaneous paralysis of the facial nerve*, especially of the lower branches of the facial, as the cortical field of the lower branches of the facial and of the hypoglossal undoubtedly lie very close together (the origin of the upper branch of the facial is to be looked for else-

where, see Facialis); this approximate area is found in the lower third of the central convolution—therefore almost invariably the lesion will affect both nerves. The paralysis of the facial, in such cases, is much more marked as a rule than the hypoglossal paralysis, as the innervation of the hypoglossus occurs less strictly unilaterally than does that of the facial. If the paralysis is limited to the fibres of the facial and hypoglossal nerves (*monoplegia glosso-facialis*) this is unquestionably in favour of the character of the paralysis being cortical, as in the more internal portions of the brain, especially in the internal capsule and in the crus of the cerebrum, the hypoglossal tract and the facial tract are so closely contiguous to the pyramidal tract, that a lesion almost without exception, besides producing contralateral paralysis of the tongue and muscles of the face, will also give rise to paralysis of the extremities. In favour of a paralysis of the hypoglossal of cortical origin, there are, further, especially the appearance of a monoplegia brachialis, epileptiform twitching and cortical (motor) aphasia, which is however by no means a necessary attribute to the monoplegic glosso-facialis. (See further the chapter on Aphasia.) After what we know, in reference to cortical sensory paralysis, it is likely that, similar to cortical lesions in the region of the central convolutions in general, so also in cortical hypoglossal paralysis, disease of the anterior central convolution results in sensory disturbances of the tongue; a certain opinion however in reference to this point is not yet possible.

Paralysis of the Hypoglossal as Secondary Manifestation of Diseases of the Internal Capsule and of the Peduncles.—On the other hand, simultaneous sensory disturbances will be absent provided the hypoglossal tract is affected farther down in the *internal capsule*, especially as is so frequent, in the genu of the same and the upper part of its posterior crus. As the pyramidal tract somewhat farther down in the capsule joins the facial-hypoglossal fibres, there must be expected, in a lesion of the previously mentioned area of the capsule, besides paralysis of the hypoglossal, also paralysis of the extremities upon the crossed side, which at the same time shows permanent hemianæsthesia, provided the lower third of the posterior crus of the capsule is also injured. The deviation of the tongue, and this towards the paralyzed side, is well pronounced in paralyses of the internal capsule, whereas aphasia (the subcortical variety would be expected) is almost invariably absent, or, if it be present at all, can only be explained as the distant action of the affected lesion upon the cortical areas or portions of the corona radiata which are in connection with speech.

Also in disease of the peduncles of the cerebrum, besides the contralateral paralysis of the extremities, in a number of cases, paralysis of the hypoglossal (and facial) is noted; whether it is accompanied by hemianæsthesia, depends upon the extent of the affected area in the tegmental tract of the peduncles of the cerebrum. Of especial importance diagnostically, for the establishment of a connection between hypoglossal paralysis and an affection of the peduncles, is the simultaneous presence of an alternating (partial or complete) oculo-motor paralysis. (Compare Fig. 51.)

Nuclear Hypoglossal Paralysis.—The pathological picture produced by a lesion in the *nucleus of the hypoglossal nerve* in the medulla oblongata, is well characterized. The sign of this affection is a disturbance of the mechanism of articulation, in so far as the movements of the tongue are concerned with them—the *anarthria*, further, the difficulty in chewing and (the first phases) of swallowing, in which acts the movement of the tongue play an important part, and finally the atrophy of the paralyzed tongue, which is moved with difficulty besides showing a diminution in volume, and also presents DeR, and fibrillary contractions, but does not reveal disturbances of sensation. These symptoms, however, are not exactly characteristic of nuclear paralysis; they are the general signs of disease of the hypoglossal nerve in its *peripheral* course, no matter whether the nerve be more

injured in its peripheral or in its central ends, or of its nucleus. The implication of the latter can only be determined, if there are *accompanying phenomena* pointing to disease of the medulla oblongata—i. e., if besides a (bilateral) hypoglossal paralysis, complete dysphagia (IX, X, XI, XII-paralysis), aphonia and alterations in the pulse frequency (X, respectively XI-paralysis), dyspnœa and eventually unilateral or bilateral paralysis (and anæsthesia) of the extremities exist. Paralysis of the nucleus and of the intrabulbar fibres of the hypoglossal are found in various affections of the medulla oblongata (softening, hæmorrhage, etc.), further in a greatly disturbed tabes dorsalis, especially one travelling far upward, and in progressive muscular atrophy; frequently, however, nuclear hypoglossal paralysis is a symptom of multiple sclerosis and of progressive bulbar paralysis, as shall be more accurately described later on.

Anarthria also occurs as a secondary symptom *in disease of the pons*, due to the hypoglossal fibres being injured, prior to their entrance into the nucleus (before their crossing). In this form of anarthria, and in the difficulty of movement of the tongue, the atrophy of the latter is absent as well as DeR, and shows itself by other symptoms as an affection of the pons, by paralysis of the abducens and facial with alternating paralysis of the extremities, etc.

Paralysis of the Trunk.—If the paralysis of the hypoglossal nerve shows the unquestioned character of a peripheral lesion which has several times been referred to, i. e., DeR, atrophy, wrinkling and deep furrowing of the tongue upon one side, or (in bilateral paralysis) upon both halves of the tongue, and a nuclear paralysis of the nerve may be excluded, there may be considered diagnostically only now, *paralysis of the trunk and branches* of the nerve. This, in general, is rare, due to trauma, cicatrices, enlargement of the glands of the neck, disease of the upper vertebræ of the neck and at the base of the skull; neuritis is the rarest cause of a peripheral hypoglossal paralysis. In the diagnosis of this condition the ætiology must always be considered.

PARALYSIS IN THE COURSE OF THE CERVICAL NERVES

Of the paralyses in the course of the four upper cervical nerves, which besides supplying the muscles of the throat and back of the neck, also supply the diaphragm by means of the *phrenic nerve*, only the paralysis of the last-named nerve is of greater diagnostic interest.

Paralysis of the Diaphragm.—This is characterized by *marked changes in respiration*. As the principal muscle of inspiration is paralyzed, it is not possible for the patient to take deep, full inspirations. If he makes the attempt, the thorax will be partly dilated in its upper portions, whereas the lower parts of the thorax in the region of the epigastrium will be drawn in, due to the external pressure of the atmosphere, again to come forward with the succeeding expiration. The liver following this movement, will move upward and inward in inspiration and on expiration downward and outward. This pathological respiratory movement, however, only occurs in forced breathing in which case dyspnœa and increased frequency of respira-

tion will also be noted; in quiet respiration only the costal respiratory type can be discerned. If but the slightest pressure be made upon the epigastrium with the hand, every movement of the diaphragm, this usually so powerful muscle, may be entirely hindered, yes, even the arching of the abdomen may be prevented. Percussion shows a permanent high position of the lower pulmonary borders; in an upright position of the patient, the lower pulmonary border is farther down than in the recumbent posture. As the lower pulmonary areas are less distended, the formation of atelectasis and hypostatic congestion is favoured.

Besides the mentioned changes in the respiratory movement, the influence of paralysis of the diaphragm is also seen in all so-called "abnormal respiratory movements," as the movement of the diaphragm in general is also necessary for their performance, such as blowing the nose and especially in coughing. If the explosive expiratory acts are to be marked, a deep diaphragmatic inspiration must precede it, for this reason, the bronchial secretion of an intercurrent bronchitis or pneumonia can with difficulty be expectorated. As the action of the abdominal pressure in the inspiratory position of the diaphragm is the most easy (in which the most marked diminution in the abdominal space is possible), defecation becomes difficult in paralysis of the diaphragm, also vomiting and especially if it be exhaustive, can only result incompletely, as the act of vomiting must be accompanied by energetic contraction of the abdominal muscles, due to very deep inspiratory movements by the diaphragm with a simultaneously closed glottis.

Ætiology.—According to what has been stated, the diagnosis of paralysis of the diaphragm is easy; it is more difficult, if it be unilateral as is the case in compression or injury of *one* phrenic nerve at the neck. As a rule, however, it is bilateral, occurring in disease of the cervical vertebræ, and as secondary phenomena in diphtheritic paralysis, in multiple alcoholic neuritis, in lead-poisoning, in hysteria, in Landry's paralysis, in progressive muscular atrophy and in tabes; a rheumatic form of this paralysis has also been noted. These ætiological conditions must be considered in the diagnosis in every individual case.

Of the paralyses in the course of the *four lower cervical nerves* those of the terminal branches of the brachial plexus are the most important, but occasionally the paralysis also affects branches of the upper part of the brachial plexus; especially worthy of note in this respect are paralyses of the subscapular nerves and of the posterior thoracic nerve (paralysis of the serratus).

Paralysis of the Subscapular and Suprascapular Nerves.—*Paralysis of the subscapular nerves* shows itself by the disturbance of the function of these nerves in the muscles supplied by them, the latissimus dorsi, teres major, and the subscapularis, which are all three inserted into the bicipital groove of the humerus. If the paralysis affects the *latissimus dorsi*, the principal action of the muscle, bringing the upper extremity in contact with the buttock, as well as the downward and sidewise movement of the raised arm, is disturbed. The paralysis of the *teres major* and *subscapularis* principally produces difficulty in the inward rolling of the upper arm, so that the arm which has been rotated outward can no longer be actively rolled inward, which is especially noted in the use of the hand (for example in washing). In contrast with this is the picture of paralysis of the *suprascapular nerve* which supplies the supraspinatus, infraspinatus and occasionally the teres minor (usually supplied by the axillary nerve). These muscles are all inserted into the greater tuberosity of the humerus and rotate the upper arm around its axis outwardly. In paralysis of the suprascapular nerve, principally the outward rotation of the arm becomes difficult, in which the various movements of the hand: Writing, sewing, etc., are markedly disturbed.

Paralysis of the Serratus.—More frequent, and better understood is the isolated paralysis of the long *posterior thoracic nerve* which supplies the *serratus anticus major*. This muscle, arising from the external surface of the eight upper ribs and being inserted into the entire internal surface of the scapula (basis scapulæ) determines principally the position of the shoulder-blade; it draws the scapula outward and forward, and in its contraction holds the scapula pressed tightly against the trunk. If the latter movement, due to paralysis, is incomplete or no longer possible, the arm in raising turns the lower angle of the scapula towards the spinal column, the base and with it the entire scapula upward; this causes the shoulder-blade to distance itself from the thorax, *so that, with the arms raised forward, the scapulæ stand out like wings.* As a rule, *raising the arms above the horizontal is then impossible.* Raising the arm up to an angle of 90 degrees is accomplished by the action of the deltoid; if the arm is to be raised higher, the scapula must be turned around its sagittal axis and tightly pressed against the wall of the thorax, which under normal conditions is accomplished by the serratus anticus in connection with the trapezius. If the scapula of the patient is turned somewhat forward and is held simultaneously tightly against the body, raising the arm above the horizontal plane now can be accomplished without difficulty. In some few cases, in spite of the paralysis of the serratus, the arm may be raised without difficulty above the horizontal plane which is accomplished by an energetic action of the trapezius, especially of its middle or upper portions. If the trapezius is also paralyzed, the arm can no longer be raised. The scapula in this case (i. e., in paralysis of the trapezius) shows an exquisite oblique position so that its inner border from within and below runs from without upward (swinging position, see p. 480). But also in isolated paralysis of the serratus, even in rest—i. e., with the arm hanging downward, a changed position of the shoulder-blade may be noted: *High position of the scapula, approximation of the same to the vertebral column, and the lower angle of the scapula standing off from the thorax.* The reason of this abnormal position may be looked for in the fact, that in paralysis of the serratus anticus its antagonists, the rhomboidei, gain the supremacy and draw the base of the shoulder-blade towards the vertebral column and upward. The position of the lower angle of the scapula which stands off, is assisted by the increased action of the coracobrachialis and of the short head of the brachial biceps, as both these muscles arise from the coracoid process. The primary spasmodic contraction of the antagonistic muscles of the serratus would naturally produce a similar position of the scapula; it may however be differentiated from paralysis of the serratus in that in the latter case the scapula is easily movable passively. If both serrati are paralyzed, the shoulder-blades approximate almost to the point of touching. Besides the impossibility of raising the arm above the horizontal (which occurs in the majority of cases), paralysis of the serratus is further seen in the difficulty with which the arms are crossed and brought forward. An influence upon the respiration, due to paralysis of the serratus, is not to be observed. The action, which was formerly ascribed to this muscle, regarding respiratory movements—in that it was said it assisted during overexertion in

inspiration (N. "respiratorius externus" Bell)—is in all cases very subordinate, as only the upper portions of the muscle, by raising the ribs, might have a very slight influence.

Ætiology.—The nerve which supplies the serratus is the *posterior long thoracic.* It pierces, arising from the upper portion of the brachial plexus, the scalenus medius and running in the axillary line behind the brachial plexus at the wall of the thorax, enters into the serratus and distributes itself by numerous branches exclusively into this muscle. It is obvious that the nerve in its long course is exposed to injury. Therefore, in the diagnosis of serratus paralysis, we must look for trauma, affecting the region of the back of the neck and especially the region of the brachial plexus, to preceding pressure of the scalenus or its nerves, due to carrying heavy weights upon the shoulder, twisting of the muscle, by forced movements of the neck, etc. As the rhomboidei, which are supplied by the dorsalis scapulæ (simultaneously with the thoracic posterior) pierces the scalenus medius, the first-named nerve may be very readily paralyzed simultaneously, which will markedly modify the serratus paralysis in its intensity, that is, in lessening its effects, as the antagonistic action of the rhomboidei will prevent, to some extent, the deviation of the scapula. If no cause can be found, which may be said to be due to the anatomical relations of the nerve, for paralysis of the serratus, the thought should now arise, whether the paralysis is due to infectious diseases (influenza, etc.) or to rheumatic infectious processes. Not rarely is serratus paralysis an accompanying condition of progressive muscular paralysis, especially of the juvenile form.

PARALYSIS IN THE COURSE OF THE TERMINAL BRANCHES OF THE BRACHIAL PLEXUS

Paralysis of the Axillary Nerve—Deltoid Muscle.—The nerve which arises from the posterior portion of the brachial plexus, supplies the teres minor and the *deltoid.* The paralysis of the latter muscle has practical importance, and may be due to a neuritis affecting the axillary nerve, especially however may it result from fracture of the humerus or dislocation of the humerus, particularly of the head posteriorly; it may also be due to pressure from a crutch, etc. The arm can no longer be raised to a horizontal position, nor can it be moved sideways from the thorax wall (neither forward nor backward). Especially easy, according to my experience, may paralysis be determined, if the arm is raised above the horizontal position and now is allowed to fall. In this act, the raised arm falls slowly to the horizontal position, but thence rapidly, almost resembling a dead mass, and falls against the trunk. If atrophy occurs in the muscle, the previous roundness of the shoulder becomes flat and is very conspicuous, and as the deltoid covers the capsule of the humeral articulation, an abnormally marked passive movability of the head of the humerus in the socket and a flail-joint occurs. In isolated paralysis of the axillary nerve also paralysis of the sensory branches of the axillary, upon the back and the outer surface of the upper arm, may be determined. (Compare Figs. 14 and 15.)

PARALYSIS IN THE COURSE OF THE RADIAL NERVE

Ætiology.—This is the most frequent form of paralyses in the course of the brachial plexus, and may be readily explained from the exposed superficial position of the nerve, being susceptible to pressure and trauma of all kinds. The nerve winds around the posterior surface of the bone of the upper arm from within outward and becomes superficial at the beginning of the lower third of the bone, then running between the brachialis internus and the origin of the supinator longus to divide in two branches around the external condyle of the humerus, giving off the sensory ramus

superficialis and a branch for the innervation of the extensor muscles of the forearm, the ramus profundus. Compression of this nerve most frequently occurs during deep heavy sleep, the head being pressed against the underlying arm, or the arm is rested against a hard substance beneath. Less rarely does it occur that a crutch in the axillary space presses the nerve against the humerus, or that, in an extraordinarily marked contraction of the triceps, the nerve lying upon the bone and covered by the triceps brachii, cannot deviate quickly enough and in this manner is compressed. Further, a blow upon the arm, a fracture of the humerus and callus formation, a chemical irritant (for example, an injection of ether in the arm, etc.) may injure the nerve and give rise to radial paralysis. More rare is a radial paralysis as the result of neuritis and rheumatic influences.

Radial Paralysis from Lead-Poisoning.—Especially interesting in a genetic respect is a frequent form of radial paralysis—which arises as the result of chronic *lead intoxication*. For the greatest number of cases we may assume a peripheral neuritis, for the other portion, those occurring primarily in the course of the radial nerve due to lead-poisoning, a spinal origin. A distinct separation of these cases however, according to our present views regarding the structure of the nervous system from neurons, is no longer consistent and a dispute in reference to this subject is of very slight importance. According to the results of pathological research, it appears that the action of lead (which like other poisons attacks certain *isolated* areas of the nervous system, in a manner not yet understood) as a rule, only affects certain peripheral nerve fibres, rarely on the other hand, and only in severe cases, special ganglion-cell groups in the anterior horns, which innervate limited groups of muscles belonging functionally together. Very probably such ganglion-cell groups are present in the intumescentia cervicalis, which are intended for the innervation of the extensor muscles, whereas those cells intended for the innervation of the biceps, brachialis internus and supinator longus (muscles which together bend the forearm) form a group which is separated by some little distance. This would most easily explain, 1, why the supinator longus, although innervated by the radial nerve, is almost without exception unaffected in a saturnine radial paralysis and, 2, inversely, if in exceptional cases, not the extensors but the supinator is affected by the action of lead, simultaneously with it, the biceps and the brachialis internus appear to be affected. An example of this unquestionably rare condition was observed by me not long since and the brief history of the case will be related here:

Case of Unusual Localization of Radial Paralysis due to Lead.—F. H., varnisher, twenty-seven years of age, was taken sick as an apprentice twelve years ago with epileptic attacks, which have since recurred at shorter or longer intervals within the last six months. During the last year severe colics occurred, and a few days before his admission to the hospital, drawing pains in the legs were noted. The motor power of the upper extremity did not appear to be markedly altered, the test of the electrical reaction of the arm muscles however, showed very conspicuous deviations from the normal. Upon the *right* side, the electrical test of the triceps and extensor digit. comm. with a weak constant current showed *normal* conditions, whereas the *supinator longus* showed the well-developed picture of DeR, (prominence of ACC, sluggish contraction), *also the biceps* in its lower area and the *deltoid* in individual fibres, towards the pectoral muscle. Upon the *left* side this very conspicuous localization of the changes in reaction which favoured lead intoxication, were at least indicated in the same manner, in that the *biceps* in the same region as on the right side showed distinct DeR, the deltoid CaCC = ACC, the other muscles showing normal reaction.

Closely related to toxic radial paralysis is the infectious form following enteric fever, etc. Cerebral radial paralyses are usually accompanying phenomena of hemiplegia; they are very rarely isolated as the result of cortical lesions.

Symptoms of Radial Paralysis.—The symptoms of radial paralysis are very marked, so that there is rarely difficulty in the diagnosis provided we understand the distribution of the radial nerve.

The nerve supplies the triceps and the anconeus quartus, the supinator longus, extensor carpi radialis longus and brevis, extensor digitor. communis, extensor digiti minimi and carpi ulnaris, further the more deeply lying muscles, supinator brevis, extensor pollicis longus and brevis, the extensor indicis and abductor pollicis longus—i. e., therefore all the muscles upon the dorsal side of the forearm. In keeping with this, the hand in paralysis of the radial nerve in flexion, is towards the palmar side, in slight pronation, the fingers are also slightly flexed and the thumb is adducted (by the ulnar nerve ending in the adductor pollicis).

FIG. 22.—POSITION OF THE HAND IN RADIAL PARALYSIS. (CASE OF TRAUMATIC PARALYSIS).

The forearm cannot be placed in an extensor position in case the triceps and anconeus quartus are also paralyzed; above all, the hand and fingers cannot be dorsally flexed; an extension of the latter is still possible, but only in the region of and due to the action of the interossei and lumbricales (ulnar nerve), i. e., an extension of both terminal phalanges in simultaneous flexion of the first phalanges. The thumb which is found to be in a position of flexion and adduction cannot be extended (extensor pollicis longus and brevis), nor can it be abducted with force, whereas a partial separation of the thumb from the index finger is still possible, due to the action of the abductor pollicis brevis (median nerve).

Description of Radial Paralysis.—The paralysis of the *extensor digitorum communis* is seen, besides from the fact that it is impossible for the patient to extend the fingers, also in that the grasp of the hand is weakened. By the change in the position of the hand as a result of paralysis of the radial nerve, the points of the insertion of the flexors has become closer, their action therefore less powerful than in simultaneous extension, and this is also true regarding the action of the abductors and adductors of the fingers. *Supination* of the forearm is impossible, on account of paralysis of the supinator brevis; however, this loss of supination, due to the previously mentioned muscle being affected, can only be noted provided the arm is brought into an extensor position, as in a simultaneous flexion of the forearm the supination of the hand is brought about by the biceps (musculo-cutaneous nerve). The paralysis of the supinator longus is shown in the absence of contraction of the muscle, which can be noted if the forearm is forcibly flexed, after it has been brought into a median position between pronation and supination. The action of the supinator longus consists in flexing the forearm, in a half-pronated supinated posi-

tion, whereas the muscle assists supination but little and only provided a marked pronated position of the forearm has been previously assumed.

Very frequently paralysis of the radial nerve is also conspicuous, from a marked atrophy of the extensor muscles, so that the dorsal surface of the forearm appears to be completely flattened; with this, as an expression of trophic disturbance, nodular enlargements appear at the carpal and finger joints and in the course of the tendons of the extensors. By no means constantly, but in a certain number of cases, besides the very pronounced motor paralysis, there is a *diminution of sensation* in the distribution of the radial nerve, i. e., in paralysis of the radial nerve high up—anæsthesia upon the dorsal surface of the first two fingers and upon the radial border of the middle finger. (Compare Fig. 15.)

PARALYSIS IN THE COURSE OF THE ULNAR NERVE

Paralysis of the Ulnar Nerve.—Paralysis of the ulnar nerve, upon the whole, is rarely due to trauma or compression of the nerve (crutch pressure, sleeping upon the underlying arm, pressure upon the elbow—usual in certain occupations: In glass blowers, xylographers and others—further from wounds, compressions, etc.), neuritis and rheumatic conditions or to infectious diseases (enteric fever, syphilis). The result of paralysis of this nerve however is noted in certain muscles which atrophy and cause functional weakness, which is frequently seen in the clinical picture of progressive muscular atrophy. The paralysis is characterized by a disturbance in the use of the muscles supplied by the ulnar nerve: The flexor carpi ulnaris (the inner half), the flexor digitorum profundus, the palmaris brevis, the flexor, abductor and adductor digit. min., *the interossei*, partly the lumbricales (the fourth and usually also of the third) and finally of the *adductor pollicis*.

The ulnar flexion of the hand is therefore limited, as well as the flexion of the finger in the end phalanges, especially of the last finger, but also the flexion of the fingers in the terminal phalanx, on account of *paralysis of the lumbricales and interossei*, the latter in fact producing the most characteristic phenomenon in the picture of paralysis of the ulnar nerve. As the combined action of the *interossei and lumbricales* (flexion of the terminal phalanges, extension of the middle and end phalanges) disappears, the antagonists gain (in paralysis of the ulnar in the lower half of the tract of the nerve in the forearm after the branches have been given off for the flexor digitorum profundus and especially also in progressive muscular atrophy) the upper hand (the extensor digitorum communis and the flexores digitor.), so that the well-known claw position of the hand appears.

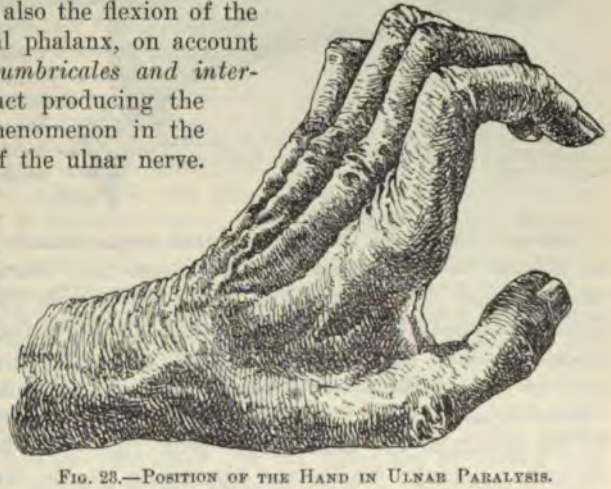


Fig. 23.—Position of the Hand in Ulnar Paralysis. (Case of Traumatic Paralysis).

In both last fingers the claw position is more marked than in the first and second finger, the lumbricales of which are supplied by the median nerve. By paralysis especially of the *interossei, separation of the fingers is impossible*; the little finger has entirely lost motion and the thumb can no longer be adducted. The paralyzed muscles become atrophic, *the spaces between the bones in the middle of the hand are empty and show deep grooves, the ball of the little finger loses its roundness and thickness and the hollow of the hand, especially as a result of the atrophy of the lumbricales, shows conspicuous flattening.*

In case the paralysis affects the sensory cutaneous branches of the ulnar nerve, the back of the hand will show almost exact anæsthesia of the ulnar half, in the vola manus the smaller half (the little and the ulnar side of the ring finger) (compare Figs. 14 and 15) will be affected.

FIG. 24.—POSITION OF THE HAND IN ULNAR PARALYSIS. (CASE OF PARALYTIC PARALYSIS). SIDE VIEW.

PARALYSES IN THE COURSE OF THE MEDIAN NERVE

Paralysis of the Median Nerve.—The *median nerve* supplies both pronators, and the following muscles: The flexor carpi radialis, the flexor digitor. sublimis, and the radial half of the flexor digit. profundus, the flexor pollicis longus and all the muscles of the ball of the thumb, except the adductor pollicis (ulnar nerve); also the first and second lumbricales are innervated by the median nerve. Regarding *ætiology*, what has been said of ulnar paralysis is also true here; of the excessive use or compression of the nerve in certain occupations by which median paralysis is acquired there should be specially mentioned: The paralyses of milkers, lock-makers, etc.

Radial flexion of the hand (flexor carpi radialis) is weakened, as well as flexion of the fingers in the second and third phalanges (the end phalanges by the flexor digit. prof., the middle phalanges by the flexor digit. sublimis), whereas the terminal phalanges may be flexed by the interossei (ulnar nerve) which accounts for the fingers frequently being held in the lumbricales-interosseus position. The flexion of the latter of the index and middle fingers, is partly retarded, namely the end phalanges by the action of the flexor digit. prof. (the inner half being supplied by the ulnar nerve); flexion and opposition of the *thumb* are quite impossible. *The thumb is permanently extended* (radial nerve) and adducted against the index finger (ulnar nerve). This is called "the ape hand." Pronation of the hand is naturally prevented.

If paralysis extends to the *sensory* fibres of the median nerve, anæsthesia of the vola manus occurs, from the articulation of the hand to the

finger tips, and especially in the thumb, index and middle fingers, and on the radial side of the ring finger; simultaneously the dorsal surface of the end phalanges of these fingers becomes anæsthetic, especially the summit (compare Fig. 15). Trophic disturbances: Pemphigus vesicles, exfoliation of the nails, smoothness of the fingers ("lustre fingers") is also noted in above fingers and thumb. The paralyzed muscles of the flexor surface of the forearm and the hand atrophy, especially does *the ball of the thumb lose its roundness*; in passing, it may be observed that in the pathological picture of progressive muscular atrophy just this last-mentioned thinning of the ball of the thumb is the most conspicuous symptom in the first stage of the disease.

The sensory and motor fibres of the median nerve are by no means always uniformly strongly affected in paralysis of the median nerve. The sensory as well as the motor functions in the course of the median nerve, in spite of a severe lesion of the nerve, may be retained upon one side. This apparently depends upon an *anastomosis, a communication of the median and ulnar nerves,* which allows a vicarious function of their fibres among each other.

COMBINED PARALYSES OF THE NERVES OF THE ARM

Combinations of various individual paralyses in the course of the nerves of the upper extremity may be easily recognised according to the diagnostic criteria given above. They are mostly the result of trauma due to the exposed position of the brachial plexus (luxation of the shoulder, etc.); according to the one or the other nerve trunk not being affected or but slightly implicated, the picture of paralysis in the individual case will vary greatly.

Duchenne-Erb's Paralysis.—A certain constancy in form and appearance is shown by a relatively frequent paralysis in the course of the brachial plexus; it was first described by Duchenne in children, which were born with the aid of instruments ("obstetric palsy"). Traction of the shoulders, forcible loosening of the arms, slipping of the forceps towards the neck, these are usually the direct causes of a compression of the plexus and of the paralysis in question. This later affects the deltoid, biceps, brachialis internus and supinator longus, eventually also the infraspinatus and teres minor. But other causes are also described by Erb as a source of this special form of paralysis (trauma affecting the shoulder, tumours of the neck and in the vicinity of the vertebra, neuritis, rheumatic influences). Erb was the first to give a satisfactory explanation: The fibres of the nerve (the axillary, musculocutaneous and a part of the radial nerve), that supply these muscles, enter the brachial plexus by its roots, which are formed by the fifth and sixth cervical nerves. If now a point in the brachial plexus is affected by lesion, in which these fibres still lie together, a paralysis of the previously mentioned muscles is the result (supplied by various nerve trunks) and Erb succeeded in finding a point in the neck, above the clavicle, between the scalenii in which faradaic irritation produced a general contraction of these muscles. The paralysis of the deltoid, biceps, brachialis internus and supinator longus is always most markedly developed; besides there are usually pareses in the super- and infraspinatus (suprascapular nerve), teres minor (axillary nerve), supinator brevis (radial nerve) and in the tract of the median nerve. The sensory disturbances contrasted with the motor are ill defined, especially is anæsthesia absent in the peripheral parts of the arm. According to observations conducted in my clinic and observed and analyzed by J. Müller, it is likely that the upper roots of the brachial plexus supply the *motor* fibres for the shoulder and upper arm, the two lower roots (the eighth cervical nerve and first dorsal nerve) those for the lower arm and the hand, and that this occurs without relation to the peripheral trunks of the arm; further, that the sensory nerves for the peripheral parts of the arm arise in the two lower roots, whereas the upper roots add but few sensory

fibres to the plexus (the cutaneous, axillary, and perhaps also the lesser cutaneous nerve).

Klumpke's Paralysis.—We should expect, *that in paralysis of the lower roots of the brachial plexus, atrophic paralysis of the forearm and hand,* especially of the thenar and hypothenar and of the interossei muscles, as well as *anæsthesia below the elbow joint* would be noted. It is still further characteristic that, besides these phenomena of motor and sensory paralysis, in the tract of the lower roots of the plexus *disturbances on the part of the sympathetic* arise: Myosis and narrowing of the palpebral fissure. The oculo-pupillary symptoms depend, as Klumpke has shown, exclusively upon a *lesion of the communicans nerve of the first dorsal nerve*; they are therefore entirely absent in Duchenne-Erb's paralysis.

PARALYSES IN THE COURSE OF THE DORSAL NERVES

Paralyses in the course of the muscles supplied by the dorsal nerves (the deep muscles of the back, which have the function of extending and keeping erect the vertebral column, as well as those of the intercostal spaces and abdomen) rarely occur isolatedly, as frequent as is their combined paralysis in affections of the spinal cord.

Paralysis of Extensors of the Back.—Paralytic kyphosis develops in paralysis of *the extensors of the back* upon both sides, forward bending of the body and the inability to straighten the body and hold it in this position. The vertebral column bent posteriorly and uniformly rounded, is found to be straight or may easily upon passive movements be straightened, in contrast to those cases in which kyphosis is due to disease of the vertebræ or due to muscular contractures. In unilateral paralysis, paralytic scoliosis is noted; especially characteristic is the picture of paralysis of *the extensors of the back covering the lumbar region.* Owing to the relatively great movability of the vertebral column in the region of the loins, the stiffening of the vertebral column requires special muscular action in the erect posture, principally on the part of the extensor dorsi communis. If this muscle is paralyzed, an important link is absent for straightening the body to the erect position. The body would fall forward; to prevent this the upper part of the body is bent strongly back and the abdomen is protruded forward. If the patients bend forward they fall over, due to paralysis of the muscles of the loins and can no longer assume the erect position, excepting they place their hands upon the knee and alternately bring the hands higher and higher up the thighs actually "climbing up" in this manner, until finally by bending backward of the upper body, they again reach the old equilibrium required for standing erect; the walk is waddling. In *sitting straight* the faulty stiffening of the vertebral column and insufficient balance of the trunk is shown in that the patients have a tendency to fall forward, and that the vertebral column in the lumbar region shows kyphosis.

Paralysis of the Abdominal Muscles.—The diagnosis of *paralysis of the abdominal muscles* is not difficult. If the paralysis is *bilateral*, it is noted by the following symptoms: Flaccid abdominal walls, tympanites, weakness of the abdominal press in the various expiratory acts, especially in coughing, hawking, and in defecation and so on. As the contraction of the abdominal muscles may bend the trunk forward, it is obvious that in paralysis of these muscles, the erection of the body from the horizontal position is extremely difficult. In *unilateral* paralysis, as the result of energetic expiration, the umbilicus is drawn towards the normal side, and the lateral turning of the trunk towards the side of paralysis is impaired.

PARALYSIS IN THE COURSE OF THE LUMBAR AND SACRAL NERVES

Paralysis limited to individual lumbar and sacral nerves, in the region of the lower extremity, as opposed to the markedly distributed paralyses due to central causes (especially by disease of the spinal cord) of all the nerves of the lower extremity, is of secondary clinical importance and

upon the whole, of very rare occurrence. A few diagnostic main points will be sufficient, which will denote paralyses in the course of the crural nerve and the obturator as well as of the gluteal nerves and of the sciatic nerve, respectively of the main branches of the latter. The diagnosis is based upon the observation of the absence of function of the muscles supplied by the paralyzed nerve; the sensory disturbances which usually accompany the paralysis may be easily determined regarding their distribution according to the rules laid down in the description of the sciatic nerve, and according to Figs. 19 and 20.

PARALYSIS OF THE CRURAL NERVE

This nerve supplies during its course in the pelvis the psoas muscle and the iliac muscle, after its exit below Poupart's ligament, the muscles upon the anterior surface of the thigh, except the adductors and the gracilis. Therefore, in paralysis of the nerve, the flexion of the thigh (in fixing the thigh) and the erection of the trunk (the iliopsoas, pectineus, and rectus femoris) is hindered. The *extension of the lower part of the thigh*—i. e., the extended lifting of the same anteriorly (the quadriceps)—is *impossible*. Walking, standing, in short all complicated movements of the lower extremity are hindered to a marked degree or entirely suspended, especially if a bilateral paralysis be present. In a simultaneous affection of the sensory branches, anæsthesia occurs in the lower two thirds of the anterior surface of the thigh, in the entire anterior region of the knee, and (saphenous nerve) along the inner surface of the lower thigh at the inner border along the course of the foot up to the large toe (compare Fig. 16).

PARALYSIS OF THE OBTURATOR NERVE

This nerve supplies, besides the obturator externus, the adductors and the gracilis, as well as the skin along the inner surface of the lower two thirds of the thigh up to the knee. Besides anæsthesia in the last-mentioned cutaneous area, there is seen in this very rare paralysis, besides what has been mentioned, *paralysis of the adductors*—the patients can no longer close the thighs tightly nor can they cross them (adductor muscles); the inward turning of the tibia and flexion of the knee (gracilis muscle) and further the outward rolling of the thigh (obturator externus) are but slightly impaired, as these functions are supplied by other muscles and other nerves.

The paralysis of the obturator nerve is important in so far as this nerve may be injured in other ways than the usual ætiological ones (trauma, fracture of bone, neuritis, etc.), as the head of the child *intra partum*, or an incarcerated hernia, may compress this nerve.

PARALYSIS OF THE GLUTEAL NERVES

The superior and inferior gluteal nerves arising from the sciatic plexus supply: The superior nerve, the following muscles, glutæi medius and minimus, the pyriformis, and the tensor fasciæ latæ; the *inferior* nerve, the glutæus maximus and with a branch which is not always present, the outward rotators of the thigh (the obturator internus, gemelli, quadratus femoris). The paralysis of the latter nerve besides producing difficulty in the outward rotation of the thigh, gives rise above all to impossibility in raising the lower extremity from the trunk (the principal function of the glutæus maximus) and in standing upright to fix the thighs and the ischium laterally and posteriorly. The paralysis of the superior gluteal nerve especially hinders the action of the glutæus medius and minimus—i. e., the adduction

and the rotation of the thigh inward (and outward); the function of the glutæus maximus which is assisted by the glutæus medius would also be difficult in a paralysis of the superior glutæus alone. According to this, the result of paralysis of the gluteal nerves would always cause disturbances in standing and walking; atrophy of the paralyzed muscles is usually noted at a glance.

As a rule paralysis of the gluteal nerves is only a part phenomenon in general paralysis of the sacral plexus, respectively of the sciatic plexus, the principal branch of which, the sciatic nerve, as a result of its long and exposed course, is more frequently paralyzed alone, partly completely and partly in its individual branches.

PARALYSIS OF THE SCIATIC NERVE

The sciatic nerve in its exit from the pelvis gives off branches to the outward rotators of the thigh (gemelli, quadratus femoris, etc.), further beyond in its course upon the posterior surface of the thigh, branches to the semitendinosus, semimembranosus, and biceps femoris, and divides at about the middle of the thigh into two large terminal branches, the *peroneal*, and *tibial nerves*. The ***peroneal nerve*** supplies *the muscles upon the anterior surface of the lower leg and the dorsum of the foot*, viz., the tibialis anticus, the extensor digitorum. commun. longus et brevis and the hallucis longus et brevis, and the three peroneal muscles (peroneus longus, brevis and tertius). The *tibial nerve* innervates all muscles upon the *posterior surface of the lower leg and the plantar surface of the foot*, especially the gastrocnemius, soleus, plantaris, popliteus, tibialis posticus, flexor digit. et halluc. longus and the small muscles of the sole of the foot (the interossei, lumbricales, abductor et adductor hallucis, flexor digitor et hallucis brevis, etc.). The *cutaneous area* supplied by the peroneus is the outer and posterior surface of the lower leg, as well as the back of the foot, except the borders (the inner one being supplied by the saphenous nerve branch of the crural nerve, the outer by the suralis, a branch of the tibial nerve); the cutaneous twigs of the tibial nerve find their terminal distribution upon the sole of the foot and heel (compare Figs. 19 and 20).

The phenomena arising from paralysis of the sciatic nerve and its branches, besides anæsthesia in the last-mentioned cutaneous area, are as follows:

Peroneus Paralysis.—*In an isolated paralysis of the peroneus nerve, the musculature of the anterior surface of the leg is paralytic,* eventually atrophic. *Dorsal flexion of the foot is suspended*; the point of the foot hangs flaccidly, and in walking scrapes upon the floor; this difficulty in gait is somewhat corrected by the fact that the patients flex the leg higher up and with this, lift the foot from the floor. The difficulty in dorsal flexion of the foot is due to the paralysis of the tibialis anticus and peroneus tertius, also of the extensor hallucis and of the digitor. comm. longus. *The toes can no longer be extended* (extensor hallucis and digit. comm. long., extensor digit. and halluc. brevis). The adduction of the foot and lifting of the inner border of the foot become somewhat difficult (tibialis anticus with intact condition of the tibialis posticus supplied by the tibial nerve); lifting the external border of the foot is impossible (all the peronei muscles) and also the abduction of the foot (peroneus longus and brevis), no longer especially in its position of plantar flexion.

Paralysis of the peroneus nerve is comparatively frequent not only in peripheral neuritis but also in tabes, progressive paralysis and poliomyelitis; besides trauma, prolonged forced kneeling in which the markedly tense tendon of the biceps femoris may compress the peroneus nerve against the head of the fibula, etc., give rise to

peroneus paralysis and it may also arise from a lesion of the trunk of the sciatic *in toto* and be more prominent than paralysis of the tibial nerve. Gerhardt, Jr., has lately endeavoured to give an explanation for the greater frequency of lesions of the peroneus nerve. It was shown in his experiments that the DeR, in the peroneus nerve occurs more rapidly than in the muscles innervated by the tibial nerve, that peroneus paralyses arise isolately from injury to the ganglion cells in the spinal cord, and finally that after paralysis of the muscles of the extensor side of the leg, these muscles more rapidly lose their contractility than the flexors. The assumption therefore appears to be justified, that in the course of the sciatic nerve similar conditions occur, as we have seen in the branches of the inferior laryngeal nerve in connection with the lessened power of resistance of the nervous branches leading to the posterior cricoarytænoid muscles.

Paralysis of the Tibial Nerve.—*If the tibial nerve is paralyzed, the muscles upon the posterior surface of the lower leg are paralyzed and atrophic, the "extension" primarily,* i. e., *the plantar flexion of the foot is suspended* (gastrocnemius, soleus, plantaris, tibialis posticus, with retained function of the peroneus longus and brevis) *and also the flexion of the toes* (flexor hallucis and digit. comm. long. et brevis) *and the lateral movement as well* (interossei, abductor and adductor hallucis, and abductor digit. min.).

While in the course of paralyses of the peroneal and tibial nerves contractures of the antagonists of the paralyzed muscles and secondary changes in the joint arise, there occur anomalous positions of the feet—paralytic flat-foot, point-foot, club-foot, and heel-foot.

Sciatic Paralysis.—*In paralysis of the trunk of the sciatic,* there occur, besides the phenomena of paralysis of the peroneus and tibial nerves, also the results of paralysis of the muscles supplied by the sciatic, from its exit out of the pelvis and in its course upon the posterior side of the thigh and its branches. Accordingly there is a hindrance in the outward rotation of the thigh (pyriformis, obturator int., gemelli, etc.), further drawing in of the lower leg against the thigh, and farther on, after the leg has been fixed, the raising and stretching of the trunk (biceps femoris, semitendinosus, and semimembranosus).

The diagnosis of paralysis of the sciatic nerve and its branches accordingly is not difficult, provided the anatomical relations and the innervation of the individual muscles of the lower extremity is sufficiently understood. The diagnosis becomes more simple by remembering that simultaneously with paralysis of the muscles, as a rule as the result of the lesion of the cutaneous branches of the sciatic, respectively of the peroneus or tibial nerves, localized *anæsthesia* occurs, the distribution, respectively limitation, of which gives a certain control for the correctness of the local diagnosis of the paralyzed muscles.

SPASM IN THE COURSE OF MOTOR NERVES

Preliminary Remarks.—In contrast to paralytic conditions, we understand by "*spasm*" abnormal muscular contractions due partly to physiological irritation, being an expression of the increased reaction of the motor nerves, partly to a pathological reaction affecting the nervous system. On account of the type in which spasm arises, several varieties may be noted, which must be considered in diagnosis:

Tonic spasm, i. e., more or less prolonged contraction of the muscles, in contrast to *clonic* spasm, in which contractions of brief duration alternating with flaccid conditions of the muscles occur; in the former spasm, the muscular areas affected are rigid, in clonic spasm the picture of "muscular twitching" occurs. Diagnostically, we may differentiate certain modifications of the two forms of spasm: ***Cramp***, by which we understand *severe painful* tonic muscular contractions usually of brief duration, further *cataleptic spasmodic conditions*, i. e., a widely diffused tonic rigidity of a slight degree, so that the extremities are retained by the patient in the position into which they have been easily placed by passive movement. By the expression ***cramp*** we designate conditions usually limited to individual muscles; we speak of ***convulsions*** when (clonic) spasms are distributed and are well marked. If the spasm is limited to particular muscle groups which under normal conditions produce a common action, we speak of *coordinating spasm*, and in cases in which these occur against the will of the patient and in a disturbing manner we speak of "***constrained movements.***"

The diagnosis of spasm in general gives rise to no difficulty—neither the recognition of the spasm as such, nor its seat. In the latter respect, regarding the diagnosis, all that has been said of the individual paralyses is true also of the diagnosis of spasm. More difficult to decide is the question regarding the ***origin*** of the latter, in the individual case, whether the cause producing spasm acts centrally or in the peripheral course of the nerves; whether ***directly*** or ***reflexly*** the motor nerves are irritated, and whether in the latter case an increase of irritability in the reflex arc or an absence of the inhibitory fibres produces the spasm. Unfortunately for diagnosis, we have as yet no certain differentio-diagnostic points regarding this. Especially is this true, in deciding the question whether the spasm be of a central or peripheral origin, and, speaking generally, there is still a great deal which has as yet not been satisfactorily explained relating to spasm. To describe the clinical pictures under which spasm of the individual muscles arise *in extenso*, after we have described the absence of function of the various nerves individually, would be decidedly tiresome; no doubt it will be sufficient in the following chapter to describe the most important and most frequent forms of spasm, dwelling especially upon the principal diagnostic phenomena.

SPASM IN THE COURSE OF THE CRANIAL NERVES

Masticatory Spasm.—*Spasm of the motor portion of the trigeminus, trismus.* Tonic and clonic spasms arise in the region of the muscles of mastication; in the former instance the rows of teeth are tightly pressed together (*lockjaw, trismus*), and the muscles of mastication are palpable as hard, board-like masses. If the *external pterygoid* of one side is affected alone by tonic spasm, the lower jaw will be pushed to the opposite side, as in a case that I saw not long ago, so that the lower row of teeth will be external to the upper row. If the spasm is clonic, grinding of the teeth occurs, and in clonic spasm of the masseter, temporal and internal pterygoid muscles, chattering of the teeth. The spasm of the muscles of mastication is mostly of a reflex nature, induced by irritation of the sensory fibres of the trigeminus; it arises in the course of hysteria, epilepsy, and meningitis, and is especially also an accompanying phenomenon of infectious tetanus.

Facial Spasm.—*Facial spasm* (*tic convulsif*), the most frequent of the spasms affecting the cranial nerves, involve partly the entire facial nerve and partly individual branches of the same; the spasm may show the tonic or clonic type. The *partial* spasms affect the corrugator and the zygomatic muscles (risus sardonicus), but most frequently the orbicularis palpebrarum (lid spasm). If we are dealing with a clonic form of spasm, the

eyelid will be alternately closed and opened, the result being a spasmodic blinking (nictitation); the muscles of the forehead may also take part in this process. It is usually of a reflex nature, as well as the more frequent tonic spasm of the orbicularis (*blepharospasm*), which occurs frequently in the course of various diseases of the eye and in supraorbital neuralgia, by the effect of glaring light, psychical emotions, etc. The spasm is characterized by a continued firm closure of the lids independent of the will of the patient, and may last from minutes to hours; by pressure upon certain areas ("*pressure points*") however (at the supraorbital foramen, upon the mucous membrane of the teeth, upon the mastoid process, upon the cervical vertebræ, upon the brachial plexus, etc.), the spasm may often be instantly relaxed; in other cases these pressure points are absent.

Diffuse facial spasm is for the most part *clonic* in character. In this condition, there is a frequent interchange, at one time one facial muscle, at another time another being in lightning-like contraction, so that patients affected by this spasm show the most curious grimaces. Besides the mimical facial muscles, the muscles of the palate, supplied by the facial nerve, may be affected by the spasm, as I had an opportunity of observing in a case. In the affected patient, during the spasm, the soft palate rose, and the uvula contracted almost to the point of disappearance. According to another observation of mine, the appearance of increased salivation may be the result of facial spasm. Causes of tic convulsif are cold, irritation of sensory nerves, neuralgia of the fifth nerve, trauma of all kinds, also focal affections or irritations of the cortex of the brain, especially of the anterior central convolution. In a case of Schultze's, an aneurysm of the vertebral artery, which irritated the facial trunk lying contiguous to it, was the source of a clonic facial spasm. Occasional causes for the *appearance* of the spasm are: Psychical emotions, active muscular movements, closing of the eyelids, speaking, etc. A *tonic* facial spasm is also occasionally noted in which the entire face or the affected half of the face shows tonic spasm, the tension markedly affecting the patient and producing a staring expression, the palpebral fissure becoming smaller, the forehead permanently wrinkled, and the angle of the mouth appearing drawn or displaced, etc.

Spasm of the Spinal Accessory.—*Spasm in the course of the accessorius* (*torticollis nodding spasm*). The diagnosis of this spasm is easy, in the majority of cases, as the spasm only occurs in two muscles, the sternocleidomastoid and the trapezius. The spasm is due partly to reflex causes, also directly to irritation of the spinal accessory nerve in its entire course in the brain, in the medulla oblongata, (diseases of the cervical vertebræ) up to the point of its distribution in both the previously named muscles. The clinical picture varies, according to whether the sternocleidomastoid or the trapezius are affected separately or both are simultaneously implicated in the spasm.

In *unilateral clonic spasm* of the *sternocleidomastoid* there is twitching of the head and with every contraction the head is turned to the normal side while the chin is lifted; in unilateral spasm of the trapezius the head is retracted interruptedly backward and outward, the shoulder is raised backward and upward. In *bilateral clonic spasm* the head will be drawn alternately backward and forward and will be allowed to fall back with a simultaneous rise and fall of the chin, which gives rise to the nodding movements, which however may also be produced by spasms

of other muscles of the neck, especially of the various recti capitis (see below) (nodding spasm, salaam spasm).

If there be *tonic* contraction of the sternocleidomastoid in a one-sided affection the following well-known picture will occur: *Rotation of the head and face and elevation of the chin to the opposite side, and a lowering of the ear towards the irritated side*; with this the spasmodically contracted muscle stands out as a hard cord at the neck (*caput obstipum spasticum*). In a simultaneous spasm of both sternocleidomastoid muscles, the face with the chin will be raised directly upward, the occiput retracted towards the back of the neck (only in very marked spasm can the head be brought somewhat forward and downward by a bending of the cervical vertebræ).

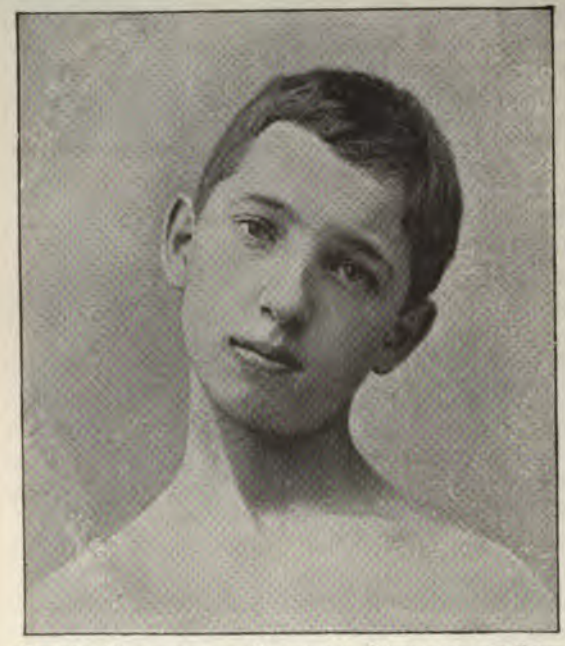


FIG. 25.—SPASM OF THE CLAVICULAR PORTION OF THE LEFT TRAPEZIUS (RHEUMATIC SPASM). THE RIGHT STERNOCLEIDOMASTOID TENSE, PROTRUDING.

In *tonic spasm of the trapezius*, if the affection be unilateral, the shoulder is raised, the shoulder-blade approaches the spinal column due to the action of the middle portion of the muscle, and is fixed by the strong muscular contraction, and eventually finally the head also drawn backward and outward (towards the affected side). In *bilateral* tonic spasm there result: Elevation of both shoulders, retraction of the shoulder-blade towards the vertebral column, and firm fixation of the same. A backward displacement of the occiput in a straight line against the back of the neck. If the spasm be limited to the *clavicular portion of the trapezius* as was already observed by Duchenne, and I myself noted in a case (see Fig. 25), there results a simple lateral inclination of the head to the affected side; with this the sternocleidomastoid of the *opposite side* springs plainly forward showing its contours (compare Fig. 25 on the right side), quite in contrast to spasm of the sternocleidomastoid, in which the muscle appears as a hard cord upon the side on which the head is inclined.

Spasm of the hypoglassal nerve, spasm of the tongue. Isolated spasms in the course of the hypoglossal nerve are rarely observed. They are characterized by unilateral or bilateral clonic contractions, rapid forward and backward drawing of the tongue, balling and dancing movements of the tongue, or by tonic contraction of individual parts of the organ. This causes difficulty in speech, in chewing, and occasionally, if the tongue is spasmodically retracted posteriorly, also difficulty in respiration. Spasm of the tongue may result from cerebral affections; especially frequently, however, it is an accompanying phenomenon of hysteria, chorea and of stuttering spasm. It may also occur reflexly in connection with neuralgia of the fifth nerve, diseases of the teeth and gums, etc. The picture of the spasm is so peculiar that a confusion with other morbid conditions is not easily possible.

SPASM IN THE COURSE OF THE CERVICAL NERVES

Spasm of the Muscles of the Back of the Neck.—Spasm of the muscles of the nape of the neck and of the arm are rare conditions and practically of very little importance on account of their extreme infrequency.

Spasm of the splenius (posterior branch of the II. cervical nerve, anterior branch of the III. and V. cervical nerve). The muscle runs along the posterior portion of the neck in the direction of the trapezius and sternocleidomastoid of the opposite side; on account of its insertion and peculiarity of position (spines of the

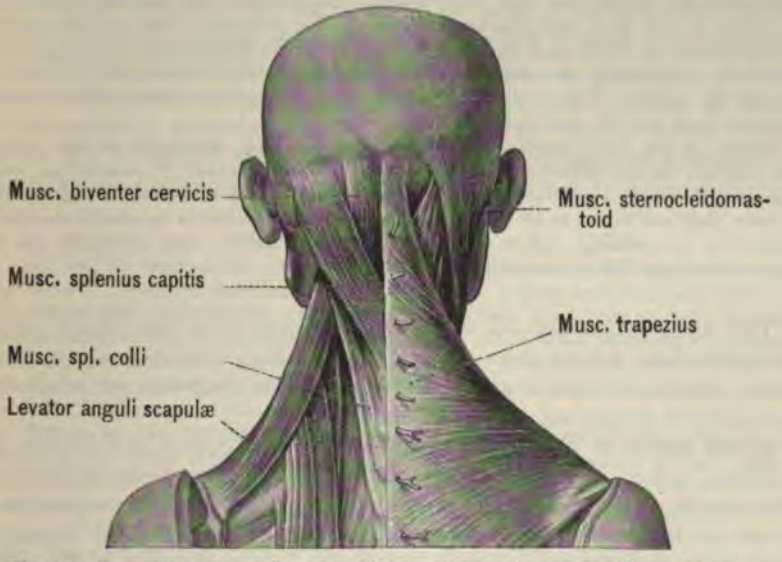


FIG. 26.—DIRECTION OF THE SPLENIUS MUSCLE COMPARED WITH THE TRAPEZIUS AND STERNOCLEIDOMASTOID MUSCLES (After Luschka).

four lower cervical vertebræ, and two upper dorsal vertebræ: Insertion into the mastoid process and the outer third of the middle oblique line of the occiput) the muscle, in regard to rotating the head, respectively of the face, is the antagonist of both of the muscles of the same side and the "partner" of the latter of the opposite side (Fig. 26). If tonic or clonic spasm occurs, the head takes the same deviating position (variety of the caput obstipum spasticum) as in spasm of the sternocleidomastoid and trapezius, so that the head inclines to the shoulder of the affected side, *the face however is rotated not towards the healthy but towards the affected side.* The contracted muscle may be felt between the sternocleidomastoid and trapezius at the upper part of the neck (Fig. 26).

Spasm of the obliquus capitis inferior (posterior branch of the I. cervical nerve). The muscle arising from the spinous process of the axis and inserted into the transverse process of the atlas rotates the atlas, besides the head, around the alveolar process of the axis, in a horizontal direction, so that the face is turned towards the opposite side; with this, however, *the chin is neither raised nor the mastoid process drawn downward.* This position is more or less permanently maintained in tonic spasm and there is resistance in the attempt at passive movements to straighten the head; in clonic spasm the head is turned towards the affected side with alternating rotatory movements (*tic rotatoire*, rotatory spasm).

Spasm of the rhomboidei (dorsal scapular nerve from the upper root of the brachial plexus, V. cervical nerve). The rhomboidei from the middle of the spinal

column running obliquely downward and outward to the base of the scapula, draw the shoulder-blades inward and upward. If these muscles are affected by tonic spasm, they lift the lower angle of the shoulder-blades and thus approximate the inner border (basic scapulæ) of the spinal column, causing the shoulder-blade to lie tightly against the thorax. The contracted muscle may be felt as a hard mass between the shoulder-blade and the spinal column.

Spasm of the levator anguli scapulæ (dorsal scapular nerve and anterior branch of the IV. cervical nerves). The muscle, running from the transverse process of the three upper cervical vertebræ downward and outward to the upper inner border of the scapula, draws in contraction the shoulder-blade, especially its upper inner angle, upward. If the muscle is spasmodically contracted the shoulder appears to be markedly raised, the neck bent towards the affected side and the muscle rises as a mass in the deep supraclavicular fossa at the anterior border of the trapezius. As this muscle is innervated by the same nerve as the rhomboidei, the spasm of the levator is occasionally accompanied with a spasm of the rhomboidei, or also occasionally by spasm of the trapezius, which similar to the levator scapulæ receives branches from the IV. cervical nerves.

Spasm of the deep muscles of the back of the neck, especially of the posterior recti capitis, of the superior oblique capitis, of the biventer et complexus, can no longer be discerned as isolated contractions of individual muscles. Unquestionably, however, they are found to be bilaterally in tonic contraction in *opisthotonus*, in clonic contraction in nodding spasm, in which latter condition very probably the recti capitis antici play the principal part.

The most important of the spasms arising from the upper cervical nerves are those taking place in the course of the *phrenic nerve* (4 cervical nerve), producing *spasm of the diaphragm*, the diagnosis of which must be considered somewhat more in detail.

Clonic Spasm of the Diaphragm.—The most frequent well-known form of clonic spasm is *singultus* [hiccough], the contractions which occur paroxysmally with a loud inspiratory sigh, affecting the diaphragm, in shorter or longer intervals, following one another and often lasting for hours, yes, even for weeks; with this there is usually pain in the epigastrium. Hiccough cannot be confounded with any other condition; the diagnosis never gives rise to difficulties; only the cause is not always easily found, as hiccough occurs in the most varied affections of the central nervous system, in psychical emotions, above all, however, produced by irritation of the nerves of the various organs, (of the stomach, of the kidneys, the uterus, etc.). In close anatomical connection with the course of the phrenic nerve (through the mediastinum, along the pleura, and the pericardium) is the presence of hiccough in pleurisy, pericarditis, mediastinal tumour, aneurysm, and (on account of the peritoneal branches of the nerve passing between the muscular fibres of the diaphragm) in peritonitis. Finally, it is also plain that singultus occurs in diseases of the liver (on account of the connection of the phrenic nerve, especially of the right, with the plexus diaphragmaticus-cœliacus, from which several branches go to the liver), and in diseases of the stomach and intestines (on account of the supply of both these organs with branches from the plexus cœliacus, to which the left phrenic nerve gives off branches), as complication.

Tonic Spasm of the Diaphragm.—Much rarer, but of more importance on account of its consequences, is *tonic* spasm of the diaphragm. The

diagnosis is not difficult: The lower part of the thorax is expanded, remains immovable during respiration; the epigastrium protrudes, whereas the upper parts of the thorax execute forced and increased respiratory movements. The effect of the latter is however insufficient; rapid cyanosis will gain the upper hand, and danger of suffocation appears. If the pulmonary borders are percussed, they are found to be displaced downward and are immovable; the cardiac borders are pressed downward. Tonic spasm of the diaphragm may be differentiated from an attack of bronchial asthma by the existence of the difficulty in expiration, the sibilant râles especially, the diminution in cardiac dulness, and the excursions of the diaphragm, which still occur, even if weak; whereas cyanosis and dyspnœa, with their results upon the pulse, are common to both conditions. The diagnosis gains in certainty if the attack occurs in the course of muscular or articular rheumatism, of tetany (in the course of which I lately saw a marked example), in tetanus, in epilepsy or hysteria—conditions which according to experience may give rise to tonic spasm of the diaphragm.

By way of supplement, the occasional spasmodic so-called "abnormal respiratory movements" shall be mentioned: *Sneezing spasm, crying and laughing spasm, yawning spasm* and *cough spasm.* The diagnosis of these conditions is easy, the detection of the cause, however, more difficult; as a rule, in all these varieties of spasms, hysteria is the underlying condition, but rarely are they due to anatomically demonstrable lesions of the central nervous system. As reflex phenomena these spasms may be noted in diseases of the uterus, helminthiasis, etc.

Spasm in the Course of the Nerves of the Brachial Plexus.—Spasms in the course of the lower cervical nerves, in the nerves of the *brachial plexus,* arise in the most manifold forms, at one time widely spread, at another time localized in this or that muscle. They are partly of peripheral, partly of central, origin; in one of my cases, a cerebral abscess showed itself primarily, appearing as a tonic spasm in the arm. To mention and describe the various spasms individually, is unnecessary, the diagnosis offers no difficulty if the innervation and action of every individual muscle is clearly remembered—conditions which were especially emphasized in the diagnosis of paralysis, and on this account we may refer to this special chapter.

However, on account of their practical importance some few spasmodic conditions shall be mentioned, in which groups of muscles performing complicated movements of the hand, act coordinately and which may be spastically affected. These are the so-called "*occupation neuroses*"; the best known and most frequent of which is writer's cramp.

Writer's Cramp.—As a rule, this cramp develops after prolonged writing during the stage of exhaustion while writing; later on the mere handling of the pen is sufficient to produce spasm. With this, tonic and clonic spasms of individual muscles which are utilized in writing, especially of the thumb and index finger, occur in a disturbing manner: Spasmodic flexion of the index finger and opposition of the thumb, spasm of the pronators and supinators, the extensors and flexors of the hand, etc. Also spasm in the muscles of the shoulders may inhibit the continued use of the pen in spite of all attempts on the part of the patient to control the same. The muscles of the fingers may be free from spasm, even if it is a rule that in writer's cramp the spasm begins there. Spasmodic contractions of this or that muscle group affecting the regular combined action

of the muscles utilized in writing cause an altered handwriting in many ways. It is deformed by strokes and hooks or becomes wave-like on account of the superadded *tremor,* or it may become angular and finally entirely illegible. From this spasmodic variety of ***mogigraphia*** (***graphospasm***) must be differentiated that form which arises through weakness and rapid tiring of the affected muscles and by pain in the arm, producing an inability to write (*paralytic mogigraphia*).

While the muscles refuse service in writing in the most marked manner, the individuals affected by writer's cramp are usually able to perform grosser movements and work with the muscles of the hand and arm faultlessly. In other cases there may even be weakness of the muscles in these acts, and especially other fine movements of the hand, such as sewing, etc., are also impossible. The electrical contractility of the muscles used in writing is sometimes normal, occasionally quantitatively, or even qualitatively, altered. Besides the already-described disturbances in the motor area there occur at times in the affected extremity also pains, formication, etc.

Other Co-ordination Spasms.—Similar to graphospasm in persons that write very much, there is also noted in *piano-players* or *violinists* and others, spasmodic contraction in the handling of their instruments. In the co-ordinated movements of the muscles, besides a sensation of tiredness, pain in the shoulder, etc., occurs. There also arise in *telegraphers, painters, sculptors, in smiths,* in the excessive use of the muscles in their special work, in persons that have to do with milking ("*milker's cramp*") functional weakness and (usually tonic) spasms in the muscles which are especially utilized in the various occupations. These disturb the co-ordinated common action of the muscles used for the intended purpose, partially or entirely. The anatomical seat of this nervous disease is still unknown, and is still a question of hypothesis.

The recognition of the previously mentioned affections as occupation neuroses rarely offers any difficulty. It is true, in various cerebral and spinal affections, as well as in some individual neuroses, in multiple sclerosis, in progressive muscular atrophy, in general tremor, chorea, paralysis agitans, etc., disturbances arise in writing and in the finer movements of the hand, which may remind us of the clinical picture of the occupation neuroses. But the circumstance that the affected co-ordinated action of the latter, as a rule, is exclusively hindered and that, on the other hand, the disturbance of these same muscular actions in other morbid conditions is only a minor link in the general symptom-complex, usually at once points to their proper recognition.

SPASM IN THE COURSE OF THE LUMBAR AND SACRAL NERVES

Isolated spasms in the course of the lumbar and sacral nerves and the muscles supplied by them, are as a rule quite rarely observed: Psoas spasm as a result of caries of the lumbar vertebræ and coxitis, spasm of the glutei, of the adductors, of the quadriceps, in the muscles supplied by the peroneus nerve, etc., have been variously described, usually due to hysteria and tetany, further, as an accompaniment of paralyses of the antagonistic muscles, or reflexly. They may be easily recognised

according to the diagnostic rules which were described under Paralysis, regarding the innervation and function of the individual muscles.

Spasm of the Calf of the Leg.—A relatively frequent spasm, in the course of the tibial nerve, is spasm of the *calf of the leg.* This spasm which is of short duration, repeating itself rapidly, being tonic and combined with marked pains ("cramp"), in the musculature of the calf of the leg, usually occurs at night, especially after exhaustion in running, dancing, etc. Besides, spasms of the calves of the legs also occur in the course of sciatica, perhaps also due to "varicose veins," further, in the course of diseases of metabolism and circulatory disturbances, in cholera and diabetes. As a rule the spasm disappears rapidly after a few hours or minutes, if another position is taken by the leg, which under normal circumstances is combined with a relaxed condition of the muscles of the calf of the leg. Tonic spasms of the leg of longer duration occur in tetany or as contractures, the result of paralyses in the course of the peroneus nerve. In the latter case, there also occurs permanent marked plantar flexion with flexion of the toes, the *pes equinus*, as, *vice versa*, in paralysis of the muscles supplied by the tibial nerve, those innervated by the peroneus, the antagonists which produce dorsal flexion, become contracted and show *talipes calcaneus* position.

NEURITIS, MULTIPLE NEURITIS

Incidentally in the description of the diagnosis of neuralgia, paralysis and of spasm, neuritis has been mentioned in the ætiology of these affections of the nervous system. Until recently it was supposed that this was not a very frequent affection; in the last few years, however, neuritis has been better studied and it was found that this disease has a more individual character and occurs more frequently—especially as multiple neuritis—than was formerly supposed. According to my opinion, there is dainger that the limits of neuritis may be too greatly extended and many conditions which have as yet not been cleared up will be acknowledged without positive proof as being due to neuritis. It must therefore be our province closely to encircle the region of this affection and to determine as precisely as possible the phenomena characteristic of neuritis.

Neuritis is a substantive affection of the nervous system, primarily running an acute or chronic course, as an inflammatory disease of the peripheral nerves in a more or less widely disseminated manner (multiple or circumscribed neuritis), leading to degeneration of the peripheral nerve fibres and not associated, or at least only secondarily, with minor changes in the central nervous system.

Circumscribed Neuritis.—If we examine primarily the symptoms which are of value in the diagnosis of *circumscribed neuritis,* it will be seen that the morbid picture will vary, according as to whether a motor, sensory, or a mixed nerve has become the subject of inflammation in its peripheral course.

If a *sensory* nerve is the seat of neuritis, this will be seen by changes in the reaction in the course of the affected nerve especially by irritative phenomena. These consist in usually spontaneous (especially in the acute form of neuritis) very marked *pains* which are most intense in the distributionary area of the inflamed nerve, and in contrast to the attacks of pain in neuralgia, which occur in paroxysms, are more continuous, but may be increased by external influences, especially by pressure upon the inflamed nerve trunk. Besides the *sensitiveness on pressure* and *the pain, hyperæsthesia* may occur, and comparatively very early, possibly even after a few days, *anæsthesia* may occur in the affected cutaneous areas (*anæs-*

thesia dolorosa). Anæsthesia may be absent provided the neuritis is moderate, or the conduction of the sensory irritation is assumed by anastomosing nerves. As the sensory nerves also contain ***vaso-motor*** fibres, it is clear that, in the course of neuritis, pallor, erythema, œdema of the skin, swelling of the joints, and especially the eruptions of herpes, and pemphigus may occur in the chronic forms of neuritis. There may also be a fissuring and desquamation of the epidermis, disturbance of nutrition of the nails and falling out of the hair. Also, apparently spontaneously arising gangrene of the extremities, etc., has been noticed as the result of neuritis.

The affection of the *motor* fibres is characterized by ***paralysis***, which may be preceded, in rare cases, by irritative phenomena (spasm, contractures). The paralytic phenomena obviously show, according to the intensity of the inflammatory process or of the compression of the motor-nerve fibres, the most varied degrees, from a slight tired sensation up to complete paralysis. The latter condition is characterized as a peripheral paralysis from the fact *that the paralyzed muscles atrophy* and show the picture of *flaccid paralysis*. Then the other symptoms of severe lesions of the motor apparatus are also present: The various forms of DeR, and the disappearance of the reflexes.

The previously mentioned phenomena are combined in neuritis of mixed nerves. There are also added symptoms which may be determined by inspection and palpation, which are in connection with the anatomical changes in the inflamed nerve: The *swelling of the nerve trunk* and the reddening of the skin in the course of the same. The more important of these two symptoms is the swelling. This may be absent in a part of the cases, especially in those forms of neuritis which do not arise from the perineurium (*perineuritis*) or from the interstitial tissue (*interstitial neuritis*), but which is limited to the nerve fibres themselves ("*parenchymatous*, degenerative neuritis"). If the swelling of the nerve trunks can be recognised, this forms an important diagnostic mark of neuritis. The thickening, especially by perineuritic changes, is sometimes continuous, so that the inflamed nerve may be felt as a uniformly thick band; sometimes it is interrupted, so that only individual parts appear thickened (*neuritis nodosa*). Fever is inconstant, and if it is present, it varies greatly in intensity as a symptom of circumscribed neuritis.

Multiple Neuritis.—Whereas formerly circumscribed neuritis was only known as the result of trauma, or due to the action of pressure or as the effect of the continuation of an inflammation to the nerve from inflamed contiguous organs, it soon became known that in many cases neuritis was a consequent condition of various infectious diseases (enteric fever, diphtheria, etc.), at one time arising in the form of a circumscribed neuritis, but also as a "multiple" neuritis, being more or less *distributed* and affecting the entire nervous system. In a case which has become celebrated, Duménil in 1864 showed that the peripheral nerves under some circumstances may *primarily* and *en masse* be the seat of degeneration. The affected patient showed *intra vitam* rapidly developing atrophic paralyses of the extremities and sensory disturbances and died after a few months of illness; at the autopsy there were no anatomical changes in the spinal cord or in the nerve roots. With this and by similar observations made after that time the proof was furnished that a generalized neuritis (*polyneuritis, multiple neuritis*) occurs, without affecting the

spinal cord, in spite of the generally distributed paralyses, and that the clinical picture of such forms of polyneuritides resembles certain diseases which up to that period were believed to be diseases of the spinal cord, such as poliomyelitis anterior and Landry's paralysis. Our information regarding multiple neuritis was markedly increased by Leyden, whose labours added especially in awakening an interest in the affection, particularly in Germany, and that, upon the basis of his investigations and since then also of those very numerous investigations of other authors, we have attained an insight into the origin and nature of polyneurites.

Ætiology.—Especially in an *ætiological* respect a very great amount was gained. The origin of the disease, by the action of the various acute and chronic *infectious diseases* (diphtheria, angina follicularis, influenza, enteric fever, scarlatina, measles, variola, sepsis, puerperal fever, acute articular rheumatism, pneumonia, tuberculosis, syphilis, etc., especially also by leprosy and beri-beri) is unquestionably determined. Further, it may be looked upon as certain that a cause of neuritis is *alcoholism*, which is frequently in connection with neuritis, and also other *intoxications* (poisoning by lead, copper, phosphorus, arsenic, mercury, ergot, carbonic-acid gas, etc.), and finally certain *constitutional diseases* (diabetes, carcinoma, gout, anæmia), *chronic endarteritis* and the *puerperal state*. In some of the cases, it appears that several of the previously mentioned causes acting at the same time and together produce neuritis, for example alcoholism and diabetes mellitus, etc. Also in the course of *tabes*, neuritic affections were found in the peripheral nerves; in the latter connection it has become likely that the affection of the peripheral nerves and the posterior columns is due to the same cause which damages the peripheral neurons sometimes exclusively centrally, at other times exclusively peripherally from the spinal ganglion cells, occasionally at both places simultaneously, and in all these instances producing in general similar morbid pictures. In a part of the cases the influence of the causative deleterious effect cannot be determined. In such cases we must recur to severe *refrigeration* and *overexertion* (at least as predisposing causes) or to the action of *certain infectious products the nature of which is not known exactly* and which must be assumed, showing themselves singly and alone or at least principally by giving rise to a generalized neuritis. For the correctness of the latter assumption the local and timely increased origin of "spontaneous" neuritis and the occurrence of the disease among several persons living under the same external conditions, is favoured. Finally, as the result of clinical investigation in regard to the causation of neuritis, it has been observed that not all cases of *poliomyelitis acuta et chronica* or even of *progressive muscular atrophy*, etc., as was at first supposed, may be referred to peripheral polyneuritis, but that the realm of the above-mentioned diseases should be considerably decreased and, in its place, quite a number of cases of polyneuritis must be assumed. An exact fine differentiation of primary degeneration of the peripheral motor nerves from diseases of the cells of the anterior horns is no longer correct, since we know that the ganglion cells of the anterior horn as well as the peripheral motor fibres are parts of the peripheral motor neurons and organically belong together; and the same deleterious effects may damage each individually or both simultaneously (compare Differential Diagnosis). We shall attempt in the following description to explain the clinical picture of multiple neuritis as precisely as possible and also to define the differential diagnosis as sharply as is consistent at this time. But naturally a clinical picture of multiple neuritis which shall meet all cases cannot be described, as the distribution and development of the neuritic process gives rise to all possible deviations from the typical picture, if I may so call it, of the well-developed disease. Nevertheless, the latter will at least serve as a guide in determining the diagnosis.

The symptoms of multiple neuritis with which we are concerned are in the main not different and cannot be different from those which have already been mentioned in the diagnosis of circumscribed neuritis.

Sensory Disturbances.—*Sensory disturbances* of all kinds are found in this affection: drawing pains which may be continuous or occur

paroxysmally being especially severe and localized in the peripheral parts of the extremity, paræsthesia arising spontaneously or upon pressure, *hyperæsthesia* and anæsthesia. In the latter respects various modifications of sensory paralysis have been observed: Retarded sensory conduction, latent pain sensation, partial sensory paralysis; symptoms, which as was formerly mentioned, in general favour the central character of the anæsthesia in the individual case, but which may also occur in neuritis, provided the sensory nerve trunks which are contiguous and supply different functions are partially affected by the neuritic process, or if the intensity of the affection varies in the individual nerve fibre. Very rarely are sensory disturbances *entirely* absent, and upon post mortem in such cases the degeneration is also missed in the cutaneous twigs of the nerve. It is also true that sensory alterations in general are subordinate to the *motor* disturbance, and the severe sensory irritative phenomena that appear at the onset of the affection, as a rule disappear rapidly.

Motor Phenomena.—Motor paralyses may be ushered in by sensations of weakness, stiffness, trembling, and spasm in the calves of the leg. These symptoms, however, increase rapidly to more or less well-developed *paralyses.* Especially is the lower extremity attacked frequently and early; the course of the peroneus nerve particularly shows the signs of paralysis; in the upper extremity, the extensors of the hand are predisposed. In the majority of the cases only the *spinal* nerves are affected; in a smaller number, however, neuritic affections also occur in the course of the *cranial nerves,* and disturbance of function has been observed in every one of the cranial nerves from the optic nerve to the hypoglossus; the details of which, however, need not be considered here. Special mention, however, must be made of the rare occurrence of optic neuritis in the course of multiple neuritis as well as certain symptoms depending upon a neuritic affection of the vagus: The *increased pulse frequency* with or without cardiac palpitation, dysphagia, dyspnœa, spasm of the larynx, etc. By changes in the phrenic nerve difficulty in respiration may also arise (in these cases parenchymatous degeneration of the diaphragm has been found post mortem), and this may be the cause of the fatal termination.

The character of the paralysis is that of the "*flaccid*" type, in the course of which the *paralyzed muscles atrophy.* In keeping with this *the electrical reaction of the muscles changes,* in general in the sense of *reactions of degeneration.* However, we would be greatly mistaken, if we should expect, in all cases, the fully developed signs of the reactions of degeneration; on the contrary, there are found all possible changes of the electric contractility in a quantitative and qualitative respect, varying from the normal up to the complete DeR; it is interesting to note that frequently in spite of the continued full possibility of function of the nerves, DeR have been noted. With the obliteration of the sensory and motor tracts, a diminution or *disappearance of the reflexes,* especially of the tendon reflexes is to be expected. In fact as a rule the patella tendon reflex is absent; if they are exceptionally retained or even increased, it must be assumed in such cases, as appears very likely from observation, that the affected reflex tract has not been attacked by the neuritic process,

or has only become partially diseased, so that the tendon reflexes are retained, or perhaps by a simultaneous functional disturbance in the spinal cord even may appear as exaggerated. Besides paresis in the extremities, there has been observed, especially in the course of alcoholic neuritis, but also in other forms of neuritis, especially of infectious origin (after enteric fever, sepsis, and above all also after diphtheria, etc.), *ataxia.*

Occasionally ataxia becomes more prominent; especially is this the case in drunkards in whom there may develop acutely or more gradually a morbid picture that resembles true tabes dorsalis (*pseudo tabes of alcoholics*). But also in previously healthy persons a neuritic ataxia may occur, without a determinable cause and occasionally without any disturbance of sensation and without giving rise to paralysis ("primary acute ataxia"), a form of ataxia which in contrast to that occurring from central changes is usually susceptible of *cure.*

Disturbances of Co-ordination.—The unquestioned occurrence of exquisite *disturbances of co-ordination* in a purely *peripheral* nervous disease is interesting from a theoretical standpoint and its explanation gives rise to some difficulties. The process of co-ordination, i. e., the orderly simultaneous activity of certain muscle groups, to produce a common action, which shall be uniform seems *a priori* impossible without participation of the central nervous system;[1] in fact lesions of certain tracts in the latter are constantly combined with disturbances of co-ordination. If now in cases of neuritis, which is a peripheral disease of the nervous system, without any anatomical changes in the brain or spinal cord, marked ataxia occurs, this can only be explained in the manner that the peripheral nerve fibres leading to the central apparatus of co-ordination or being given off from the same have partly suffered in their property of conduction, this giving rise to the difficulty in co-ordination. It is clear, that in unilateral disturbances of the sensory as well as of one of the motor tracts (it being understood that in the latter only a partial and not complete loss of conduction has occurred) ataxia may be produced. In fact *both are peripheral forms of ataxia,* as I should like to call them, the sensory as well as the motor, which have been observed by competent investigators in the course of neuritis. We shall first consider the *peripheral sensory form of ataxia* of which Dejerine has lately communicated an excellent example. In this case the centripetal influences acting in the central organ by motor irritation (upon the peripherally damaged sensory tracts which are unilaterally affected in such cases of neuritis) arrive in insufficient amounts in the intact central apparatus of co-ordination, and therefore the regulation of the innervation of the motor fibres suffers. If therefore in other cases of neuritis no noteworthy disturbances of sensation can be observed, sensory irritation therefore being nearly or quite normal, the uncertainty of the movements must be looked for in the fact that the innervation of the motor fibres occurs from the central organ in a uniformly graded intensity, in keeping with the intended action, whereas the conduction of the irritative innervation takes place peripherally by the way of the muscle, here and there having suffered stronger or weaker interruptions, so that the end effect, the harmonious common action of the individual muscles appears difficult or is impossible. As the paralyses in the cases in which ataxia was observed, were not complete, and from an anatomical standpoint, to which especially Minkowski called attention, the individual bundles of the neuritically affected nerves showed marked variation, it appears to me that the explanation of these ataxias as being *peripheral motor conduction ataxias,* is unobjectionable. That the absence of the regulating optical influences (showing themselves by the Romberg symptom) allows such peripheral, especially sensory, conduction ataxias to appear increased, is self-evident.

[1] For details regarding the process of co-ordination in the brain and spinal cord see farther on.

Of *trophic disturbances*, respectively ***vaso-motor changes,*** the most manifold forms have been observed in neuritis: From simple red spots upon the skin, urticaria and herpes, up to ulcerations and gangrene, further cutaneous œdema, changes in the hair, hyperplasia of the subcutaneous cellular tissue, *enlargement of the joints,* etc. From the implication of the cardiac and cœliac plexuses, the ***visceral crises*** may be made to depend which may occur in the course of neuritis and now and then appear prominent in the morbid picture. ***Oliguria*** which is occasionally noted at the onset of the disease is perhaps also due to an irritation of the sympathetic, especially of the splanchnic nerve; in some cases *hyperidrosis* has been observed.

Rarer Symptoms.—Finally it must be especially emphasized that, besides the paralyses of the extremities and those in the course of the cranial nerves, even if rarely, *disturbances of the function of the bladder and rectum,* also impotence, have been observed in cases which were certainly well-characterized examples of neuritis. This fact is in so far of diagnostic interest, as these morbid phenomena are of much more frequent occurrence in affections of the spinal cord than in the course of peripheral diseases of the nerves. The explanation of their appearance offers no difficulties in such a markedly *disseminated* degenerative process of the nervous system, as occurs in neuritis. As soon as the pudendal nerve or the sensory and motor-nerve fibres of the urinary apparatus, which run into the posterior sacral nerves and produce the sphincter reflex, are affected to a marked extent by neuritis, and are paralyzed, incontinence must be the invariable result. Farther on it will be mentioned that if the neuritis is progressive, affecting the nerves one after another, especially those concerned with the acts of deglutition, production of the voice, of speaking and of chewing, there develops, as has been variously noted, *the picture of bulbar paralysis,* without changes being found post mortem in the medulla oblongata.

Lately, peculiar *psychoses* have been observed on several occasions in the course of polyneuritis. Especially, the form first described by Korsakow, which is relatively frequently associated with peripheral neuritis, showing itself in a loss of the faculty in regard to locality and to time, in a weakness of memory affecting especially the latest past, and in many illusions of memory, and which has been designated as "*polyneuritic psychosis.*" This form is obviously the effect of certain intoxications and of infections, which in the majority of cases lead exclusively to multiple neuritis, but in some cases also give rise to a specific toxic reaction on the part of the brain. This latter condition may exist alone, or as is usual, be combined with polyneuritis.

Onset and course of the disease occur partly gradually, creeping, sometimes acutely, with symptoms which remind us of the onset of an infectious disease, with *fever* up to 105° F., vomiting, vertigo, diarrhœa, jaundice. With this, phenomena occur which point to an implication of the nervous system: Pains, spasm in the calf of the leg, etc. An initial symptom-complex that commonly precedes the severe phenomena for some time in alcoholic neuritis, consists in coldness, pallor, numbness, formication, etc., in the fingers and toes, to which are added, later, the paralysis,

especially in the distribution of the radial and peroneal nerves. The duration of the disease varies greatly—from a few weeks up to ten years and longer.

Differential Diagnosis.—As easy as it is, upon the basis of the described symptom-complex: The pains and hyperæsthesia with rapidly following anæsthesia, the trophic disturbances, the flaccid paralyses with changed electrical contractility of the nerves and muscles, the absence of the reflexes and the conspicuous wasting of the muscles, finally the eventual well-developed swelling and sensitiveness upon pressure of the inflamed nerves, to make a diagnosis of *circumscribed neuritis*, so difficult it may become in an individual case, to diagnosticate with certainty a *polyneuritis*. This disease shows itself as a result of the diffused distribution of the process, usually in such manifold morbid pictures that confusion with diseases of the brain and especially with certain affections of the spinal cord is easily possible, and in some instances absolutely unavoidable.

Anterior Poliomyelitis.—The latter is especially true of *anterior poliomyelitis*. As the anterior horns of the spinal cord represent the first points of origin of the peripheral nervous system, and the cause giving rise to a neuritis may affect the most varied parts of the peripheral neurons at the most external end of the same, in the *muscles* as well as farther upward in the trunk, up to the roots and ganglion cells, it is self-evident that occasionally the anterior horns may be alone and primarily affected by the process, so that *anterior poliomyelitis naturally belongs to the category of neuritic affections*. The degeneration of the peripheral nerves and muscles observed in cases of poliomyelitis and the succeeding wasting of the muscles are obviously secondary phenomena. However, according to the principle of division which exists in nosology and in diagnosis (compare also p. 431), poliomyelitis is to be separated from peripheral neuritis, and we must put the question, whether it is possible, in the individual case, diagnostically to differentiate poliomyelitis from multiple neuritis. In fact, in a great majority of cases this is possible. As the anterior horns have nothing to do with sensory conduction, *an exclusive affection of the anterior horns is to be excluded in all cases in which sensibility appears to be disturbed; and this is true in by far the majority of cases of neuritis (at least at the onset, in the form of sensory irritative phenomena)*. If, however, in rare cases of neuritis, only *motor* disturbances are present—flaccid paralyses with DeR, diminution of the reflexes (which may be due also to exclusive affection of the sensory tracts), the absence of sensory disturbances takes from the differential diagnosis its best points of support.

It might be supposed that peripheral neuritis, at least in those cases in which the spinal nerves are affected, would always show sensory disturbance. Clinical experience however has shown that this is not true of all cases of neuritis, sensory disturbances, moreover, may be absent entirely so that we are forced to assume that, in these rare cases, the exciting cause of the neuritis has electively damaged the motor fibres. In these cases, a differentiation of anterior poliomyelitis from this purely motor neuritis could only be made in that the former muscle groups which

are functionally associated are exclusively affected (corresponding in this respect to the existing arrangement of the groups of ganglion cells), in neuritis, however, the muscles are affected independently of their functional kindred action receiving their innervation through individual nerve trunks. But even this differential point has not shown itself to be conclusive, in that, quite apart from affections of the roots, also in varieties of neuritis, which affect merely the peripheral nerves, muscles functionally related have been found affected, other muscles supplied by the same nerve trunk may be spared from disturbance of their function, so that here even an elective damage of the individual peripheral motor nerve fibres must be thought of, due to the exciting cause of the affection.

The cases of exclusive disease of the motor fibres or even of motor fibres that supply muscles functionally associated, are very rare. To make a differential diagnosis between *this* form and poliomyelitis with *certainty* I regard as impossible, according to the present condition of our knowledge.

Landry's Paralysis.—Still less is a positive opinion to be given regarding the question whether and in how far a polyneuritis running an acute course may be differentiated from acute Landry's paralysis. We refer, in regard to this, to the description of the diagnosis of the latter in the chapter upon Diseases of the Spinal Cord, and shall only say now that the exciting cause of Landry's paralysis, in some cases, may show its effects particularly as a polyneuritis, without giving rise to any affection of the central nervous system, and when this is the case, a differentiation between acute ascending paralysis and polyneuritis in general, is no longer possible.

Tabes Dorsalis.—*Polyneuritis* may be more easily differentiated from *tabes dorsalis.* The latter affection must be considered in a differential respect, in the sensory and especially in the ataxic form of neuritis. Common to both processes are the pains, sensory disturbances, the visceral crises, the ataxia, the disappearance of the tendon reflexes, and the paralyses of the muscles of the eyes. Very rare in neuritis is the characteristic reflex rigidity of the pupils which occurs so commonly in tabes; also the bladder disturbances are usually absent in neuritis. In fact the differentiation of both diseases is not difficult in by far the greatest majority of cases. The clinical picture of tabes is a well-rounded, typical one, and the differentio-diagnostic difficulties are artificially constructed, due to the appearance of certain symptoms in neuritis (myosis, rigidity of the pupils, etc.), which rarely occur in practice. In *favour* of neuritis are: *The absence of the reflex rigidity of the pupil and the girdle sensation, the sensitiveness upon pressure of the nerves and muscles, the prominence of weakness or even paralysis of the muscles* in contrast to the well-preserved muscular power in tabes, *the rapid and early development of paralytic conditions, the amyotrophia, the changes of the electrical contractility* (the appearance of DeR in a wide sense), *the often transitory absence of the patella tendon reflex and the special variety of the gait,* differing from the ataxic gait of tabes. Whereas the patient, affected by tabes, throws his legs forward with a superfluous extension at the knee-joint and brings the foot down with a certain amount of force, the individual affected by ataxic neuritis raises the leg high up at the hip-joint whereas the lower leg remains bent and allows the foot to fall to the floor, which gives the gait a somewhat poking character, which may be referred to a

paralytic condition which is so frequent in neuritis and occurs early in the distribution of the personal nerve. The diagnosis becomes difficult if, in the later course of tabes, paralyses develop which owe their origin partly to a propagation of the process to the anterior parts of the gray substances of the spinal cord, and partly to a complication with peripheral neuritis; in both instances DeR are present. Whereas the fundamental symptoms of tabes continue, the neuritic paralysis may be retarded and in this manner show its nature as a *complication* of tabes. Finally, it must be mentioned that *optic neuritis* may occur in the course of multiple neuritis, which however in the case of tabes is extremely rare. The prominence of vaso-motor disturbance, the psychical alteration, the relative rarity of bladder and rectal disturbances, etc., in neuritis, in contrast to tabes, are uncertain differentio-diagnostic points of support. The circumstance, that in neuritis (especially in alcoholic neuritis, by withdrawal of the alcohol) frequently improvement may occur in the symptoms, in tabes, on the contrary, the process advances, in spite of every therapeutic measure, even if remissions may occur, nevertheless, it shows an essentially progressive character, this fact may be of advantage in the differential diagnosis.

Syringomyelia.—The rare affection *syringomyelia* may give rise to differentio-diagnostic difficulties on account of some symptoms being common to both affections, such as the vaso-motor disturbances, the anæsthesia, the amyotrophia with DeR, and disappearance of the tendon reflexes. However, in the case of syringomyelia, the sensory irritative phenomena, which are so characteristic of neuritis, the sensitiveness upon pressure of the nerves and muscles is absent, and on the other hand, the almost pathognomonic type of paralysis of sensation in syringomyelia (at least at the onset), analgesia and thermo-anæsthesia alongside of a relatively better-retained tactile sense—even if present in polyneuritis (if in rare cases partial perception paralysis occurs) is never so purely developed as in syringomyelia. To enter more minutely into the differential diagnosis of the latter affection before its diagnosis has been described is not advisable. Regarding the details, in the differential diagnosis of neuritis and in the individual diseases of the spinal cord, these will be found in the chapter relating to the latter.

It must still be mentioned that an important aid in the diagnosis of neuritis consists in the *ætiology*. This may be very marked, although I do not believe it proper to describe individual forms of the disease, from an ætiological standpoint (alcoholic, infectious, toxis, etc.) and to separate them from each other symptomatically—the diagnosis of neuritis gains nothing by this! But the proof that a debauch, an infectious disease, a lead intoxication, etc., preceded the onset of the disease, often gives additional support to the diagnosis of a polyneuritis. This is also of value from the *therapeutic* standpoint, as neuritis, in contrast to similar morbid conditions occurring in diseases of the central nervous system, is susceptible of cure.

DIAGNOSIS OF DISEASES OF THE SPINAL CORD

THE diagnosis of the individual diseases of the spinal cord has been improved and extended in an unprecedented manner in the last three decades. From the formerly recognised morbid pictures numerous symptom-complexes have been separated as distinct diseases of the spinal cord; whether correctly must remain undetermined for the present. In the description of the diagnosis of polyneuritis, we have seen how careful we must be in the interpretation of certain morbid pictures as diseases of the spinal cord. But also in general must we, starting from the principle that only definite and distinct pathologico-anatomical changes, which may be controlled and accurately limited, shall be the standard for the recognition of substantive affections, always remember the fact that particularly in the boundary of diseases of the spinal cord the necessary reserve has by no means always been observed in diagnosis.

During the usually slow course of affections of the spinal cord which frequently last for decades, a certain number of cases pass from the observation of one physician to that of another; the diagnosis is made according to the method in vogue to-day, frequently having no opportunity to control the correctness of the diagnosis by autopsy, and content ourselves with the thought that there is no doubt about the accuracy of our diagnosis. However, also those cases of diseases of the spinal cord upon which an autopsy is held, and in which during the life of the affected patient the symptoms were observed with care for a long time, and the examination conducted with all the knowledge available at the time, even such cases are only calculated to increase our diagnostic knowledge in general to a slight degree. It is no longer sufficient at this time to determine the macroscopico-anatomical changes in the various cross sections of the spinal cord; such findings have scarcely any value, regarding the control of the diagnosis and in the setting up of general diagnostic conclusions. Moreover, in every individual case the process must be investigated minutely, from the stage of its development and distribution, and the microscopic examination of numerous cross sections, prepared according to the best methods in use to-day—by one especially skilled in work of this kind—should precede the final decision of the case in question. But even in the fulfilment of all of these requirements, the sceptical diagnostician will have to admit in not a few cases that the often very complicated anatomical changes found in the spinal cord are not *exactly* in keeping with the symptoms which he had assumed during the life of the patient as the substratum on the part of the spinal cord for the functional disturbances. Under no circumstances is it allowable to content one's self at the autopsy that, *among other* portions, also those parts of the spinal cord were found affected which were assumed to be pathologic during the course of the disease, in cases in which simultaneously other areas of the spinal cord are diseased which according to the

diagnosis were expected to be intact. And the latter must be allowed to be by no means a rare occurrence, as every one will admit who is not prejudiced by his own opinion and awaits the impartial results of the autopsy.

If our knowledge were farther advanced regarding the course and the significance of the special fibre tracts in the spinal cord, our diagnosis regarding diseases of the spinal cord would rest upon a much sounder foundation. Nevertheless, something has unquestionably been definitely determined, and much at least may be looked upon as very likely. We are therefore justified to formulate the special diagnosis in such a manner at least that, to conclude from the alteration of certain functions which are in connection with distinct areas of the spinal cord, certain special divisions of the spinal marrow are especially affected in the individual case. I therefore advise, upon the whole and in the main, to confine the diagnosis to the different individual forms of spinal affections known at this time, and in deviations from the usual picture, however, even if they be insignificant, not to make a particular and certain diagnosis, but, rather, to formulate the diagnosis in such a manner that an implication of certain areas of the spinal cord is indicated, therefore, for example, "sclerosis of the spinal cord with special affection of the pyramidal lateral column tracts," etc. By this method less exact diagnoses will result, but we will be spared from disappointment at the autopsy. An important general rule in diagnosticating diseases of the spinal cord, in my opinion, is not to force an affection into the framework of spinal diseases if the symptoms do not agree with a recognised disease of this organ. The most important requisite for any one who desires to gain a definite knowledge in the diagnosis of diseases belonging to this category, is to acquire all that is known regarding the anatomical structure and the functional significance of the individual portions of the spinal cord, as far as they have been determined or as has become likely up to the present time. Only with these points can the necessary certainty be acquired, in the frequently very difficult diagnosis relating to these parts of the body. Before we proceed to the description of the diagnosis of individual affections of the spinal cord we shall therefore give a hurried *résumé* of the anatomy and physiology of the spinal cord.

ANATOMICO-PHYSIOLOGICAL INTRODUCTION

The spinal cord consists of two substances easily distinguishable, the *white* and *gray* substance, which may be macroscopically discerned upon transverse sections. The white substance incloses the centrally lying gray substance, completely surrounding it, and from the upper lumbar cord downward in contrast to the upper portion of the spinal cord it becomes less and less obvious compared with the gray substance. The cord is divided by two median longitudinal spaces (anterior longitudinal fissure and posterior longitudinal fissure) into a right and a left half. These two parts of the spinal marrow are combined by the middle portion of the gray substance—the commissure, which itself is divided by the central canal into an anterior (com. ant. s. *alba*) and a posterior (com. post. s. *grisea* sensu strictiori) portion. In both, especially towards the base of the median fissure, numerous transverse nerve fibres are found, (coming from both halves of the spinal cord and crossing to the opposite side), their course and functions will be described farther on. At the two lateral parts of the gray substance (columns) combined by the commissure, the dorsal and

ventral ends are differentiated as the more slender *posterior* horn and the thicker *anterior horn*, at the lateral dorsal part of which in the cervical and upper dorsal cord, the more marked developed *lateral horn* is found. In the gray matter, two substances may be differentiated: The *spongious* and the *gelatinous*. The first forms the principal portion of the columns; the latter, the gelatinous substances, surrounds the posterior and also partly the lateral surface of the posterior columns like a mantle (*substantia gelatinosa Rolandi*). By the entrance and exit of the nerve roots of the spinal cord, the white substance is divided into three columns: The *anterior, lateral* and *posterior columns*, the last being further divided into a median and a lateral portion (the column of Goll and the column of Burdach).

In these columns the combined longitudinal fibres of the spinal cord run, and in distinctly separated bundles, which serve various functions. We are entitled to

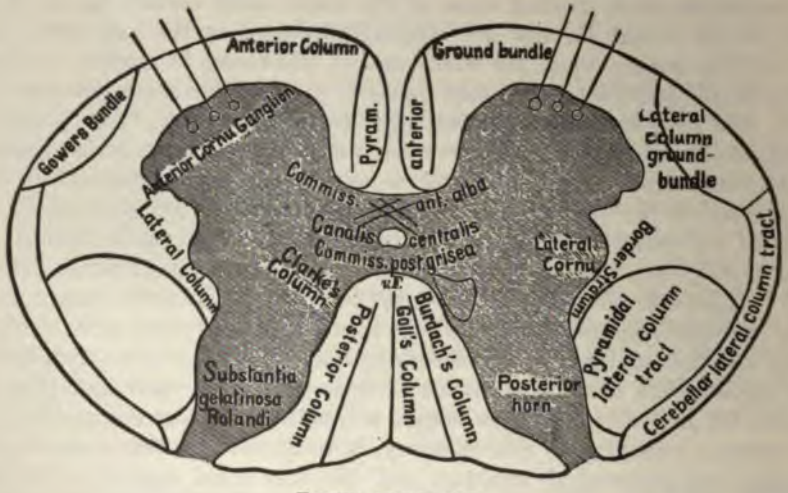


Fig. 27.—Cross-section of the Spinal Cord.

the latter assumption, partly by clinical experience (by the secondary degeneration of certain fibres of the spinal cord in disease of certain parts of the brain, discovered by Türck), partly by the result of experiments, in severing the cord, and partly, finally, by the observation that the various fibres of the spinal cord during their embryonic development receive their nerve medulla at different periods (Flechsig). Accordingly, it is customary now to differentiate the following special, functional and developmental "*fibre-column systems*" which belong together in the anterior, lateral and posterior columns (see Fig. 27).

In the *posterior column*: The external—*cuneate*—(Burdach's) columns. The internal—*gracile*—(Goll's) columns.

Of these main columns a small semilunar area lying contiguous to the posterior commissure, being a part of the posterior columns, must be differentiated as a "*central field*" (Fig. 27, v. F.). This appears to contain a special fibre system, which in disease of the posterior columns (as in tabes dorsalis) may be constantly spared by the degeneration.

In the lateral column: The *pyramidal lateral column tracts*; the "*narrow cerebello-lateral column tracts*" situated at the periphery of the lateral tract taking in the greatest length of the same (tractus cerebello-spinalis dorsales), anteriorly—i. e., ventrally from the cerebello-lateral column tracts, [illegible] (tractus cerebello-spinalis ventrales); the remaining parts of the lateral columns, the "lateral column ground bundles"; the part contiguous to the gray substance is specially designated as "boundary stratum."

In the *anterior column*: The *anterior pyramidal column tracts*, (the innermost portion alongside of the anterior longitudinal fissure, of the anterior column).

The remaining parts of the anterior column, the "*anterior column ground bundles.*"

To adhere to this division of the columns into individual, functionally differentiated column systems, is advisable in the interest of diagnosis of the diseases of the spinal cord, not only because certain diseases concentrate themselves, at least principally anatomically, to distinct column systems (so-called "system diseases"), but also because by this means the analysis of the functions of conduction of the white substance is made easier and its meaning can be more readily understood.

Microscopical Conditions.—Regarding the *microscopical structure of the spinal cord* it must first be mentioned that the *white substance* consists of medullated, centripetally and centrifugally conducting, longitudinally running nerve fibres, whereas the *gray* substance contains a fine mixture of medullated and non-medullated nerve fibres and multipolar *ganglion cells* of various sizes. The latter are found partly distributed (in the anterior and posterior horns and as has been lately shown, also in the substance of Rolando), partly running together to form larger groups. The two *principal groups* (*motor cells, anterior cornu ganglion cells*) lie in the anterior horn and in the median part of the posterior cornu against the gray commissure (Clarke's *columns, columnæ vesiculares*). As a *point of support* for the nerve elements serves partly the connective tissue coming from the pia mater (consisting of connective tissue) and which enters the white substance and is restricted to the same, and which brings about a separation of the nerve fibres of the white substance into different bundles, and partly also the *neuroglia*. This is found as a cement substance, between the individual nerve fibres and between these and the ganglion cells and contains stellate nuclear branching cells (the *glia cells*); a more marked collection of glia substance (horn-like) is found in the surrounding parts of the central canal, the sparsest amount of glia fibres is found in the gelatinous substance of Rolando.

Course of the Longitudinal Fibres and their Collaterals.—Of extraordinary importance for physiology as well as for pathology, is the special distribution of the nerve fibres in the spinal cord and the anatomical behaviour of the nerve cells and their nervous continuations. The researches of Golgi, Ramón y Cajal and Koelliker, in the embryo and in the new-born, have shed light on this subject. The most important results, which are given in the following description, have been taken especially from the works of Koelliker.

After the *posterior* (*sensory*) *root fibres* have gradually ascended into the spinal marrow, they travel towards the posterior column and those situated externally in the gelatinous substance towards the lateral column at the border zone of the posterior cornu (Koelliker); they now divide, so that they separate into two fibres each, the one radiating upward and the other downward. The *descending* fibres after a longer or shorter course, later unquestionably all of them, turn into the gray substance; of the *ascending* ones, however, a small portion after a short course also enters the posterior column into the gray substance ("short tracts"), the larger part, however, rises in the posterior column to the medulla oblongata (Fig. 28). In the posterior column they are arranged so that the fibres which have entered below are forced inward and anterior by those which have entered farther above, the fibres originating from the lower part of the body therefore in the upper parts of the spinal cord no longer lie in Burdach's columns but in the columns of Goll (Fig. 28). Besides these fibres of the posterior root which enter the most centrally (Fig. 28, 1) and which, as has been remarked, rise directly into the posterior columns without entering into connection with cells, other fibres branch off which instead of winding medially into Burdach's columns twine laterally (Fig. 28, 2), passing through the posterior cornu, also without entering cells, to traverse farther in the lateral column especially in its boundary layer. *All of these sensory column fibres in their longitudinal course give off fine lateral branches, "the collaterals,"* (Ramón, Koelliker) which enter the gray substance of the posterior cornua obliquely, dividing themselves in the latter and in various parts of the gray substance: terminating free in the posterior cornu, especially also in Clarke's columns as well as in the lateral and

anterior cornua of the same side (reflex collaterals of the sensory roots, Fig. 28, hrc.). These terminations and entwinings are found in especially great numbers in the boundary region between the gelatinous and the spongious substance in Clarke's columns, forming at both places a compact fine fibre blanket. The *longitudinal fibres of the anterior and lateral columns* bend, forming right angles in the gray substance and terminate here. They *represent* partly *centrifugal* conducting tracts (pyramidal tracts) which branch off farther into the gray substance, partly *centripetal* fibre masses which originate from cells in the gray substance and in the anterior lateral columns as well as in the cerebellar lateral columns, in which

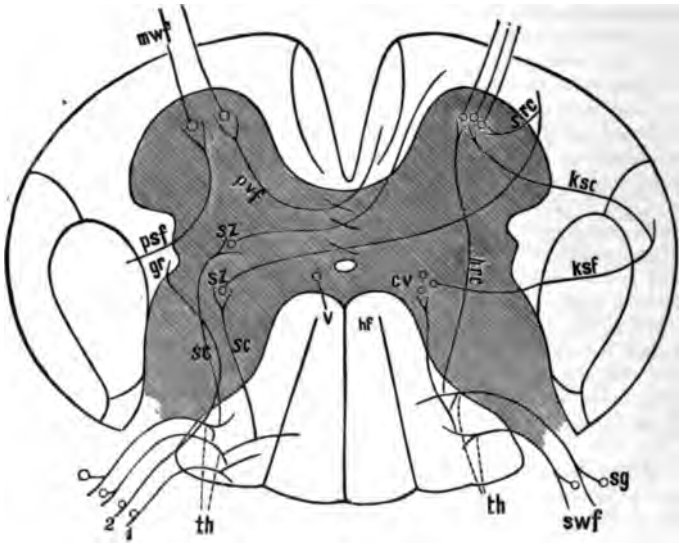


FIG. 28.—DIAGRAM OF THE COURSE OF THE COLUMN FIBRES. COLLATERALS AND NERVE PROCESSES IN A SECTION OF THE SPINAL CORD. (After Koelliker.)

sg, Spinal ganglion; *swf*, sensory root fibre; *mwf*, motor root fibre; *psf*, pyramidal lateral column fibre; *prf*, pyramidal anterior column fibre; *vhf*, ventral posterior column field; *gr*, boundary layer; *sz*, column cells; *sc*, sensory collaterals to the column cells (*sz*) and to the columns of Clarke (*cv*); *hrc*, sensory collaterals from the posterior column to the anterior horn cells (reflex collaterals); *src*, lateral column reflex collaterals; *ksf*, cerebello-lateral column fibre; *ksc*, cerebello-lateral column collateral; *th*, division of the posterior root fibres into an ascending and a descending branch, which associate with the posterior columns; 1, fibres entering directly into the posterior column; 2, fibres which extend directly into the boundary layer of the lateral column.

they travel upwards. As the sensory longitudinal fibres so all longitudinal fibres of the lateral and anterior columns appear to give off *collaterals* to the gray substance. The *lateral column collaterals* enter partly the posterior column, partly the anterior cornua going to the motor cell groups, partly they cross by the anterior commissure and perhaps also by the posterior commissure, towards the opposite side. The *anterior column collaterals* all run posteriorly; they partly cross in the commissura alba, partly going directly towards the anterior, lateral, and posterior horns. It is important, that *the collaterals of the column fibres*, no matter where they originate, or whence they travel, but also those of the gray substance surrounding the column fibres themselves, *all terminate in the same manner*. After much ramification, by which a net-like structure of the finest nerve fibres arises, the collaterals terminate in fine "*terminal arborizations*." These consist of numerous short twigs, *completely encircling the nerve cells*. According to Ramón y Cajal and Koelliker, *these terminal arborizations are never in direct contact with the cells, and nowhere are there found direct anastomoses between contiguously lying collaterals or terminal arborizations.*

Nerve Cells and their Processes.—The *nerve cells of the gray substance* according to their anatomical distribution and their physiological importance produce various formations. We may differentiate:

1. *Root cells*—i. e., *nerve cells whose axis cylinder processes terminate in roots* (partly in the anterior and partly in the posterior roots): (a) those neurites entering the *anterior roots* almost invariably come out of the motor root fibres without branches, originating as axis cylinders, being surrounded by a medullary sheath (Fig. 28, *mwf*); (b) those going into the *posterior roots* (arising from cells that are situated more in the dorsal part of the anterior horn) are probably to be looked upon as sympathetic nerve fibres (Fig. 30, *cff*).

2. A *second* variety of nerve cells, which form the principal mass of the nerve cells of the gray substance, are the *fundamental cells of the columnar fibres*, the "*column cells.*" These are cells, the processes of which originate in the gray substance, enter the white and become longitudinal fibres of the column; it appears that only anterior and lateral column fibres have such a cell origin in the gray substance, whereas the origin of nerve cell processes in the posterior columns, at least those which are in connection with roots, is quite rare—perhaps the cells in the posterior gray which give off processes to the ventral posterior column field may be counted here (compare Fig. 28, *v*). Column cells are found in the lateral column, especially in the region between the anterior and posterior horns and most probably also in Clarke's columns. Besides there are found in the vicinity of the central canal especially around its ventral side large column cells, the nerve processes of which run through the anterior commissure, before its entrance into the anterior column of the opposite side ("commissure cells").

3. As a third variety of nerve cells, cells are met with *whose nerve processes branch off into the finest twigs and end in the gray substance*, i. e., therefore not finding exit there ("*internal cells*"). They are apparently only found in the posterior horns, (inclusive of the gelatinous substance and Clarke's column); their processes may, as it appears, run vertically. In reference to the termination of the nerve-cell processes of the latter, nothing certain is known as yet, only that they do not form terminal arborizations like the collaterals.

The "*protoplasm processes*" ("*dendrites*") of all nerve cells finally branch out greatly, and even partly enter the white substance. They do not, however, give origin to any nerve fibres; neither do they anastomose (Ramón, Koelliker).

Both *commissures* consist of various nerve fibres which radiate from the one into the other half of the spinal cord. In the *anterior commissure* there are found: Nerve processes of column cells, which continue in the longitudinal fibres of the anterior lateral columns (of the opposite side) further crossing collaterals of the lateral and anterior columns. The *posterior commissure* undoubtedly consists of collaterals of the sensory root fibres which have crossed, perhaps also of crossed collaterals of the posterior lateral columns and of crossings of the nerve processes of cells from the region of the central canal and of the gelatinous substance. Besides, very probably, in both commissures protoplasm continuations cross of the contiguous anterior and posterior horn cells (Ramón y Cajal).

From the anatomical description just given, in connection with the results obtained by investigation of the embryonic development of the individual parts of the spinal cord and by the results obtained by experiments in severing the cord, by secondary degenerations and by clinical investigations, certain conclusions may be gathered regarding nerve conduction in movement, sensation, and reflex processes, which shall be discussed in the following chapters especially in so far as they are of value and of interest in pathology. (Compare Introduction to Diseases of Peripheral Nerves.)

As has already been explained, irritation and conduction occur by means of the neuron, each one consisting of a nerve cell with its dendrites, of

anterior cornua of the same side (reflex collaterals of the sensory roots, Fig. 28, hrc.). These terminations and entwinings are found in especially great numbers in the boundary region between the gelatinous and the spongious substance in Clarke's columns, forming at both places a compact fine fibre blanket. The *longitudinal fibres of the anterior and lateral columns* bend, forming right angles in the gray substance and terminate here. They *represent* partly *centrifugal* conducting tracts (pyramidal tracts) which branch off farther into the gray substance, partly *centripetal* fibre masses which originate from cells in the gray substance and in the anterior lateral columns as well as in the cerebellar lateral columns, in which

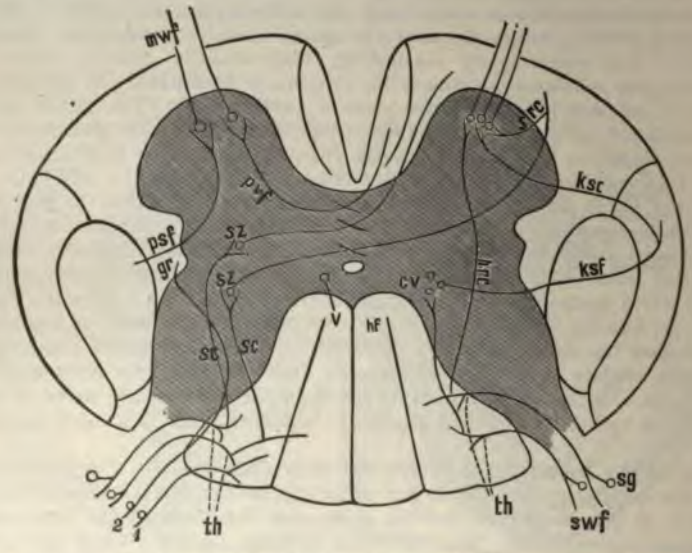


FIG. 28.—DIAGRAM OF THE COURSE OF THE COLUMN FIBRES. COLLATERALS AND NERVE PROCESSES IN A SECTION OF THE SPINAL CORD. (After Koelliker.)

sg, Spinal ganglion; *swf*, sensory root fibre; *mwf*, motor root fibre; *psf*, pyramidal lateral column fibre; *pef*, pyramidal anterior column fibre; *vhf*, ventral posterior column field; *gr*, boundary layer; *sz*, column cells; *sc*, sensory collaterals to the column cells (*sz*) and to the columns of Clarke (*cv*); *hrc*, sensory collaterals from the posterior column to the anterior horn cells (reflex collaterals); *src*, lateral column reflex collaterals; *ksf*, cerebello-lateral column fibre; *ksc*, cerebello-lateral column collateral; *th*, division of the posterior root fibres into an ascending and a descending branch, which associate with the posterior columns; 1, fibres entering directly into the posterior column; 2, fibres which extend directly into the boundary layer of the lateral column.

they travel upwards. As the sensory longitudinal fibres so all longitudinal fibres of the lateral and anterior columns appear to give off *collaterals* to the gray substance. The *lateral column collaterals* enter partly the posterior column, partly the anterior cornua going to the motor cell groups, partly they cross by the anterior commissure and perhaps also by the posterior commissure, towards the opposite side. The *anterior column collaterals* all run posteriorly; they partly cross in the commissura alba, partly going directly towards the anterior, lateral, and posterior horns. It is important, that *the collaterals of the column fibres*, no matter where they originate, or whence they travel, but also those of the gray substance surrounding the column fibres themselves, *all terminate in the same manner*. After much ramification, by which a net-like structure of the finest nerve fibres arises, the collaterals terminate in fine "*terminal arborizations.*" These consist of numerous short twigs, *completely encircling the nerve cells*. According to Ramón y Cajal and Koelliker, *these terminal arborizations are never in direct contact with the cells, and nowhere are there found direct anastomoses between contiguously lying collaterals or terminal arborizations.*

Nerve Cells and their Processes.—The *nerve cells of the gray substance* according to their anatomical distribution and their physiological importance produce various formations. We may differentiate:

1. *Root cells*—i. e., *nerve cells whose axis cylinder processes terminate in roots* (partly in the anterior and partly in the posterior roots): (a) those neurites entering the *anterior roots* almost invariably come out of the motor root fibres without branches, originating as axis cylinders, being surrounded by a medullary sheath (Fig. 28, *mcf*); (b) those going into the *posterior roots* (arising from cells that are situated more in the dorsal part of the anterior horn) are probably to be looked upon as sympathetic nerve fibres (Fig. 30, *cff*).

2. A *second* variety of nerve cells, which form the principal mass of the nerve cells of the gray substance, are the *fundamental cells of the columnar fibres*, the "*column cells.*" These are cells, the processes of which originate in the gray substance, enter the white and become longitudinal fibres of the column; it appears that only anterior and lateral column fibres have such a cell origin in the gray substance, whereas the origin of nerve cell processes in the posterior columns, at least those which are in connection with roots, is quite rare—perhaps the cells in the posterior gray which give off processes to the ventral posterior column field may be counted here (compare Fig. 28, *v*). Column cells are found in the lateral column, especially in the region between the anterior and posterior horns and most probably also in Clarke's columns. Besides there are found in the vicinity of the central canal especially around its ventral side large column cells, the nerve processes of which run through the anterior commissure, before its entrance into the anterior column of the opposite side ("commissure cells").

3. As a third variety of nerve cells, cells are met with *whose nerve processes branch off into the finest twigs and end in the gray substance*, i. e., therefore not finding exit there ("*internal cells*"). They are apparently only found in the posterior horns, (inclusive of the gelatinous substance and Clarke's column); their processes may, as it appears, run vertically. In reference to the termination of the nerve-cell processes of the latter, nothing certain is known as yet, only that they do not form terminal arborizations like the collaterals.

The "*protoplasm processes*" ("*dendrites*") of all nerve cells finally branch out greatly, and even partly enter the white substance. They do not, however, give origin to any nerve fibres; neither do they anastomose (Ramón, Koelliker).

Both *commissures* consist of various nerve fibres which radiate from the one into the other half of the spinal cord. In the *anterior commissure* there are found: Nerve processes of column cells, which continue in the longitudinal fibres of the anterior lateral columns (of the opposite side) further crossing collaterals of the lateral and anterior columns. The *posterior commissure* undoubtedly consists of collaterals of the sensory root fibres which have crossed, perhaps also of crossed collaterals of the posterior lateral columns and of crossings of the nerve processes of cells from the region of the central canal and of the gelatinous substance. Besides, very probably, in both commissures protoplasm continuations cross of the contiguous anterior and posterior horn cells (Ramón y Cajal).

From the anatomical description just given, in connection with the results obtained by investigation of the embryonic development of the individual parts of the spinal cord and by the results obtained by experiments in severing the cord, by secondary degenerations and by clinical investigations, certain conclusions may be gathered regarding nerve conduction in movement, sensation, and reflex processes, which shall be discussed in the following chapters especially in so far as they are of value and of interest in pathology. (Compare Introduction to Diseases of Peripheral Nerves.)

As has already been explained, irritation and conduction occur by means of the neuron, each one consisting of a nerve cell with its dendrites, of

the nerve process (root process, neurite, axis cylinder) arising from the nerve cell and of the terminal splitting of the same (terminal arborization). The irritation occurs in the neuron in the following manner: It travels from the cell to the neurite and thence to the terminal arborization, and is transferred from there to other ganglion cells the fibres of which are in intimate contact with the terminal arborization (without connecting directly with them) by way of the dendrites of the ganglion cell (compare Fig. 12). The performance of the function of the neuron, respectively of the neurite, depends upon the anatomical intactness of the nerve cell in question, so that in lesions that part of the neurite which is no longer in connection with the nerve cell primarily degenerates up to its terminal arborization. Later, on account of the interruption of the usual functional irritation and its conduction or derivation, the affected cell and with it the portion of the neurite in direct connection may secondarily degenerate.

Motor-Nerve Tracts.—As regards especially the *motor-nerve tracts,* their course has been recognised for some time, especially by the distribution of certain focal diseases of the brain, causing secondarily in the spinal cord a descending degeneration due to this cause. In such cases we find two distinctly separated portions of the spinal-cord column degenerated: The one in the anterior column next to the anterior longitudinal fissure on the same side of the focal lesion of the brain, the other more diffused in the lateral column of the opposite side (compare Figs. 28 and 34). It follows from this demonstration that the great majority of fibres of this motor tract cross before entering the spinal cord and this occurs in the well-known region, the decussatio pyramidum (pyramidal lateral column tract); only a small part of the fibres remain upon the same side and do not cross, to cross over to the opposite side, farther on into the spinal cord, especially in the anterior commissure to the anterior horns (pyramidal anterior column tract). The crossing of the fibres of the pyramidal anterior column and lateral column tract to the anterior horns occurs in the following manner: The column fibres and their collateral encircling successively in the gray substance and with their terminal arborizations entwine the motor cells, respectively, their dendrites. These cells then give origin to the axis cylinders of the motor-root fibres, which on their part again terminate in the muscle as terminal arborizations. If we add that the anterior and lateral pyramidal column tract combined may be followed farther up into the cerebrum, and that their fibres originate as axis cylinders in distinct nerve cells in the cortex of the brain (pyramidal cells), it is easily understood that the motor chief innervation tract—i. e., the pyramidal tract—consists of two distinct nerve units (with cell, fibre and terminal arborization) of which the one, the *central,* originates in the cortex of the brain, the other, the *peripheral,* in the spinal cord. If the continuity of a motor neuron be anywhere interrupted, secondary degeneration occurs peripherally from the point of lesion up to the terminal arborization. The degeneration therefore does not continue to the cell of the new neuron in contact with the terminal arborization, this remains intact in all its anatomical parts. A continua-

towards the periphery is impossible under any
nduction is completely interrupted at the

certain important clinical facts,
plained. In an interruption of the
ne
pon
y to
ion is
s. Ana-
secondary
from the
ourse of the
terior columns
nd of the lateral
ssed side; on the
eripheral nerves do
nor do the muscles
em atrophy. The same
ppens in cases in which
cord is severed or injured,
from the point of lesion
rd a secondary degeneration
interrupted column fibres in
spinal cord takes place up to the
int where they cross in the gray
ubstance, intertwining in their terminal arborizations. But even in this instance degeneration of the peripheral neuron does not occur—i. e., in the peripheral nerves and muscles—although they do not react to impulses of the will. As soon, however, as the *anterior cornu ganglion cells are also affected by the spinal-cord affection or are exclusively injured, flaccid paralysis with atrophy of the muscles occurs and the peripheral nerves degenerate,* just as in the case of the peripheral nerve itself being interrupted in its continuity.

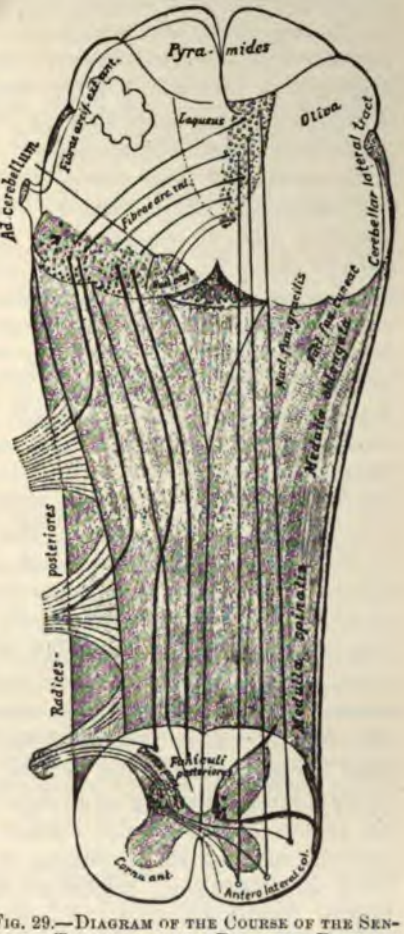


FIG. 29.—DIAGRAM OF THE COURSE OF THE SENSORY TRACT FROM THE POSTERIOR ROOTS TO THE MEDULLA OBLONGATA. (After Edinger.)

Regarding the *connection between the anterior horn cells with the motor-root fibres and these with the peripheral nerve trunks,* it can no longer be assumed, according to the latest investigations, that every root supplies *one* distinct peripheral nerve trunk. On the contrary, quite a number of *different* nerve fibres from various roots reach a *single* nerve of the extremity. Lately it has become further probable that synergistically acting muscles (no matter whether they are supplied by different

the nerve process (root process, neurite, axis cylinder) arising from the nerve cell and of the terminal splitting of the same (terminal arborization). The irritation occurs in the neuron in the following manner: It travels from the cell to the neurite and thence to the terminal arborization, and is transferred from there to other ganglion cells the fibres of which are in intimate contact with the terminal arborization (without connecting directly with them) by way of the dendrites of the ganglion cell (compare Fig. 12). The performance of the function of the neuron, respectively of the neurite, depends upon the anatomical intactness of the nerve cell in question, so that in lesions that part of the neurite which is no longer in connection with the nerve cell primarily degenerates up to its terminal arborization. Later, on account of the interruption of the usual functional irritation and its conduction or derivation, the affected cell and with it the portion of the neurite in direct connection may secondarily degenerate.

Motor-Nerve Tracts.—As regards especially the *motor-nerve tracts,* their course has been recognised for some time, especially by the distribution of certain focal diseases of the brain, causing secondarily in the spinal cord a descending degeneration due to this cause. In such cases we find two distinctly separated portions of the spinal-cord column degenerated: The one in the anterior column next to the anterior longitudinal fissure on the same side of the focal lesion of the brain, the other more diffused in the lateral column of the opposite side (compare Figs. 28 and 34). It follows from this demonstration that the great majority of fibres of this motor tract cross before entering the spinal cord and this occurs in the well-known region, the decussatio pyramidum (pyramidal lateral column tract); only a small part of the fibres remain upon the same side and do not cross, to cross over to the opposite side, farther on into the spinal cord, especially in the anterior commissure to the anterior horns (pyramidal anterior column tract). The crossing of the fibres of the pyramidal anterior column and lateral column tract to the anterior horns occurs in the following manner: The column fibres and their collateral encircling successively in the gray substance and with their terminal arborizations entwine the motor cells, respectively, their dendrites. These cells then give origin to the axis cylinders of the motor-root fibres, which on their part again terminate in the muscle as terminal arborizations. If we add that the anterior and lateral pyramidal column tract combined may be followed farther up into the cerebrum, and that their fibres originate as axis cylinders in distinct nerve cells in the cortex of the brain (pyramidal cells), it is easily understood that the motor chief innervation tract—i. e., the pyramidal tract—consists of two distinct nerve units (with cell, fibre and terminal arborization) of which the one, the *central,* originates in the cortex of the brain, the other, the *peripheral,* in the spinal cord. If the continuity of a motor neuron be anywhere interrupted, secondary degeneration occurs peripherally from the point of lesion up to the terminal arborization. The degeneration therefore does not continue to the cell of the new neuron in contact with the terminal arborization, this remains intact in all its anatomical parts. A continua-

tion of the nerve irritation towards the periphery is impossible under any circumstances, as the tract of conduction is completely interrupted at the point of lesion.

From these anatomical observations certain important clinical facts, which are to be observed, may be explained. In an interruption of the pyramidal tract (in the pons, crus cerebrum, or cerebrum) above the decussation; *hemiplegia occurs upon the opposite side,* and a tendency to greater tension and contraction is noted in the paralyzed muscles. Anatomically there develops secondary *descending degeneration,* from the point of lesion in the course of the affected area in the anterior columns upon the same side and of the lateral columns of the crossed side; on the other hand, *the peripheral nerves do not degenerate, nor do the muscles belonging to them atrophy.* The same condition happens in cases in which the *spinal cord* is severed or injured, naturally from the point of lesion downward a secondary degeneration of the interrupted column fibres in the spinal cord takes place up to the point where they cross in the gray substance, intertwining in their terminal arborizations. But even in this instance degeneration of the peripheral neuron does not occur—i. e., in the peripheral nerves and muscles—although they do not react to impulses of the will. As soon, however, as the *anterior cornu ganglion cells are also affected by the spinal-cord affection or are exclusively injured, flaccid paralysis with atrophy of the muscles occurs and the peripheral nerves degenerate,* just as in the case of the peripheral nerve itself being interrupted in its continuity.

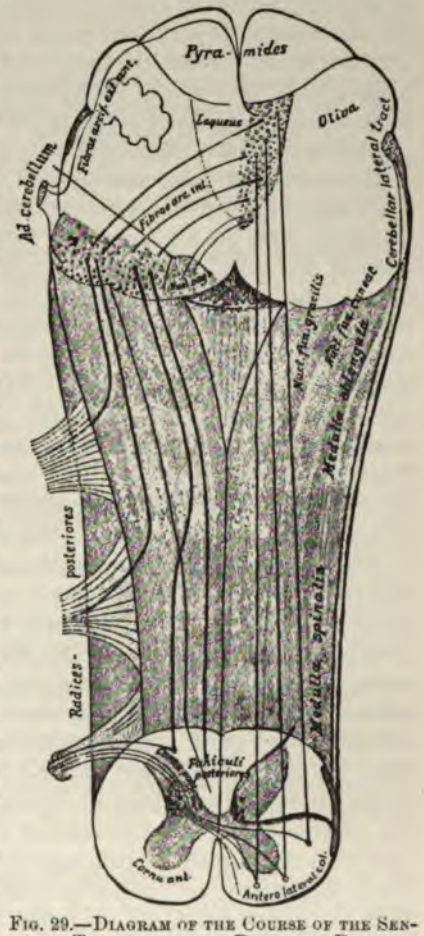


FIG. 29.—DIAGRAM OF THE COURSE OF THE SENSORY TRACT FROM THE POSTERIOR ROOTS TO THE MEDULLA OBLONGATA. (After Edinger.)

Regarding the *connection between the anterior horn cells with the motor-root fibres and these with the peripheral nerve trunks,* it can no longer be assumed, according to the latest investigations, that every root supplies *one* distinct peripheral nerve trunk. On the contrary, quite a number of *different* nerve fibres from various roots reach a *single* nerve of the extremity. Lately it has become further probable that synergistically acting muscles (no matter whether they are supplied by different

peripheral nerves or not) are innervated by the same root, and further that the root fibres arise in ganglion cells which in keeping with the origin of the former are arranged in groups ("segmental"), and represent in a functional respect a more or less uniform collection of cells. Of some members of this cell group it is already known which nerves (and muscles) they serve as the nucleus of origin. Thus the nerves supplying the flexors of the arm and hand originate in the lateral cell group in the anterior horns of the cervical cord, the extensors for the arm and hand originate in that group lying more medianly. We have already referred to this condition, in describing certain conspicuous varieties of paralysis.

Sensory Tract.—More difficult to determine and also more complicated, is the course of the *sensory tract* in the spinal cord. It is certain that the conduction of the sensory impressions in the cord results from the posterior roots after passing the spinal ganglion. The sensory fibres originate in the cells of the *spinal ganglion* and in such a manner, that the fibres arising in the spinal ganglion cell separate, going in two directions—namely, towards the periphery and through the posterior root fibres to the spinal marrow. According to another conception, it might be assumed that the peripheral part of the neuron may be looked upon as dendrite, the central part, i. e., the posterior root fibre, as the neurite, of the spinal ganglion cell. In all cases the peripheral severance of a nerve from the ganglion results in a secondary degeneration of the peripheral sensory fibres, whereas the posterior root fibres remain intact; in a division of the latter, however, the peripheral nerve fibres remain normal, as they are still in continuous connection with their cells of origin, whereas the nerve fibres of the posterior roots, being separated from the ganglion, degenerate so far, until they are brought in contact with other terminal arborizations of other nerve cells. This is the case in various areas of the spinal cord (compare Figs. 28 and 30). A portion of the sensory fibres enters the gray substance and there comes in contact with column cells (after forming terminal arborization), from which a second takes its beginning, being *crossed* by the ascending nerve unit in the anterior lateral column ground bundle; another part of the sensory fibres of the posterior root at once reaches the posterior column after its entrance into the cord (without coming in contact with cells) and ascends in the form of longitudinal posterior column fibres into Burdach's column and farther up in the Goll's column to the medulla oblongata. Here the longitudinal fibres come in contact with the nucleus of the funiculus gracilis and with the funiculus cuneatus, from which numerous fibres cross over to the opposite side into the lemniscus ("lemniscus decussation," situated above the pyramidal decussation), to join the first-mentioned ascending sensory fibres which have already crossed into the spinal cord in the ground bundles of the anterior lateral columns (see Fig. 29), and combined with these reach the lemniscus of the middle brain—i. e., the main central sensory tract. Accordingly it is clear that, in a division of the posterior roots, *secondary ascending degeneration of the posterior columns* arises travelling upward (especially in Goll's column) and reaching the medulla oblongata.

The conditions just described are unquestionably true of the greatest majority of the fibres of the sensory spinal-cord fibres. This much is certain, that in a division at the peripheral side of the spinal ganglion *all* sensory fibres of the *peripheral* nerve degenerate; in a division of the posterior root upon the side of the ganglion leading to the spinal cord, there was found in the peripheral nerve (in keeping with the retained connection with the ganglion), besides the majority of sensory fibres which were still intact, *some few which had degenerated*, and *vice versa*, upward in the posterior root, besides the ascending degeneration of all of the central fibres, *some few fibres were found intact.* (Compare Fig. 30, *cff.*) If this finding proves to be constant, later on, in other cases also, it must necessarily follow that the smallest part of the sensory nerve fibres which run in the posterior root have their cells of origin (as all others) not in the spinal ganglion, but further centrally in the spinal cord. Very probably, according to the latest investigations of Ramón y Cajal and v. Lenhossek, certain anterior cornu cells may so be regarded; the nerve fibres arising from the latter, travelling posteriorly and to the posterior roots (without coming in contact with spinal ganglion cells) running centrifugally, must be presumed as belonging to the sympathetic (v. Koelliker).

Cerebellar Lateral Column Tract.—Important, especially from a pathological standpoint, is further the connection of the columnæ vesiculares with certain elements of the posterior roots. Very probably, numerous *collaterals* of the posterior column fibres end in Clarke's columns (Ramón and Koelliker) and perhaps also individual nerve fibres of the posterior root. From Clarke's columns, as Koelliker has shown, nervous processes enter the lateral column, especially into the periphery of the lateral column, in the *cerebellar lateral column tracts* which are situated there, in which the fibres, after giving off collaterals, rise to the restiform body (cerebellar lateral column tract collaterals, perhaps reflex collaterals *ksc*, Fig. 28) and thence travel to the worm of the cerebellum (see Fig. 29). The tract conducts *centripetally* and degenerates in ascending as soon as the continuity of the spinal cord, and with this the connection of the cerebellar lateral longitudinal fibres with their cells of origin, is interrupted. According to what we know at present, the fibres in question play a part in a functional connection, as sensory conduction tracts in *coordination*; by their collaterals going towards the gray substance (to the anterior cornu cells, *ksc*, Fig. 28) the possibility arises, that the cerebellar lateral column tracts are active in the reflex mechanism. Later investigations have shown that only the fibres of the dorsal part of the cerebellar lateral column tract take their origin from cells of Clarke's column, whereas those of the ventral portion of the cerebellar lateral column tract, of Gowers's bundle (see Fig. 27), arise from other cells of the gray spinal matter. The *course* of this tractus cerebellospinalis ventralis is a different one. After both tracts, lying contiguous, arise to the medulla oblongata, the dorsal part enters the restiform body and rises to the cerebellum, whereas the ventral portion only enters the cerebellum very much higher up (in the region of the inosculation of the cerebellar peduncles).

Conduction of the Various Qualities of Sensation.—It will be of the greatest importance, in a physiological as well as a pathological respect, if we understood, not only the course, but also the *functional importance of the sensory fibre conduction in its individual parts*, especially the *exact* course of the tracts for the individual qualities of sensation; this, however, is not the case at present. We may assume, according to physiological and clinical experience, *that the conduction of the sensations of heat and pain occurs in the gray substance.* In what manner the *sensation of pain* occurs, has already been minutely described upon p. 435. According to what has been mentioned there, it is clear that a greater number of cells of the gray substance, especially of their posterior part, is brought into action. The tracts for the *sensation of temperature*, as we may also assume, run through the gray substance and by means of column cells

and from the processes which radiate into the ground bundles of the anterior lateral columns. This is also true of a portion of the conduction fibres for tactile sensation, whereas for another part there is a direct course, i. e., without passing cells—to the border stratum of the lateral column of the same side (see Fig. 28, 2) is open. We certainly know that the tracts for tactile sensation are not, as was assumed for a long time,

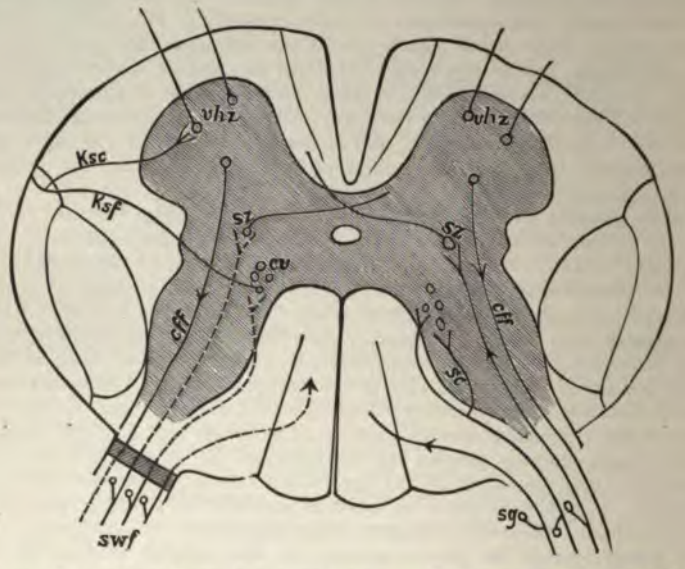


FIG. 30.—DIAGRAM OF FIBRE DEGENERATION AFTER SEVERING THE POSTERIOR ROOTS OF THE LEFT SIDE.

sg, Spinal ganglion; *swf*, sensory root fibres; *sz*, column cells; *vhz*, anterior horn cells; originating in one of the latter, a (not degenerating in the spinal cord) nerve-cell process (*cff*) which extends into the posterior root; *sc*, sensory collaterals; *cv*, columnæ vesiculares (Clarke's columns); *ksf*, cerebello-lateral column fibres originating in Clarke's columns; cerebello-lateral column collaterals: ———; intact . . . . . degenerate owing to lesion.

to be found in the *posterior columns*; the latter, instead, represent the area for the *sensory muscle fibres.*

Reflex Tracts.—Of especial importance for the diagnosis of diseases of the spinal cord is the *condition of the reflexes,* as their preservation and diminution or increase in the individual case varies greatly, according to the seat of anatomical changes in the spinal cord. To understand this variable condition, a knowledge of the action of reflexes and their course in the nerve tracts is necessary. In general, in the reflexes arising through the spinal column, a sensory fibre, the gray substance rich in nerve cells of the spinal cord, and a centrifugal motor-fibre tract are irritated. The causation of the reflex action under normal circumstances presupposes a certain intensity of the irritation as well as the elapse of a certain period for the action of the irritation in the sensory and motor-fibre tract, above all, however, in the reflex arc of the spinal marrow, in which the conduction of the irritation under all circumstances requires a ten times longer period than in the afferent and efferent fibres. A certain

resistance to reflex conduction in the spinal cord may therefore be assumed. This appears reasonable, according to the latest determined anatomical variety of connection of the fibres and cells, the link not being immediate. However, it dare not be forgotten that also the sensory tract of conduction from the periphery to the brain, as we now know, is not a continuous one, but is interrupted by the insertion of nerve cells, of nerve cells which on one side are surrounded by terminal arborization of the nerve fibres and are only irritated by "contact." The same is also true of the motor-conduction tract. According to my opinion, nothing remains even now for the explanation of the origin of reflex phenomena than the hypothesis that the tracts upon which sensory irritations are conducted to the brain are those which conduct the motor irritation from the brain centrifugally, therefore the tracts which are utilized as a rule (in spite of the cells enclosed in them), conduct more *readily* than the reflex tracts, in which the transference of sensory fibres to (motor) cells occurs in an unusual manner, thus giving rise to a certain degree of resistance. If the usual irritation of the anterior horn cells by the brain is permanently absent, their production may now occur through sensory irritation more readily, and increased reflex irritability may be the result. This is in fact the case in an interruption of the pyramidal tract by pathological processes. It is usually assumed that certain *reflex inhibitory fibres* which lead out from the brain running centrifugally in the anterior lateral columns become incapable of conduction and their absence produces an increase of the reflexes, an assumption which is not necessary, according to the hypothesis proposed by me.

A facilitation of the occurrence of reflexes, as we have seen, may occur from the absence of the usual irritation of the anterior horn cells by impulses of the will. It need, however, not occur. On the contrary, cases have been observed of a complete lesion of the transverse part of the spinal cord, in which, in spite of the microscopically proved integrity of the reflex arcs below the point of lesion, the skin and tendon reflexes were *absent*. Rosenthal and Mendelssohn upon the basis of their investigations, have explained this fact in the manner that in the absence of function of the upper parts of the spinal cord, of the cervical cord, and of the medulla oblongata, the reflexes no longer occur, as at this point under normal conditions the reflexes originate, i. e., the bulbo-cervical region being the point at which all irritation entering from peripheral sensory nerves in the spinal cord is most easily transferred to motor tracts. The short reflex arcs (compare Figs. 12 and 28) are only then utilized when more powerful irritation occurs, as in these reflex tracts a greater resistance takes place than in the long tracts. Although this view has the best experimental foundation, and a portion of the observed deviations from the normal conditions of the reflexes in morbid conditions may easiest be explained thereby, I do not believe that this eliminates all the difficulties for the explanation of the conduct of the reflexes in pathological states. Above all it appears necessary for me to know whether in the absence of motor irritations for the anterior horn cells, provided they no longer receive the ordinary kind of irritation, they become more susceptible to sensory irritation. Why this does not occur under all circumstances has as yet not been explained in a satisfactory manner.

Crossed and General Reflexes.—The origin of "*crossed*" *reflexes* finds its anatomical substratum in the combined action of the collaterals, which leave the column fibres, arising from column cells and ascending in the antero-lateral columns upon the opposite side and come in contact with motor cells (compare Fig. 28, *src*). Some light has also been spread by the latest anatomical investigations in reference to

the origin of the *distributed reflexes* (by means of the so-called "*long*" reflex arcs). It must be remembered that every sensory fibre group divides into an upper and lower fibre (compare Fig. 31, *sth*), and from these fibres, probably in their entire course, collaterals enter the gray substance; further, that those column fibres which are indirectly connected with sensory root fibres, and which arise in the column cells and go over to the ground bundles of the anterior lateral columns, after they have divided into an ascending and descending branch, also give off collateral twigs in their entire course and that further on also the cerebellar lateral column fibres originating from Clarke's column send collaterals to the gray substance, especially to the anterior horns (Fig. 28, *ksc*). If we reflect further, that these collaterals arising from different parts may act upon motor-nerve cells, with their terminals (as reflex collaterals), it is easily understood that this anatomical arrangement of the origin and termination of the collaterals allows the distribution of an irritation, which reaches the spinal cord over various portions, and in this manner favours the production of distributed reflexes.

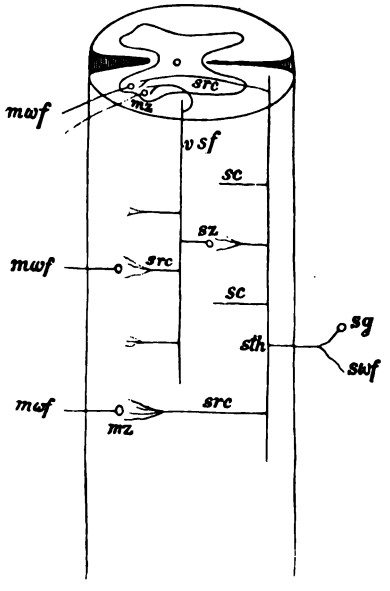


FIG. 31.—DIAGRAM OF THE COURSE OF THE REFLEX TRACTS, ESPECIALLY OF THE REFLEX COLLATERALS. (After Koelliker.)

sg, Spinal ganglion; *swf*, sensory root fibre; *mz*, motor cell of the anterior horn; *mwf*, motor root fibre; *sz*, column cell; *sc*, sensory collaterals; *src*, reflex collaterals (sensory collaterals to the anterior horn cells); *sth*, division of the sensory root fibre into a superior and an inferior fibre; *vsf*, anterior lateral column fibre.

This fact also explains that sensory irritations arising externally, especially coming from the muscles, may irritate motor cells at *various heights* in the spinal cord, and cause a *simultaneous* orderly action. The neurites arising in the column cells, with their ascending and descending branches and the collaterals going off from them, which divide at various heights around motor cells, and further the dendrites of the motor cells themselves, are unquestionably able to combine the individual motor-cell groups functionally among each other, and to allow of variously united, anatomically well-founded associations (see also chapter on Co-ordination and its Disturbance). Figs. 28 and 31 will more easily explain what has just been said.

As may be gathered from what has been said, the latest anatomical investigations regarding the explanation and origin of the reflexes are greatly simplified, but they do not elucidate some individual details of the reflex process. We are forced to assume that, for the production of reflexes, a certain resistance to conduction must be overcome. It appears that certain poisons increase this condition (potassium bromide, etc.), whereas others, primarily strychnine, lower it, i. e., therefore greatly increasing reflex irritability.

Spinal Centres.—Certain cell groups lying in the spinal cord, which by afferent and efferent nervous influences, therefore in a reflex manner, are able to originate certain orderly movements or cause secretion, are designated by the name "*spinal centres.*"

Functional disturbances of these centres are not rare, according to the seat of the spinal-cord affection, especially a functional disturbance in the *cilio-spinal centre* situated in the inferior cervical cord and in the upper dorsal cord, producing a dilatation of the pupil (this centre is stimulated by shading the retina, and its fibres enter the cervical sympathetic); further, by the centres lying in the lumbar cord, for the *evacuation of urine and fæces and for erection*, and, finally, the *sweat-*

secreting centres. It was also assumed that the *production of heat* was influenced by the spinal cord, in so far as fibres which were supposed to originate in a heat centre in the brain, which checked the conduction of heat, descended through the spine to the pons and spinal cord, so that in a destruction of this tract of conduction increased production of heat and a rise in temperature would result. A great part of the increase and decrease in temperature, occurring in man, in injuries of the spinal cord, may be regarded as due to changes of *vaso-motor innervation*, the latter playing the principal part in the regulation of heat, being influenced by the gray substance, respectively by the *vaso-motor centres* lying in the gray substance. *Trophic disturbances* in the skin may be referred to paralytic conditions of the vaso-motors, a condition that is often noticed in the course of diseases of the spinal cord.

PRELIMINARY DIAGNOSTIC REMARKS

Localization Diagnosis.—We shall attempt, according to the description of the course and functional importance of the individual fibre tracts in the spinal cord, by comparison with what clinical experience teaches, to determine *which morbid phenomena may be due to changes in distinct areas of the spinal cord, and in how far in this respect diagnoses of localization are possible.* At the onset it must be emphasized that in the following description we are not concerned with absolutely unalterable rules, but, rather, that much still requires careful clinical and physiological investigation, and probably, in the future, in various directions will be subject to correction.

Disease of the Antero-Lateral Pyramidal Column Tracts.—*Disease of the pyramidal tracts of the spinal cord,* i. e., *the central motor neuron,* may be assumed if *paralyses or paresis of the extremity without atrophy of the muscles exists,* there being present, simultaneously, a tendency to *spastic contraction, increased irritability,* especially of the tendon reflex, and *normal electrical reaction of the nerves and muscles. Sensation is intact.*

The origin of these symptoms is easily explained from the previous remarks regarding the function of the nerve fibres forming the anterior and antero-lateral pyramidal column tracts. In this respect it must be particularly remembered that certain centrifugally running nerve fibres in the antero-lateral columns are in a hypothetical manner brought in connection with the *inhibition of the reflexes,* the absence of their function very easily explaining the increase of the tendon reflexes.

Disease of Anterior Horns and Anterior Roots.—*Disease of the anterior horns,* i. e., *the beginning of the peripheral motor neuron, produces* flaccid *paralysis of the extremities with atrophy of the muscles, signs of DeR; reflex irritability,* as soon as the morbid process has reached a marked extent, is entirely suspended. In the latter cases, especially in disease of the anterior horns in the lower part of the spinal cord, disturbances in excretion of urine ought to be expected; up till now this is always absent in such cases. This may probably be explained in that a *complete* destruction of all ganglion cells of the affected reflex arcs are necessary, for a cessation of the reflexes of the muscles of the bladder, or, as appears to be anatomically possible, that these reflex arcs are situated more in the border region against the posterior horns and are not affected in disease of the anterior cornua. In keeping with the origin of certain

fibres of the sympathetic, in the anterior horns (see p. 517, 1), vaso-motor disturbances should be expected in disease of the anterior horns. *Sensation,* however, remains completely intact.

Disease of the anterior nerve roots produces the same symptoms as disease of the anterior horns. *Distinct* differential points between both diseases *do not exist* (compare diagnosis of polyneuritis), even if the presence of vaso-motor disturbance after what has just been said, is somewhat against disease of the anterior roots.

Combinations of diseases of the central and peripheral neuron occur especially in amyotrophic lateral sclerosis. The clinical symptoms in such cases are: Pareses and spasm, besides muscular atrophy with preservation of sensation.

Disease of the Posterior Nerve Roots and Posterior Horns.—As the result of *disease of the posterior nerve roots* there are: *Loss of sensation of every quality,* i. e., *total anæsthesia,* in the cutaneous field supplied by the affected root, *sensory ataxia, cessation of reflexes with preserved motility.* Also absence of the sensation of *urinary pressure* is to be expected.

As result of *disease of the posterior horns,* respectively *of the dorsal division of the gray substance,* there are to be expected: *Disturbance of the vaso-motor innervation (trophic disturbances),* partly also in the tactile sense in the skin, marked *diminution of heat and pain sensation* (in a unilateral lesion of a posterior division of the gray substance, partly tactile anæsthesia of *the same side* (compare Fig. 28, 2), and absence of pain and temperature senses upon the opposite side of the lesion; for the explanation of the latter phenomenon an anatomically founded substratum is given by the fibres going from the column cells (Fig. 28, *sz*) to the antero-lateral column ground bundles), eventually also *sensory ataxia, whereas the muscle sense and motility remain intact.* The *reflexes* may be partly impaired, according as to whether the reflex collaterals of the sensory roots are interrupted in great numbers, which go to the tracts of the posterior and to the anterior horns.

Disease of the Posterior Columns.—*Disease of the posterior columns,* provided that neither the gray substance nor the roots are simultaneously affected, as a rule produces *no marked disturbance of the tactile sense* (their main tracts unquestionably crossing through the gray substance by means of column cells into the antero-lateral columns), *no disturbance of the temperature or pain sense,* but, on the other hand, *disturbance of muscular tonus, muscle sensation, and sensory ataxia. Reflex irritability* may be diminished, provided the *reflex collaterals* going off from the posterior columns are affected in great numbers. With this there may be *incomplete sensation of urinary pressure and disturbance in the excretion of urine;* these are to be referred to disease of sensory-, perhaps also partly of motor-, conduction tracts, which latter running into the posterior columns, in a division of the spinal cord, degenerate *downward,* and in the lumbar cord in the sæptum medianum are found as the "oval field" (Flechsig). *Motility* naturally is entirely *intact.*

Disease of the Antero-lateral Column Ground Bundles.—It cannot be definitely determined whether disease of the *anterior and lateral column ground bundles* produces distinct phenomena which may be diagnostically separated from the symptoms just described and occurring in distinct affections in areas of the spinal cord. These bundles are unquestionably traversing areas, partly for the winding motor pyramidal lateral tracts and pyramidal anterior column tracts and their collaterals, partly for reflex collaterals of the sensory-cell column processes, and further they

are the region of the ascent of the latter to the medulla oblongata, and also for those fibre tracts which rise centrifugally and travel downward from Deiter's nucleus, and which serve co-ordination. Therefore, all kinds of symptoms may occur in disease of the antero-lateral basic bundles: Paresis, crossed anæsthesia, reflex disturbances. Ataxia should also be expected, as the lateral column basic bundles represent areas through which the processes from Clarke's column travel to the cerebellar lateral column, and in the anterior lateral column the areas from which the fibres travelling downward from Deiter's nucleus enter the spinal cord.

Diseases of the Cerebellar Lateral Column Tracts.—***Disease of the cerebellar lateral column tracts,*** finally, gives rise to ***disturbance of co-ordination,*** and as I

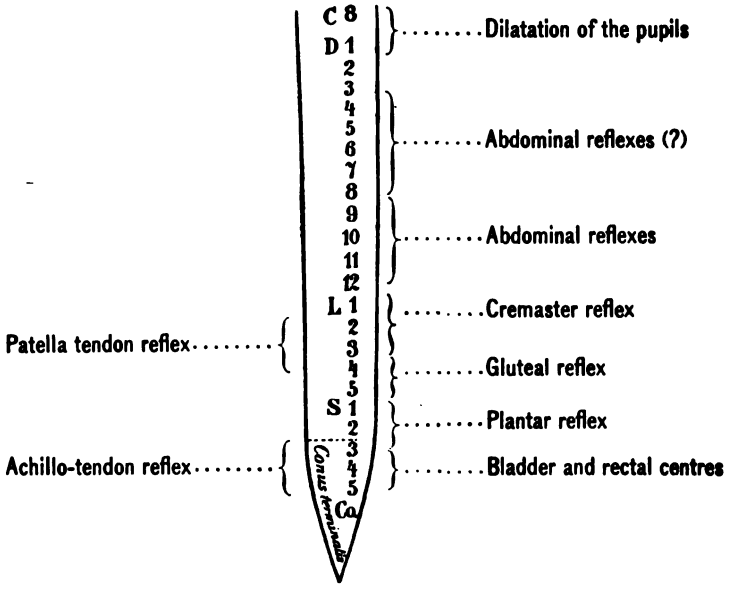


FIG. 32.—DIAGRAM SHOWING THE POINTS OF ORIGIN OF THE MOST IMPORTANT REFLEXES.

should like to assume, disturbance in the region of the *reflexes*, as the collaterals of the cerebellar lateral tract fibres enter the gray substance, especially the motor cells of the anterior horns. Motility as such is in no way disturbed.

In a number of affections of the spinal cord, the diagnostician must decide less which individual fibre tracts and ganglion-cell groups in the spinal cord are diseased, than *at which height* the spinal cord is affected in the given case. It must therefore still be explained which function is supplied by the individual spinal-cord segments and the nerves which arise from them. As a guide, a review of the findings which are known in this respect will be given; we owe this particularly to Starr.

2–3. Cervical Segment: *Motor* fibres for the sternocleidomastoid, trapezius, muscles of the back of the neck, diaphragm; *sensory* fibres for the posterior part of the head and back of the neck.

4. Cervical Segment: *Motor* fibres for the diaphragm, the supra- and infra-spinatus, deltoid, biceps, and coracobrachialis, rhomboideus and supinator longus; *sensory* fibres for the neck, the upper shoulder region and the outer surface of the arm.

5. Cervical Segment: Motor fibres for the deltoid, biceps and coracobrachialis, the supinators, serratus magnus, rhomboideus, and the brachialis anticus; *sensory* fibres for the posterior surface of the shoulder and the outer and posterior surface of the upper and forearm. *Tendon reflexes* for the corresponding muscles.

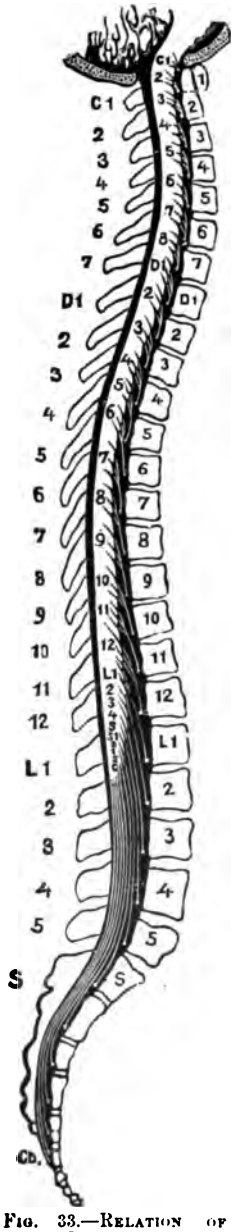


FIG. 33.—RELATION OF THE INDIVIDUAL CORD SEGMENTS TO THE VERTEBRAL COLUMN. (After Gowers.)

6. Cervical Segment: *Motor* fibres for the biceps, brachialis anticus, pectoralis, serratus magnus, pronators, triceps, extensors of the hand and fingers; *sensory* fibres for the outer side of the forearm and the back of the hand. *Reflexes*: Tendon reflexes for the extensors of the upper and lower arm.

7. Cervical Segment: *Motor* fibres for the triceps, the extensors for the hand and fingers, flexors and pronators of the hand, latissimus dorsi, teres major; *sensory* fibres for the radial and median tracts of the hand. *Reflex*: Tapping the vola manus causes the fingers to close.

8. Cervical Segment: *Motor* fibres for the flexors of the hand and fingers; *sensory* fibres for the tract of the median and ulnar nerves. *Reflex*: Pupillary reflex.

1. Dorsal Segment: *Motor* fibres for the interossei and the small muscles of the hand (especially for the ball of the thumb and little finger); *sensory* fibres for the tract of the median and ulnar nerves. *Reflex*: Pupillary reflex (eighth cervical and first dorsal).

2–12. Dorsal Segment: *Motor* fibres for the intercostal muscles, the muscles of the back and abdomen; *sensory* fibres for the skin of the breast, the back and the abdomen. *Reflex*: Abdominal reflex (lower dorsal nerves).

1. Lumbar Segment: *Motor* fibres for the lower abdominal muscles and for the quadratus lumborum; *sensory* fibres for the lower half of the abdomen. *Reflex*: Cremaster reflex (1–3 lumbar).

2. Lumbar Segment: *Motor* fibres for the ileopsoas and cremaster; *sensory* fibres for the testicles and spermatic cord, the outer side of the hip and the mons veneris. *Reflexes*: Patella tendon reflex. (Second to fourth lumbar.)

3. Lumbar Segment: *Motor* fibres for the sartorius, pectineus and the adductors of the thigh; *sensory* fibres for the anterior and inner side of the hip.

4. Lumbar Segment: *Motor* fibres for the quadriceps femoris, gracilis and obturatorius; *sensory* fibres for the anterior and inner side of the thigh, partly also for the inner surface of the lower leg up to the inner border of the foot.

5. Lumbar Segment: *Motor* fibres for the glutæus medius and minimus, biceps femoris and semitendinosus and semimembranosus; *sensory* fibres for the external side of the thigh. *Reflexes*: Gluteal reflexes (4–5 lumbar).

1. Sacral Segment: *Motor* fibres for the outward rotators for the thigh and the glutæus maximus; *sensory* fibres for the posterior side of the thigh and lower leg. *Reflex*: Achillo-tendon reflex (5th lumbar and 1st sacral).

2. Sacral Segment: *Motor* fibres for the muscles of the calves, the gastrocnemius and soleus, tibialis anticus and for the peroneal musculature; *sensory* fibres for the outer side of the lower leg and foot, also for the bladder and rectum. *Reflexes*: Plantar reflex (first and second sacral), centre for erection.

3. Sacral Segment: *Motor* fibres for the ischio- and bulbo-cavernosus; *sensory* fibres for the penis, the middle parts of the scrotum and the urethral mucous

membrane. *Reflexes*: Ejaculation centre, Achillo-tendon reflex (third to the fifth sacral).

4. Sacral Segment: *Motor* fibres, respectively reflex, of the detrusor vesicæ; *sensory* fibres for the skin of the perinæum, and the skin of the sacrum. *Reflexes*: Bladder and rectal centre (fourth to the fifth sacral).

5. Sacral and Coccygeal Segment: *Motor* fibres for the sphincter ani externus and levator ani; *sensory* fibre for the skin over the coccyx and anus.

The region of origin of the important reflexes is shown by Fig. 32.

From the preceding diagrams it is shown that the individual areas of innervation are not identical with those of the peripheral nerves, every root has a distinct cutaneous innervation field, which becomes anæsthetic if the root supplying it is severed, whereas the cutting of a peripheral nerve eventually results in anæsthesia of various peripheral root areas (because the individual nerve by receiving fibres from roots originating in the neighbourhood of the plexus, receives fibres originating from different roots). The motor innervation of the root probably occurs in the manner that those fibres which arise together in the nerve root, which are intended for muscles working synergistically (no matter whether they are supplied by various peripheral cells or not), have also distinct ganglion-cell groups which belong together. (Compare p. 519.)

If in the individual case the height is to be determined at which the spinal-cord segment is affected, the upper boundary of the anæsthetic cutaneous area must be ascertained. This shows the affected spinal-cord segment. It must, however, be observed that the latter, on account of the deeper point of origin of the affected spinal-cord nerve root from the vertebral column, lies somewhat higher than this. For example, the first dorsal root compares to the seventh cervical segment and the farther down in the spinal cord the root lies, the higher up the probably affected segment will be found, etc. (Compare Fig. 33.)

DISEASES OF THE MEMBRANES OF THE SPINAL CORD

ACUTE SPINAL MENINGITIS

The diagnosis of *acute spinal meningitis* in general offers no great difficulties. As the pia mater is richly supplied with vessels, it is readily seen that by an inflammatory exudation of the tissues of the pia, due to the plentiful vascular supply, swelling occurs and especially in the subarachnoid space, respectively in the subdural space, where larger collections of the exudate gather. This gives rise to much pressure and irritation of the numerous nerves of the pia itself as well as of the nerve roots which break through the membranes into the spinal cord. Inflammatory infiltrations may also occur in these roots and by means of the processes from the pia, the substance of the spinal cord itself may be affected by the inflammation.

Symptoms.—After what has been said it may be easily seen that *fixed pains along the vertebral column and pains which radiate to the trunk and body* (*often girdle-like*) represent a principal symptom of spinal meningitis. These pains are increased by pressure upon the vertebral column, but are especially marked in movements of the vertebral column anteriorly or laterally, as this produces more marked irritation of the inflamed parts. At the same time there is very usually *hyperæsthesia of the skin and muscles*; pressure upon the skin, raising it in folds or slight squeezing of the muscles, frequently produces severe pain. The patient stiffens the vertebral column and prevents every movement of the same; this *stiffness* is partly due to *muscle spasm* which occurs partly directly from irritation of the anterior roots, partly also reflexly, and is observed in the muscles of the neck as *rigidity*, in the extensors of the back as *opisthotonus*, showing itself in the abdominal muscles as a *scaphoid retraction of the abdomen*. Less frequently are *muscular contractions* noted in the course of spinal meningitis. *Reflex irritability* may be somewhat increased at first, later the cutaneous and tendon reflexes are reduced or absent, as well as irritative phenomena in general, which in the later course of the disease give place to *paralytic conditions*, i. e., anæsthesia and paralysis, in that the pressure upon the root fibres may become so marked and so continuous that a loss of function of the latter may be the result. In how far, in the individual case, the implication or the extension of the inflammatory process from the pia to the substance of the cord gives rise to these conditions *cannot* be determined in a simultaneous affection of the roots.

The *fever* occurring in acute spinal meningitis shows no characteristic type; it may set in with a chill and terminate with a pre-agonal excessive rise in temperature.

Rarer Symptoms.—Besides the more constant symptoms: The stiffness of the vertebral column and pain, the increase of pain by active and passive movements of the spinal cord, the eccentric hyperæsthesia, the clonic and especially tonic muscular spasms, the anæsthesias and motor paralyses, with cessation of the reflexes, which occur in the further course of the disease, there are also found, according to the concentration of the inflammation upon certain areas of the spinal cord and its membranes (in the cervical, dorsal and lumbar part), varying phenomena of importance in the special diagnosis. As such must be mentioned *interference with the evacuation of feces and urine*, in which at first retention (perhaps in the majority of cases due to spasm of the sphincter), later on incontinence is prominent. Also polyuria and glycosuria may occur in the course of spinal meningitis, as well as albuminuria, alteration in the secretion of urine, which must be referred to an implication of the spinal marrow in the inflammatory process or to disturbances of the affected peripheral innervation tracts. Further, the *skin*, besides the previously mentioned hyperæsthesia, often shows changes of a vaso-motor nature; conspicuous pallor, marked reddening, especially upon stroking the skin, more rarely eruptions. Also disturbances of the *respiration* (difficulty in inspiration due to spasm of the muscles of respiration, Cheyne Stokes phenomenon) and of the *activity of the heart* (alteration of the frequency), further, narrowing and widening of the *pupils* may occur, especially in those cases in which the meningitis travels upward, reaching the medulla oblongata and affecting the centrum ciliospinale and the more superiorly situated centres for respiration and the nerves inhibiting the cardiac action, or affecting the nerves in contact with these areas. *If the inflammation spreads to the cerebral*

meninges, the symptoms of cerebral meningitis are added: Headache, vomiting, vertigo, delirium, coma, irritative and paralytic phenomena in the course of the cranial nerves, etc.

Seat of the Meningitis.—From the foregoing description it is readily seen that the symptoms of spinal meningitis must vary greatly according to the seat and distribution of the inflammatory process.

In favour of an affection of the *lumbodorsal portion of the pia* are: Pains concentrated and fixed in the back, upon the trunk, and radiating into the lower extremities, further a limitation of the spasm and paralysis, etc., to the previously mentioned areas, marked development of bladder disturbance, difficulty in respiration, due to spasmodic and paralytic conditions in the course of the muscles of respiration.

The latter are most markedly developed, provided the *cervical part of the pia* is the seat of the inflammation. In this condition there is present rigidity of the muscles of the back of the neck, markedly distributed radiating pains and hyperæsthesia, spasm, etc., especially in the upper extremity, and variation in the size of the pupil. Difficulty in deglutition, excessive slowing of the pulse or at times increase in pulse frequency, Cheyne-Stokes's phenomenon and vomiting, show that the meningitis has reached the region of the medulla oblongata.

In spite of the symptoms just enumerated, which are even somewhat pathognomonic, I should like to advise to make it a rule *never to diagnosticate a spinal meningitis with certainty if one of the recognised causes which gives rise to meningitis cannot be proved to be present.* In following this rule, in my experience, we are protected with greater certainty from making incorrect diagnoses. We shall therefore briefly refer to the *ætiology* of acute spinal meningitis.

Ætiological Diagnosis.—Primarily it should be observed whether any change has occurred in the vertebral column, which might give rise to an inflammatory process. A special search should be made for *purulent pleurisy, caries of the vertebræ, suppurative inflammation of the cellular tissue of the pelvis* (in puerperio) or deep *bedsores*. In the last-named cases *by contiguity* a (purulent) inflammation of the dura mater (pachymeningitis or an affection especially of the external surface of the dura mater, a peripachymeningitis) develops. Also in the opposite direction, from within outward, an acute meningitis may occur if in myelitis the inflammatory process secondarily becomes implanted upon the pia.

Secondly, we must reflect whether the existence of a general infection which has become localized to the spinal meninges, is probable or not. In this manner, *croupous pneumonia, acute rheumatic fever, enteric fever*, the acute exanthems, above all, however, *septico-pyæmia* may give rise to spinal meningitis. The virus of *cerebro-spinal meningitis* also appears exceptionally to concentrate itself upon the pia spinalis, as I had an opportunity of observing recently in cases of spinal meningitis which occurred in connection with an epidemic of cerebro-spinal meningitis, and not in a few cases. Putrid bronchitis, gangrene of the lung and bronchiectasis, in a similar manner in which they give rise to (cerebral and) spinal-cord abscesses, may also cause purulent spinal meningitis. All these infectious forms of meningitis are rare compared to those which arise due to the action of *tuberculosis*. Similar to the condition in epidemic cerebro-spinal meningitis, it is almost always only a part phenomenon of a general meningitis, in which, as a rule, basilar meningitis is so prominent that the simultaneously present spinal meningitis is not noted or is entirely overlooked. In some of these cases, the affection is obviously due to an

originally existing tuberculous focus in the lung, *and a continuous propagation of the poison through the pleura and intervertebral openings to the coverings of the cord* occurs, thence travelling upward and reaching the base of the brain. In other cases the opposite condition may be present, a *descending meningitis* may occur, in that the products of inflammation of the originally infectiously inflamed pia of the brain, mechanically sink to the bottom, and secondarily produce a spinal meningitis. In other cases, finally, the tuberculous meningitis may be limited to the pia of the spinal cord, as in one of my cases, in which the possibility of cure of the tuberculous meningitis could be proved by the autopsy findings.

If after the examination of the history and the closest investigation of the case, no points of support are found for the development of spinal meningitis by any of the previously mentioned ætiological conditions, only then is it permissible to consider fractures and luxations of the vertebræ, shock of the vertebral column, or the influence of refrigeration as the cause of the disease. If these cannot be brought into direct connection with the cause of spinal meningitis, their influence as predisposing factors, by producing a local damage to the tissue resistance, should be thought of (so that inflammatory and pus-producing causes which at other times are benign, may now exert their deleterious action).

The diagnosis of spinal meningitis, as well as the special character of the infection, in the individual case, has lately acquired greater certainty by the use of *lumbar puncture*, according to Quincke, which has already been frequently practised. In purulent meningitis pus is found in the fluid obtained in this way and in the tubercular form, in by far the greatest majority of cases; in the usually very clear cerebro-spinal fluid, tubercle bacilli have been demonstrated. The proof of the presence of these organisms is easiest obtained by a previous use of the centrifuge, or by the examination of the fibrin coagulations which are found in the fluid and which contain the bacilli in large amounts.

Differential Diagnosis.—Acute spinal meningitis is not rarely mistaken for other affections. *Rheumatism of the cervical vertebræ*, and also *rheumatism of the muscles of the back of the neck and of the back*, have in common with it stiffness of the neck and back, pain upon movements, as well as sensitiveness and spastic contraction of the affected muscles. Muscular twitchings and sensory disturbances of the skin, but above all the eccentric phenomena in the extremities, the disordered function of the bladder, the changes in the pupil, etc., usually also the fever, are absent, whereas severe and marked difficulty in respiration under some circumstances may also occur in simple muscular rheumatism, provided the affection is concentrated upon the respiratory muscles of the thorax.

From *tetanus*, spinal meningitis may as a rule be easily and correctly differentiated. In tetanus, the reflex irritability is enormously increased, trismus occurs from the beginning, eventually also spasms of the muscles of deglutition and there develops a rigidity of the muscles of the face, which is absent in spinal meningitis. On the other hand, in tetanus, the fever phenomena are absent (except the premortal temperature rise), the cutaneous hyperæsthesia, the paralysis, the changes in the pupil and the appearance of cerebral phenomena. The ætiology of the individual case may also determine the diagnosis; in favour of tetanus is the history of a wound having preceded the appearance of the disease by several days or weeks; this is however not absolute, as traumatic influences also play a part in the development of spinal meningitis, and this disease is occasionally of septico-pyæmic origin.

Acute spinal meningitis can but rarely be confounded with *hysterical*

spasmodic conditions. The great variation in the clinical picture of hysteria, the entire impression which the patients convey, the exaggeration in the description of their difficulty, with the very slight affection of their general condition as a rule, and the never-absent typical implication of the psychical element in hysteria, will quickly aid the practised diagnostician in determining the correct affection.

However the differential diagnosis between *acute myelitis* and spinal meningitis will often be difficult, especially as combinations of both affections frequently arise, and some of the symptoms occurring in the course of spinal meningitis are in fact due to an accompanying affection of the cord in the inflammatory process. In general, in myelitis, the *symptoms of paralysis* are prominent both in the motor and sensory areas, the trophic disturbances of the skin, the paralysis of the bladder, eventually also the increase in the reflexes, whereas the irritative symptoms, the hyperæsthesia, and the pains are less marked, especially movements of the vertebral column and in the extremities do not markedly increase the pain, above all however the *stiffness of the back* and the *rigidity of the muscles of the back of the neck* are absent, these symptoms giving a characteristic stamp to spinal meningitis.

CHRONIC SPINAL MENINGITIS

Chronic Spinal Meningitis.—The diagnosis of chronic spinal meningitis can usually only be made in so far as we are called upon to decide whether symptoms point to an inflammatory affection of the membranes of the spinal cord, besides the anatomico-pathological changes which produce chronic alterations of the cord substance. Much rarer are other modes of origin of chronic spinal meningitis to be considered: The development of the affection takes place from a preceding acute meningitis, occasionally it may occur as a substantive affection, being chronic from the onset, arising upon the basis of syphilis or chronic alcoholism. The symptoms which are important in the diagnosis have already been described under acute meningitis—only they are less developed, and arise more gradually: *stiffness of the muscles of the back of the neck and back, pain,* localized in the latter areas, *increased by movement,* eccentric pains and a sensation of weight in the extremities, girdle sensation, paræsthesia and hyperæsthesia of the skin, muscular spasms and contractures. In the later development of the affection *paralytic phenomena* become prominent: Anæsthesia, paralysis of the bladder and paraplegic symptoms. These latter symptoms may be increased by change of position especially while in the recumbent posture, by gravitation of the exudate, or by a more marked passive engorgement of the blood in the canal of the spinal cord. If compression of the roots by the exudate, or chronic thickening and coalescence occurs, atrophy of the paralyzed muscles develops (reactions of degeneration), also paralysis of the bladder, anæsthesia, and cessation of reflexes. It can only be surmised then whether the pia and arachnoid, or the dura is more implicated in this process. An inflammation of the external surface of the dura (pachymeningitis spinalis externa) is to be thought of if the development of the symptoms of chronic spinal meningitis occurs gradually (by contiguity), being added to those of caries of the vertebræ, with deep bedsores, or purulent condition of the muscles, which are all contiguous to the vertebral column.

In general, the diagnosis of chronic spinal meningitis is very uncertain, and the ensemble of the previously mentioned symptoms may only be expected to be well developed in the rarest cases. Comparatively most frequently, chronic spinal meningitis has been observed recently in the course of syphilis; in such cases coincident spinal meningitis and tabes dorsalis could be noted. Usually, besides the

syphilitic spinal meningitis, symptoms of brain syphilis are present, especially also the symptoms of progressive paralysis.

Pachymeningitis Hypertrophica.—There is a better foundation for the diagnosis of a special form of chronic spinal meningitis, which affects the dura mater and shows a very characteristic clinical picture. It is the variety first described by Charcot: *Pachymeningitis cervicalis hypertrophica* (cervicalis interna). In this affection upon the *inner surface* of the cervical spinal dura, as a result of chronic inflammation, callosities which may attain the thickness of ½ cm. are formed, which press upon the spinal cord and the nerve roots, thereby producing marked consequent phenomena.

Phenomena of the onset (stage of irritation) are: Severe pains and stiffness of the muscles of the neck, radiating pains, hyperæsthesia and paræsthesia in the arms, eruptions (herpes and pemphigus), desquamation and roughness of the skin of the upper extremities, further, although rarely, spasm and contracture in the muscles.

A gradual transition into the second stage now occurs (in the course of from eight to ten weeks); this is called the stage of *paralysis*, being characterized by the phenomena of motor paralysis, in the region of the upper extremity. Almost always does this paralysis affect partly the course of the ulnar and median nerves, whereas the radial nerve is usually exempt. This produces antagonistic preponderance of the radial innervation, *so that the hand is held in the position of dorsal flexion.* At the same time, both end phalanges of the fingers are bent (claw position). However, this contracture position of dorsal flexion only occurs provided the radial nerve is not affected, but, if this nerve be also implicated, permanent volar flexion of the hand is observed. The paralyzed muscles are *atrophic* and show reaction of degeneration; the skin becomes *anæsthetic* in areas. Further on in the affection, due to the pressure of the hypertrophic connective tissue upon the spinal cord, paralysis occurs even in the lower extremity. The cause of the latter is due to the pressure, which the anterior lateral columns of the cervical cord and the fibre tracts for the lower extremity, which run through these tissues, experience. The paralysis in the lower extremity differs from that occurring by pressure of the root fibres, occurring in the upper extremity, in that atrophy does not develop in the leg, the electrical reaction is normal and the tendon reflexes are increased. With this the other phenomena of compression of the spinal cord develop: Anæsthesia of the lower extremities, bedsores, paralysis of the bladder, etc.

Differential Diagnosis.—The altered conditions existing in the upper extremity, compared to those of the lower extremity, show with certainty that, in the interior of the cord of the cervical vertebra, a pathological process is present that has damaged the nerve roots and further on has even involved the cord substance. The muscular atrophy in the region of the upper extremity may resemble *progressive muscular atrophy and amyotropic lateral sclerosis.* But, in these affections, the irritative phenomena, the rigidity of the muscles of the neck, the pains and sensory disturbances in general, as well as disturbances of the bladder functions are absent, whereas on the other hand, the atrophy of the muscles of the lower extremity and bulbar symptoms are absent in hypertrophic cervical pachymeningitis. The differential diagnosis is more difficult between the disease in question and *cervical spondylitis* with secondary compression symptoms. In favour of spondylitis is the painfulness of individual vertebræ upon pressure, and even more so the deformity of the vertebral column, the less typical course of the disease, and the possible evidence of tuberculosis of other organs. However, the differentiation between hypertrophic pachymeningitis and *meningeal tumours* of the cervical cord is impossible, if we do not lay stress upon the somewhat slower development of irritative phenomena in the case of tumours and the simultaneous development of tumours in other parts of the body. But even then nothing but a probable diagnosis can be made.

Pachymeningitis Hæmorrhagica. — *Pachymeningitis hæmorrhagica* (interna) with the deposition of a fibrinous exudate upon the inner surface of the dura, in the subdural space. It is important in the diagnosis to note that the disease usually occurs combined with hæmatoma of the dura mater of the brain, and that both affections are the result of psychoses (dementia paralytica), and the abuse of alcohol.

The disease cannot be diagnosticated with certainty; but we should suspect its presence if in drunkards and paralytics, and especially in the simultaneous appearance of hæmatoma of the dura of the brain, the symptoms of chronic spinal meningitis appear. Suggestive are therefore stiffness and painfulness of the back, rigidity of the muscles of the neck, eccentric pains, contractures and weakness, besides hyperæsthesia or anæsthesia of the extremities, phenomena which are intercurrently *exacerbated* and occur in an acute manner with symptoms of pressure upon the nerve roots and the substance of the cord in those cases in which suddenly marked hæmorrhages occur from the pseudo-membranes.

Meningeal Tumours.—Their diagnosis will be discussed later on in describing tumours of the spinal cord.

DIAGNOSIS OF DISEASES OF THE SPINAL CORD SUBSTANCE

PRELIMINARY REMARKS

Degenerative Processes in the Spinal Cord.—Inflammatory processes in the spinal cord play an important part in diseases of the spinal cord. If we accept as the product of these processes, fatty degeneration of the nerve substance and—at least in the cases running a chronic course—proliferation of the glia tissue ("gray degeneration"—sclerosis), the greatest part of the affections of the cord belong to the category of chronic myelitis. Lately in the various diseases of the spinal cord, both the end products of the anatomical changes have been genetically separated, and the degeneration of the nerves in certain clinical pictures of diseases of the spinal cord (tabes, lateral sclerosis, etc.) has been looked upon as the most important primary condition, the glia proliferation as subordinate and secondary, and the processes due to this condition have been distinctly separated from the inflammatory changes. It may be looked upon as certain that, in the occurrence of degeneration of the nerve elements of the spinal cord, the composition of the ganglion cell plays a great part, as its destruction regularly results in a degeneration of its neurites. This condition of dependence dominates the course and the intensity of the individual degenerative processes. Regarding the origin of degeneration itself, it is immaterial whether inflammatory or other changes are present in the individual case which no longer allow the conserving influence of the ganglion cells upon individual fibre tracts.

System Diseases.—In a certain number of affections of the spinal cord, in tabes, lateral sclerosis, etc., certain fibre tracts which anatomically and functionally belong together (also ganglion complexes) are affected with great regularity.—"fibre systems" respectively "neuron systems," so that the unfortunate designation "*systematic*" spinal-cord diseases has been chosen for them, in contrast to other changes of the cord which are irregularly diffused in different areas through the length and breadth of the cord. Why in the special case this or that distinct division of the spinal cord should be affected, is beyond our comprehension, nor can we understand why certain toxic substances (lead, strychnine, ergot, etc.) damage functionally certain fibre tracts of the cord. Lately, it has become more and more probable that a hereditary feebleness of certain neuron systems plays an important part in the development of "system diseases."

The question to be decided primarily by the diagnostician in the individual patient is this, which functions are altered in the clinical picture, and to what portion of the spinal cord do they relate, and secondarily to consider the form of lesion that has occurred—i. e., to formulate the diagnosis of the functional disturbance so that it becomes an exact localized anatomical one.

Starting from this standpoint it will be advisable first to consider the more sharply defined affections, and, therefore, in general those more accessible to diagnosis.

TABES DORSALIS [LOCOMOTOR ATAXIA]

GRAY DEGENERATION OF THE POSTERIOR COLUMNS—LEUCOMYELITIS —POSTERIOR CHRONICA

Tabes dorsalis represents one of the best and longest-known partial affections of the spinal-cord substance, running a chronic-progressive course. The *anatomical* basis of tabes depends, in the main, upon a *degeneration of the posterior columns,* which, as Leyden recognised first, occurs in connection with *disease of the sensory-root fibres,* so that a degenerative process in the spinal cord takes place which follows the course of these tracts in a centripetal direction. The most important *clinical* signs of the affection, dominating the morbid picture, are *disturbances in the sensory sphere and the ataxia.*

The diagnosis in the majority of cases is easy, in that a certain complex of symptoms is almost pathognomonic of tabes. Difficulties can only arise in the very first stages of the disease, and occasionally in the later stages, if the typical picture of tabes becomes greatly modified, or the result of phenomena which are due to the development of the anatomical changes beyond the usual frame of the affection takes place, this causing the typical picture of the affection to be more or less altered.

Diagnosis of the First Stage of Tabes.—*Incipient tabes* can be diagnosticated as soon as *lightning-like* (so-called lancinating), paroxysmal pains, in the small of the back and in the lower extremity, occur, and the *tendon reflexes are absent.* These symptoms may exist for years without giving rise to ataxia; in spite of this, as the results of autopsies teach us, in such cases a typical degeneration of the posterior cord may exist. To these two initial symptoms usually a third symptom is added, *disturbances of the movability* and in the *width* of the *pupils,* this being a *reflex rigidity of the pupil* (in about one half the cases, also, even if rare, total, i. e., affecting accommodation as well as reflex conditions), *narrowing* (*myosis*) and *inequality of the pupils. In reflex rigidity of the pupil,* the pupils are usually more contracted than normal, reacting however still to accommodation, but not to the influence of suddenly appearing light (Arygll-Robertson phenomenon). The cause of these pupillary changes has not yet been definitely determined, especially as to whether they are of spinal or cerebral origin (arising from the region of the corpora quadrigemina), influencing the pupil reaction.

Eye Symptoms in Tabes.—Rarer than these three usually combined initial symptoms in tabes, there occur in the first stages of the disease *affections of the nerve of sight* and the *muscles of the eye,* which may occasionally even precede the just-mentioned spinal phenomena and that for a long time. For this reason, the diagnostic rule must be adhered to, not only in the later stages of tabes in which the diagnosis does not usually give rise to difficulties, but in every suspected case the muscles of the eye should be examined for paralysis and especially also the function of the organ of sight should be determined and an ophthalmoscopic examination made. The findings which are noted in tabes dorsalis are briefly the following:

Paralysis of the abducens and of the oculo-motor are both equally common; they usually occur suddenly and are not rarely incomplete, *may occasionally even disappear comparatively rapidly.* Paralysis of the *trochlear nerve,* however, is very

rare. Paralysis of the muscles of the eye is found in at least one half of the cases of affections of the optic nerve and exists as a rule for some time before the latter occurs.

The *functional disturbances* in *diseases of the optic nerve* are frequently ushered in by marked blinding phenomena or by hazy sight. Usually at first the contraction of the colour field is moderate; the early appearance of blindness for red and green is suspicious. The limits of the field of vision frequently show a zigzag form. These disturbances in general advance, and in from four to five months blindness may occur, which otherwise as a rule requires from one to three years.

Examination with the *ophthalmoscope* shows as a rare finding at the onset of the affection: The optic nerve somewhat reddened, slightly opaque and of a more gray colour. Much more frequently following this so-called inflammatory stage, *pale-gray discoloration of the optic nerve (gray atrophy) with sharply defined limitation and normally filled vessels* is noted; the latter appears somewhat narrowed, if gradually the gray colour which was primarily present, changes to a somewhat white gray. A change in the optic nerve papilla may be absent upon ophthalmoscopic examination in functional disturbances that are already quite marked.

Less Constant Initial Symptoms.—Whereas the lancinating pains in the stage of onset are as a rule limited to the lower extremities, cases also occur in which they are confined to the trunk and still rarer cases in which they appear concentrated upon the upper extremity. Symptoms, which may easily be overlooked, because they are as a rule but slightly indicated but which are nevertheless important as complementing the diagnosis of the first stage of tabes, arising at times earlier, sometimes later than the already-described initial phenomena are: Sensory disturbances of various kinds, anæsthesia (especially also in the form of a ribbon-like constricting streak upon the trunk), *paræsthesia*, especially formication, fur-like sensations upon the soles of the feet (sensation as if walking upon cotton, sand, etc.) and also in the hand (and as it appears, preferably in the course of the ulnar nerve), but above all the usually constant and greatly feared *girdle sensation*, i. e., the sensation of a hoop surrounding the trunk, the feeling of slight *lassitude* which appears even in the recumbent posture but is especially noted after prolonged walking or standing. Among the symptoms which occur early, there is also not rarely *weakness of the bladder* (retention and involuntary evacuation of urine), *sexual disturbances* (priapism, painful ejaculation, etc., later permanent impotence), and *paræsthesia of the rectum*, the sensation as if a wedge were in the rectum, etc., which may increase to such an extent that marked pain occurs and gives rise to the so-called *anal crises*. Similar conditions as in the nerves of the rectum also take place in the nerves of the abdominal organs and may also cause neuralgias, even in this stage of the disease. They are designated, following Charcot, with the happily chosen names of *crises*.

Crises.—The most frequent are the *gastric crises*—severe gastric pains and vomiting, occurring in paroxysms, rarely only vomiting of copious watery masses, or only the symptoms of gastralgia, lasting for hours or days, and then ceasing for weeks. *Intestinal crises* are rarer (colic, and uncontrollable diarrhœa), *nephrocrises* (attacks resembling renal colic, with diminished diuresis and transitory albuminuria), *urethral* and *vesical crises* (urinary pressure, retention of urine, etc.), *deglutition crises* (paroxysms of difficulty in deglutition), *laryngocrises* (nervous attacks of cough

with the symptoms of laryngospasm and later of posticus paralysis) and *cardiac crises* (increased pulse-rate, angina pectoris, syncope).

In keeping with the anatomical changes in the cranial nerves, and in the cerebrum, which occur in tabes, there are found occasionally, even early, also other symptoms, which shall be described later on: Besides changes in sight and paralysis of the oculo-motor nerves and abducens paralysis, progressive difficulty in hearing, trigeminus neuralgia, vertigo, apoplectiform and epileptiform attacks, etc., are noted.

Diagnosis of the Second Stage ("The Ataxic Stage").—By an increase of the previously mentioned constant, or of the rarer initial, symptoms, under continuance of the latter, the second stage of tabes occurs, which on account of the symptom which is particularly prominent in this stage, is known as the "*ataxic stage.*" The disease is now, having attained its full development and the characteristic ataxia having occurred, readily recognised even upon superficial examination. The disturbance is characterized by the fact that *in spite of the well-retained voluntary force, the nutrition and the electrical reaction of the muscles,* there is nevertheless marked interference with their functions, producing difficulty in gait, standing, etc., which may finally become entirely impossible. The cause of these phenomena depends, as Duchenne recognised, upon a *disturbance of co-ordination of muscular action.* The harmonious combined action of the muscles in movement, which presupposes a finely differentiated measure in the intensity of the muscular contraction, is disordered or has ceased entirely. A clumsiness in locomotion appears; the patient to assure himself of the position of his extremities, causes excessive movements in the joints, and these are all the more marked the greater the disturbance of sensation. The uncertainty increases markedly if the patient closes his eyes, in which especially, if the feet are brought into juxtaposition, a more or less marked swaying occurs (Brach-Romberg's symptom). This is in keeping with the peculiar gait of patients affected by locomotor ataxia: The legs are raised abnormally high, and brought down with the heel, first stamping the ground as it were (heel gait). It is particularly difficult for the patient to turn around suddenly or to rise abruptly from a sitting posture; in complicated muscular actions, such as dancing, standing upon one leg, etc., marked swaying of the body occurs. These difficulties are noted primarily in the lower extremity, but in the further course of tabes, the ataxia may also affect the upper extremity, so that finer manipulations: Writing, buttoning the clothes, etc., are uncertain, and in attempting a movement of the fingers in a straight line, it is found to be impossible.

Third, "Paralytic," Stage.—To this stage which similar to the first stage may last for years—I knew a case in which, from the onset of tabes almost forty years had passed, however the patient was still able to walk comparatively well, his occupation being that of a canvasser—finally *the third stage of tabes* is added, provided that intercurrent affections have not shortened the life of the patient, this stage being characterized by *paralysis of the legs,* complete *paralysis of the bladder, cystitis, bedsores,* briefly, by all the severe usual affections occurring in diffuse diseases of the spinal cord.

Spinal Cord.—In the latter instance, the
the posterior columns, but rather a
motor) pyramidal-lateral column tract.
Such a marked distribution of the
cord is rare, however, as well as degen
ventricle and in the aqueduct of Sylvius.
frequently degenerative changes occur in
other peripheral (sensory) nerves. The
questionably the posterior roots and the
at exception a very small portion, the
with its fibres, which do not arise from
posterior gray matter (compare Fig. 28,
process. Those fibres of the posterior roots
which traverse the external zone (radi
ly ascending in Goll's columns and partly
sensory collaterals to the gray substance.
's columns by collaterals (Fig. 28 and 30),
remain normal; however, it appears that
wastes, especially by degeneration of the
ach the ganglion cells of Clarke's columns

s in tabes, after what we have previously
the spinal cord, may be explained in the

Lastly, in its primary stages, an affection
in tabes is involved in different areas,
the posterior roots, which partly degenerate
secondarily by inflammatory processes in the
ction being the result. The *radiating*
ete. There are especially affected among
ed from the posterior roots: 1. the *ascend*
further upward into Goll's columns, 2.
there ascending into the cerebellar lateral
s presiding over coordination, *respectively*
posterior columns into the posterior horn
anterior horn (Fig. 28, *here*) and, 4. the
into the posterior gray matter to the col
tracts, by its interruption in the root
from its cell in the spinal ganglion, its
from the point of lesion, i.e., *the*
Goll's column). The condition is dif
tracts. As Clarke's ganglion cells, as is
represent the origin cells for the cere
tract, [illegible] ascending secondary
tracts. Nevertheless, the collaterals of
on the [illegible] the original focus
collateral [illegible] Clarke's columns; the
[illegible] up to the
posterior [illegible] collaterals (3),
[illegible] to the cells
Clarke's [illegible] atrophy

noted in the [illegible] lateral column
seen in [illegible] a change in the
ns, not a [illegible] reason for this in
tabes. It is true, the crossed ascend-
bundles [illegible] before their
[illegible] latter on they
substance, the column cells—i.e.,

with a second cell station and are preserved by the latter. Another such cellular influence also occurs in the fibres which are not crossed ascending in the posterior columns (principally supplying muscular sensation), but this occurs in the medulla oblongata, this being the reason that their degenerative field in Goll's column occurs in all cases in their entire length in the spinal cord.

Besides the changes in the funiculus cuneatus and Goll's column which have been mentioned there are found, as Lissauer has shown, as a typical expression of the degenerative process in tabes, also *disease of the fine root fibres of the marginal zone situated at the apex of the posterior horns* (Fig. 34, *r*, *lff*), which represent the most lateral fibres entering and the principal mass of sensory fibres which are crossed and ascend to the medulla oblongata. *As however the marginal zone as a rule only becomes diseased later than the root zone of the funiculus cuneatus*, it is evident that the fibres in question usually are able to conduct somewhat longer than those coming from the funiculus cuneatus which radiate into the gray substance.

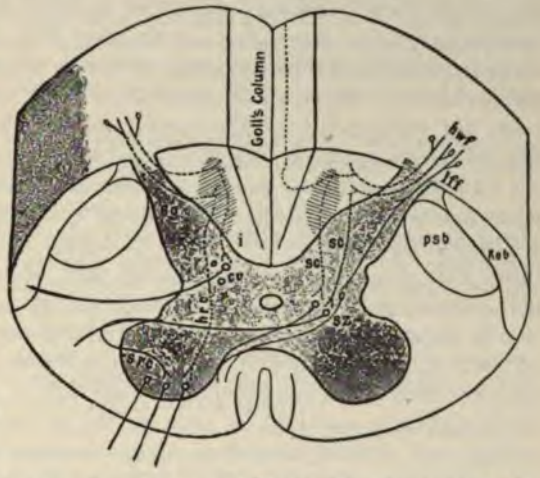


FIG. 34.—DIAGRAM OF THE PRIMARY DEGENERATIVE FIELDS AND OF THE SECONDARY FIBRE DEGENERATION IN THE FIRST STAGE OF TABES.

psb, Pyramidal lateral column tract; *ksb*, cerebello-lateral column tract; *hwf*, posterior root fibres; *lff*, laterally entering fine root fibres; point of first affection; *r*, border zone; *sg*, substantia gelatinosa; *i*, unaffected ventral posterior column field; *cv*, Clarke's columns; *sc*, sensory collaterals; *sz*, column cells; *hrc*, posterior column reflex collaterals; *src*, lateral column reflex collaterals.——intact . . . .degenerated fibres.

Lately the point of attack of tabes has been placed more peripherally in the peripheral sensory neuron, i. e., *in the cells of the spinal ganglion*, and, in fact, investigations directed to this point have discovered *degenerative changes* in the latter (usually but very slight ones). This makes it clear that the degeneration of the posterior root fibres and posterior fibre columns belonging thereto must be looked upon as secondary resulting phenomena of these changes in the spinal ganglion, only we must then expect also that a degeneration in the sensory peripheral nerve must occur, which has in fact been proved in these cases. It has not yet been sufficiently determined whether these are constant findings, and in how far the degeneration of the spinal ganglion must be developed to cause these secondary changes in the centrally and peripherally conducting nerve fibres, belonging to the spinal ganglion cells.

Upon the basis of the anatomical changes just described the explanation of some clinical facts may be attempted which at first sight appear difficult, occurring in the primary stages of tabes and that are important in the diagnosis. Apart from the lancinating pains which point to an irritation of the sensory-root fibres which radiate into the spinal cord, I shall only touch upon a conspicuous fact which has occurred to me, as well as certainly to every one that has had an opportunity of observing a great number of tabetic patients. Not rarely, in the onset of the disease, is there a striking contrast between the conspicuously slight development of

Details regarding Degeneration in the Spinal Cord.—In the latter instance, the degenerative process is no longer limited to the posterior columns, but rather a dissemination has occurred affecting the (motor) pyramidal-lateral column tract, eventually also affecting the anterior horns. Such a marked distribution of the process to the anterior portions of the spinal cord is rare, however, as well as degenerative changes in the floor of the fourth ventricle and in the aqueduct of Sylvius, or in the cortex of the cerebrum. More frequently degenerative changes occur in the optic nerve, in the pneumogastric and in other peripheral (sensory) nerves. The typical region of the tabetic process is unquestionably the posterior roots and the *posterior columns*, in which almost without exception a very small portion, the ventral posterior column field (Fig. 34, *i*) with its fibres, which do not arise from the posterior roots, but in the cells of the posterior gray matter (compare Fig. 28), remained unaffected by the degenerative process. Those fibres of the posterior roots are especially affected by the degeneration, which traverse the external zone (radiating root zone) of the cuneal columns, partly ascending in Goll's columns and partly giving off reflex collaterals or also special sensory collaterals to the gray substance, partly, finally, being connected with Clarke's columns by collaterals (Fig. 28 and 30). The ganglion cells of the latter naturally remain normal; however, it appears that the fine fibre network surrounding them wastes, especially by degeneration of the fibres and terminal arborizations which reach the ganglion cells of Clarke's columns (Lissauer).

The results of these typical changes in tabes, after what we have previously described regarding the fibre structure of the spinal cord, may be explained in the following manner:

The tabetic process is mainly and evidently, in its primary stages, an affection *of the peripheral sensory neuron* which in tabes is involved in different areas. The most frequent points of attack are the posterior roots, which partly degenerate primarily, and partly, as it appears, secondarily by inflammatory processes in the dura and especially in the pia, degeneration being the result. The ***(radiating) root zones of the cuneal columns*** degenerate. There are especially affected among the radiating fibres entering the spinal cord from the posterior roots: 1. the ***ascending fibres entering the cuneal column and further upward into Goll's columns***, 2, those *going to Clarke's columns* and from there ascending into the cerebellar lateral column tracts, especially those *root fibres* presiding over co-ordination, ***respectively the collaterals***, 3, those radiating from the posterior columns into the posterior horn and the *reflex collaterals running* into the anterior horn (Fig. 28, ***hrc***) and, 4, the radiating fibres from the root zone going into the posterior gray matter to the column cells. As the first-mentioned of these fibre tracts, by its interruption in the root zones of the posterior columns, is separated from its cells in the spinal ganglion, its tract degenerates secondarily towards the brain from the point of lesion, i. e., *the cuneal columns degenerate and farther up also Goll's columns.* The condition is different with the second of the affected fibre tracts. As Clarke's ganglion cells, as is well known, remain intact in tabes, and these represent the origin cells for the cerebellar lateral column tract fibres, naturally no reason exists for ascending secondary degeneration of the cerebellar lateral column tracts. Nevertheless, the collaterals of this second category of fibres degenerate, upon the tract between the original focus of disease (in the root zones of the funiculus cuneatus) and Clarke's columns; the sensory fibres which radiate into the posterior horns also degenerate (4) up to the point of their column cells, as well as the posterior column reflex collaterals (3). As a result of this, there appears in the region of the posterior horns up to the cells and especially in the surrounding areas of Clarke's columns a conspicuous atrophy and a scarcity of fibres of the gray substance.

As a secondary degenerative change is not noted in the cerebellar lateral column tracts and pyramidal tracts, there is also not seen in typical cases a change in the ground bundles of the anterior and lateral columns, nor is there a reason for this in the previously mentioned anatomical changes in tabes. It is true, the crossed ascending fibres in the anterior lateral column ground bundles are in contact before their entrance into the spinal cord with cells of the spinal ganglions, but farther on they come in contact with ganglion cells of the gray substance, the column cells—i. e.,

fibres also motor-ganglion-cell groups of the gray spinal
g together. To these motor-ganglion-cell complexes
ences are carried by *collaterals* from the most varied
terals which originate partly from sensory-root fibres,
fibres from the anterior lateral ground bundles, partly,
ar lateral column tract. These *centripetal* influences con-
rves of the skin, the muscles, the tendons and the joints car-
lion cells, may produce, partly as a result of the lately found
of these sensory collaterals with motor cells at various heights
a simultaneous muscular action reflexly, and may cause partly
l motor cells, by stimulation from the pyramidal tract, a sequence
vation and an intensity regulating the same, partly they may also
tive action.

tabes dorsalis the collaterals representing the co-ordinating apparatus
e., the ganglion cells of the gray spinal-cord substances *do not* usually
owever the centripetal collaterals which come in contact with them are
their conduction, this causing a member of the co-ordination apparatus
nate insufficiently. In ataxia, there is especially a disturbance of muscu-
particularly of joint, sensibility always present, whereas the sensation of
is but partly or not at all interfered with. The result of an insufficiency in
nduction, produced in this manner upon the various centripetal irritations, is
a which, according to its genesis, may be designated as *simple sensory ataxia.*
my opinion, this variety of ataxia is typical in tabes so long as the degenera-
ve process in the spinal cord is not disseminated beyond its usual borders: If this
occurs, the ataxia may also have other causes for its origin.

The simple assumptions which have been made so far only partly explain the complicated mechanism of co-ordination, but by no means completely. In the first place it has been determined that in lesions of Clarke's columns the cerebellar lateral column tracts degenerate secondarily centripetally, this giving rise to disturbances of co-ordination. The course of the cerebellar lateral column tract may be followed centralwards, through the corpora restiformia to the *cerebellum* and, in the latter, probably the middle posterior portion may be looked upon as the region in which the greatest part of the ganglia and the reflex fibres in connection with them (see cerebellum) are found, i. e., the elements serving co-ordination. We are entitled to the assumption that *in this (posterior central) portion of the cerebellum a point of collection for centripetal stimulations is situated which are communicated to motor tracts.* Into this region the previously mentioned cerebellar lateral column tract fibres radiate, first through the corpora restiformia, and also fibres from the posterior column, especially to the superior worm and from thence communications go off to the roof nucleus. The centripetal stimulations which have gathered here, may be carried to Deiter's nucleus and from thence upon recently discovered centrifugal tracts to the motor cells of the spinal cord. Further on, fibres from the corpus dentatum passing through the cerebellar peduncles go to the red nucleus of the tegmentum and to the thalamus opticus, between which and the parietal brain further fibre communications exist. Further, if we reflect that there is nothing to prevent a connection of the latter with the frontal brain and the temporo-occipital brain, from which parts of the upper surface of the brain, the frontal and temporal pontal tracts going to the cerebellum, originate, it is at least likely that this entire complex of fibre tracts belongs together functionally and serves co-ordination, all the more as in pathological lesions of all of these parts, more or less marked ataxia, especially a difficulty in maintaining the *equilibrium of the body*, is noted.

The maintenance of the equilibrium of the body unquestionably in all cases depends upon the combined uniform action of *numerous* muscles. If sufficient co-ordination is to occur, a confluence of the various sensory irritations and the action of their regulating power upon a great number of motor tracts is necessary. This is only possible *above the spinal cord*, and unquestionably happens for the greater part in the principal co-ordination tract previously described.

Among the sensory *centripetal stimulations* the confluence of which is made up for the greatest part by the apparatus of co-ordination, besides the sensory irritations

the sensory disturbances of the skin, on the one hand, the absence of the tendon reflexes and marked disturbances of co-ordination, on the other hand. After what we have just mentioned regarding the anatomical changes in tabes, this becomes somewhat clear. A part of the sensory-root fibres, especially those concerned with the conduction of tactile sensation (which probably come in contact in the gray substance with column cells, and from there crossing, enter the anterior lateral ground bundles and ascending), these fibres may as we have seen (compare also Fig. 34, *lff*) remain intact in the initial stage of tabes, whereas those posterior column fibre-complexes which surely serve to a lesser degree for the above conduction, but principally for the conduction of the muscular sense, represent the primarily diseased portions of the spinal cord.

Also the behaviour of the *reflexes* has become somewhat clearer after what has been mentioned. In general the *cutaneous reflexes* in tabes are *not markedly disturbed.* This is explained by the circumstance that their reflex arcs, evidently for the greatest part, are situated in areas of the central nervous system, above the spinal marrow, and the sensory tracts leading to them, at least in the onset of the disease, are partly intact. Very frequently, in the preataxic stage of the disease, the reflexes of the abdominal coverings are increased. In contrast to this, as is well known, the *tendon reflexes* are lost as soon as the original area of affection in the spinal cord attacks the reflex collateral going to the anterior horns (Fig. 34, *hrc*). As the arc for the patella tendon reflex transverses the lumbar cord and as the tabetic process in this area is usually primary and most developed here, it is self-evident that the patella tendon reflex almost invariably is absent, but it is also evident that the same must be retained and in fact is retained provided the tabetic process in rare exceptional cases only affects the cervical cord, in which case the tendon reflexes of the upper extremity are absent.

A special description is required for a very interesting symptom, most important in the diagnosis, yes, even dominating to a certain degree the entire process—the *disturbance of co-ordination.* The explanation of its origin has for a long time occupied numerous investigators, and has given rise to the development of the most varied theories.

Genesis and Diagnosis of the Disturbances of Co-ordination.—By co-ordination we understand the combined action of certain muscles producing a uniform action. It is obvious that any explanation of the *disturbance of co-ordination* is impossible so long as we do not possess exact knowledge of the *normal* process of co-ordination. We are however not fully clear regarding the latter; but the results recently obtained by anatomical and physiological investigation, as well as the clinical facts gained at the bedside in certain directions, give us at least some points of support, and in the following explanation an attempt will be made to analyze the mechanism of co-ordination.

The innervation of the various muscles, which have for their purpose the fulfilment of a uniform action in a regular sequence, and which act together in a definite measure of intensity in contraction, occurs in certain areas, respectively in special tracts, of the central nervous system. Let us proceed from the frequently quoted fact that every nerve of the extremity receives nerve fibres not from one, but from several spinal-cord roots, further, that probably in one root the fibres for those muscles which usually act co-ordinately arise together and that there are in relation

to *these synergistic nerve fibres also motor-ganglion-cell groups of the gray spinal cord substance which belong together.* To these motor-ganglion-cell complexes unquestionably sensory influences are carried by *collaterals* from the most varied regions, especially by collaterals which originate partly from sensory-root fibres, partly ascending sensory fibres from the anterior lateral ground bundles, partly, finally, from the cerebellar lateral column tract. These *centripetal* influences conveyed by the sensory nerves of the skin, the muscles, the tendons and the joints carried to the motor ganglion cells, may produce, partly as a result of the lately found anatomical connection of these sensory collaterals with motor cells at various heights of the spinal cord, a simultaneous muscular action reflexly, and may cause partly upon the *centrifugal* motor cells, by stimulation from the pyramidal tract, a sequence of muscular innervation and an intensity regulating the same, partly they may also produce an *inhibitive* action.

Primarily in *tabes dorsalis* the collaterals representing the co-ordinating apparatus are affected, i. e., the ganglion cells of the gray spinal-cord substances *do not* usually degenerate, *however the centripetal collaterals which come in contact with them are damaged in their conduction*, this causing a member of the co-ordination apparatus to functionate insufficiently. In ataxia, there is especially a disturbance of muscular, and particularly of joint, sensibility always present, whereas the sensation of the skin is but partly or not at all interfered with. The result of an insufficiency in the conduction, produced in this manner upon the various centripetal irritations, is *ataxia* which, according to its genesis, may be designated as *simple sensory ataxia. In my opinion, this variety of ataxia is typical in tabes so long as the degenera*tive process in the spinal cord is not disseminated beyond its usual borders: If this occurs, the ataxia may also have other causes for its origin.

The simple assumptions which have been made so far only partly explain the complicated mechanism of co-ordination, but by no means completely. In the first place it has been determined that in lesions of Clarke's columns the cerebellar lateral column tracts degenerate secondarily centripetally, this giving rise to disturbances of co-ordination. The course of the cerebellar lateral column tract may be followed centralwards, through the corpora restiformia to the *cerebellum* and, in the latter, probably the middle posterior portion may be looked upon as the region in which the greatest part of the ganglia and the reflex fibres in connection with them (see cerebellum) are found, i. e., the elements serving co-ordination. We are entitled to the assumption that *in this (posterior central) portion of the cerebellum a point of collection for centripetal stimulations is situated which are communicated to motor tracts.* Into this region the previously mentioned cerebellar lateral column tract fibres radiate, first through the corpora restiformia, and also fibres from the posterior column, especially to the superior worm and from thence communications go off to the roof nucleus. The centripetal stimulations which have gathered here, may be carried to Deiter's nucleus and from thence upon recently discovered centrifugal tracts to the motor cells of the spinal cord. Further on, fibres from the corpus dentatum passing through the cerebellar peduncles go to the red nucleus of the tegmentum and to the thalamus opticus, between which and the parietal brain further fibre communications exist. Further, if we reflect that there is nothing to prevent a connection of the latter with the frontal brain and the temporo-occipital brain, from which parts of the upper surface of the brain, the frontal and temporal pontal tracts going to the cerebellum, originate, it is at least likely that this entire complex of fibre tracts belongs together functionally and serves co-ordination, all the more as in pathological lesions of all of these parts, more or less marked ataxia, especially a difficulty in maintaining the *equilibrium of the body*, is noted.

The maintenance of the equilibrium of the body unquestionably in all cases depends upon the combined uniform action of *numerous* muscles. If sufficient co-ordination is to occur, a confluence of the various sensory irritations and the action of their regulating power upon a great number of motor tracts is necessary. This is only possible *above the spinal cord*, and unquestionably happens for the greater part in the principal co-ordination tract previously described.

Among the sensory *centripetal stimulations* the confluence of which is made up for the greates part by the apparatus of co-ordination, besides the sensory irritations

which are carried to the skin, the muscles, the tendons, etc., those *conveyed by the optic nerve* play a chief *rôle*. They may, to a certain extent, substitute the influence of the other sensory irritations upon co-ordination, and the insufficient equilibrium of the body in co-ordinative disturbances, in the most cases, is very much more prominent when the eyes are closed and the regulating influence conveyed by the act of sight is absent.

How all the previously mentioned centripetal stimulations are conveyed to the motor elements, regulating their action, and affecting the complicated movements, may only be presumed at this time. According to my opinion, their action cannot be explained otherwise than that they reflexly stimulate the motor cells, on the one hand, and, on the other hand, inhibit their stimulation, whereby partly muscle action complexes are supplemented, partly voluntary stimulations restricted in a distinct measure. Whether this occurs in the anterior horn cells and in the cerebral nerve nucleus or, when the regulation occurs more under the influence of the will, the stimulation of the ganglion cells of the pyramidal tract in the central convolutions being influenced by the sensory stimulation reaching the cerebrum, in the manner previously described, cannot be positively determined.

From what has been said it is apparent that co-ordination is a very complicated process, the individual constituents of which have by no means been determined with certainty. Very probably, only by careful clinical observation can we know what influence the lesion of certain ganglion formations has in the act of co-ordination, and how centripetal or centrifugal fibres act upon the particular variety of ataxia. For the time being there is still much that is hypothetical regarding ataxia, and it is advisable, in utilizing even a very marked, well-developed ataxia, as the basis of a topical diagnosis of individual diseases of the nerves, to be very careful.

Rarer Symptoms and Complications.—Besides the well-characterized symptoms which determine the diagnosis of tabes, which have been mentioned, there occur in individual cases rarer or less marked phenomena, the knowledge of which is necessary to complete the diagnosis and to make it certain.

Symptoms occurring in the Motor Tract.—Among the *motor* symptoms, there is very usually a certain *flaccidity of the muscles* during passive and active movements (raising the leg from the knee towards the pelvis). The reason of this *hypotonia* may depend upon a diminution of the "muscle tonus," which itself is the result of the stimulations which are conveyed to the anterior horn cells through sensory fibres. If the latter, which is the case in tabes, becomes incapable of conduction, the normal tone of the muscle suffers. Rarely are pareses noted in the lower extremity, involuntary contractions, especially during sleep, nor *paralyses in the course of individual nerves,* especially in the peroneus and radial nerves, the facial nerve, the hypoglossal, glosso-pharyngeal, vagus and spinal accessory (marked difficulty in deglutition, paralysis of the recurrent laryngeal, atrophy of the sternocleidomastoid and trapezius, increased frequency of the pulse) or paralyses of the nerves supplying the ocular muscles (ptosis, strabismus, etc.). These paralytic conditions, according to autopsy findings, provided simple paralysis of the extremities is found, are either due to an advancing degeneration in the pyramidal tracts, or, if it be a case of amyotrophy, they are due to a disease of the anterior horn cells (communicated by the reflex collaterals?) or, again, to a superadded degenerative *neuritis* affecting the spinal and cerebral nerves. Relatively frequent is there a symmetrical atrophy of the muscles of the foot and also

of the muscles of the hand found with secondary claw position, due to a degeneration of the motor peripheral nerve of the extremity. It is somewhat characteristic of the paralyses due to peripheral neuritis that they may disappear if, as in peripheral neuritic processes, a regeneration of the nerve occurs in the course of time. Should the degeneration continue, the permanent result is a consecutive muscular atrophy and a disappearance of electric contractility. Hemiplegias are also, from time to time, noted in the course of tabes. They partly show no anatomical lesion, but may present vascular changes with thromboses (probably of a syphilitic nature).

Sensory Symptoms.—As the motor, the *peripheral sensory* nerves may also, in the course of tabes, be affected by a degenerative change. This may either occur as starting from the degenerated spinal ganglion cells, or, if the latter are intact, in that the central portion of the peripheral sensory neuron in which the damage in tabes has occurred, although to a slighter extent, is also to be looked upon as the cause. The affection of the latter in some cases even appears to have preceded that of the posterior roots and posterior columns. Further there may arise, in the course of the *trigeminus,* neuralgias, anæsthesias, and paræsthesias (frequently the spinal trigeminus root is degenerated), and ataxia of the muscles of mastication may occur, or from *implication of the acusticus* progressive difficulty in hearing may appear. Also disturbances in *smell* and *taste,* as a result of the tabetic process, are noted in rare cases. Further details occurring in the *sensory sphere,* besides those which have already been mentioned, are: *Double sensations* (the patient on being pricked with a needle observes at first only the tactile sensation, later only the sensation of pain), *partial sensory paralyses,* i. e., intact condition of individual qualities of sensation with almost complete absence of others, for example, marked anæsthesia of the skin, with active appreciation of differences in temperature, *analgesia* with the presence of marked irritation of the skin showing a preservation of the conception of weak irritations, *analgesia* on pressure upon the superficial branches of the ulnar and perineus nerves, disturbances of the *locality sense,* especially noted in testing the sensations of movements, etc. Especially marked very frequently is a *slowness of sensory conduction,* so that frequently several seconds pass before the patient perceives a cutaneous irritation. All these symptoms, unquestionably, depend upon the intensity and distribution of the tabetic process in the sensory tract.

Trophic Disturbances.—The *vaso-motor,* respectively the *trophic disturbances,* which occur in the course of tabes, require special mention. Exanthems (herpes, pemphigus, etc.), hyperidrosis, alopecia and falling out of the nails, are, upon the whole, rare phenomena. More frequent are bedsores, noted in the last stages of tabes, and of special interest are the *arthritic affections.* The arthropathies, first noted by Charcot occurring in connection with tabes, most frequently affect the knee joint, less often the shoulder, elbow and hip joints, or even the vertebral articulations. The arthritic affection occasionally may be multiple, but what is especially characteristic is that pain is *entirely absent.* The affected joint swells acutely, the surrounding tissue of the joint occasionally is markedly infiltrated; in spite of this however, palpation and passive movements of the joint do not produce pain. After a shorter or longer period, the swelling may gradually disappear, function and appearance of the joint again become normal. In other cases, especially in the late stages of tabes, the characteristic phenomena of arthritis deformans, more rarely

loose joints, etc., occur. As a cause of these arthropathies, we may not, as was previously believed, suspect distinct changes in the gray substance—autopsies have not confirmed this supposition. This much is certain that locomotor ataxia produces a predisposition to arthritic involvement; probably this is due to interruption in conduction and to degeneration of the sensory nerves supplying the joints, so that trauma, twisting the joint, etc., permit a more marked reaction and favour the further development of the changes arising in the joint. Upon the same basis, i. e., due to neuritic affection, the rarefaction of the tissue of the bones, producing spontaneous fractures, the likelihood of a dissolution of the tendons, and the affection designated by the name of malum perforans pedis [perforating ulcer of the foot] which occur in tabetic individuals, often as an initial symptom, may be referred. This ulcer appears especially upon the plantar surface of the foot, consisting at first of a simple thickening of the epidermis, which later may deeply invade the tissues reaching the bone, and showing great resistance to healing.

Ætiology.—The *ætiology* of tabes also furnishes points of support for the diagnosis. Among the usual predisposing causes of tabes: Refrigeration, sexual excesses, trauma (perhaps occasionally due to an ascending neuritis), infectious diseases, especially *syphilis*, the latter has been considered to be by far the most important factor in the production of tabes. By some authorities a previous syphilitic infection is looked upon as the *only* cause of tabes, this, however, is an exaggeration of the fact that patients affected by tabes, as a rule, have acquired syphilis in previous years. The *inherited neuropathic tendency* does not play a great part in tabes, at most, in so far as the predisposition to tabes appears to be greater and the course of the affection more rapid. Finally, Edinger has lately brought up the old widely distributed opinion that bodily overexertion may cause tabes, and has furnished an experimental support, in that he produced a progressive degeneration of the posterior roots and posterior columns in rats that he had caused to become anæmic and had forced to undergo severe muscular exertion.

Differential Diagnosis.—Upon observation of the diagnostic criteria mentioned, in a superficial examination the diagnosis of tabes, even in the initial stage of the disease, may be made with ease and certainty. But sufficient cases arise in which a confusion with other affections is possible. Primarily, there are *rheumatic, neuralgic affections of the lower extremities* (above all *sciatica*), which, if they are bilateral, always occasion a suspicion of tabes, as neuralgias are almost exclusively unilateral diseases. The condition of the tendon reflexes, and an examination of the eyes, usually at once removes all doubt regarding the diagnosis. This is also the case, if in patients, not during the course, but, as not rarely occurs, at the onset of tabes, the symptoms on the part of the internal organs, especially the *gastric crises*, with their paroxysmally arising attacks of abdominal pain and vomiting, so dominate the scene that other nervous symptoms are entirely secondary. These patients, under all circumstances, regularly believe that they have a gastric affection, and are sustained in this by the physician who is not careful in his examination: An *intestinal crisis* may also be an initial symptom of tabes, and disguise the nervous symptoms: In one of my cases, for six years there was *colic*, constipation and peristaltic unrest of the intestines, these were the only complaints of the patient —the examination at once cleared the situation, as the tendon reflexes were absent, the pupils reacted poorly, and unquestioned ataxia was present. There are also other initial symptoms: The weakness of the bladder, severe attacks of vertigo and migraine, recoveries from apoplectiform and epileptiform attacks, above all, *disturbances of sight*, which lead the patients to

consult a physician, the symptoms appearing as if belonging to other affections.

Pseudo-Tabes.—On the other hand, it is not rare that patients themselves believe that they are affected by tabes, on account of wandering pains in the small of the back and the extremities, lassitude, formication, numb sensations in the soles of the feet, vaso-motor disturbances, vertigo, especially also on account of a beginning impotence, hypochondriacal symptoms, etc., but are nevertheless not tabetic, as an examination readily reveals, if the tendon reflexes are retained, even being increased, the pupils react normally, and no sign of ataxia is present. Examples of such cases of pseudo-tabes, "*spinal neurasthenia*" (if the vertebræ are sensitive upon pressure, some physicians prefer to describe this morbid condition by the name of "spinal irritation"), are usually found in persons that are overworked or have committed sexual excesses, or the condition may be due to the abuse of alcohol and nicotine, their nervous functions having suffered in consequence. Strümpell has seen in workmen, in tobacco factories, tabes-like conditions ("nicotine-tabes") which had in common with tabes, rheumatoid pains, the absence of tendon reflexes, and even the reflex rigidity of the pupil, but nevertheless differed from tabes by the presence of tremor and an increase in the cutaneous reflexes. An *engorgement of the venous hæmorrhoidal plexus* that is in connection with the anterior sacral plexus, and with the veins of the spinal cord, may produce spinal irritation without organic changes of the spinal cord and give rise to conditions which resemble the symptoms of tabes, but, nevertheless, differing from them in the most important objective phenomena. If the tendon reflexes are preserved, if the reaction of the pupil is normal, and no signs of disturbance of muscular sensation is present, the diagnosis is clear, but even if the tendon reflexes are minus, as occasionally happens in such patients, the variation in the clinical picture, the dependence of the symptoms upon the filling of the rectum, the marked improvement brought about by the administration of purgatives, or a "cure" in Kissingen, etc., will show the diagnosis to be "*hæmorrhoidal*" *pseudo-tabes.*

Arthropathies, especially of the Vertebral Articulations.—Diagnostic doubts may arise provided *arthropathies* do not occur late in the course of tabes but, as is occasionally the case, as an early symptom. In such cases, the *absence of pain* of the arthritic affection points to the origin of the same as due to a tabetic process. The diagnosis becomes more certain if this symptom and the concentration of the arthropathy upon the knee-joint influences the physician to look for the other initial symptoms, and proves their presence, besides the arthritic affection. The differential diagnosis becomes more complicated if the arthropathy should exceptionally affect the *vertebral articulations*—upon the whole a very rare occurrence—giving rise to a curvature of the vertebral column. In such cases a (tuberculous) *vertebral affection with secondary compression of the spinal cord* may come into question, all the more, as the lancinating pains, the paræsthesia, the paralysis of the bladder, etc., are due to pressure paralysis, on account of the irritation of the posterior nerve roots in common to both affections. However, in favour of tuberculous spondylitis with compres-

sion of the spinal cord are the painfulness of the affected vertebræ (especially on movement of the vertebral column), the increase in the tendon reflexes which in spite of paralysis of the lower extremity remains to the end, the frequently induced *spastic* character of the paralysis, and the retardation of the sensory disturbances, alongside of the paralytic phenomena, which will not allow the diagnosis to be doubtful very long.

Multiple Sclerosis.—The diagnosis becomes more difficult if in *multiple sclerosis* the degenerative area especially attacks the posterior columns, this producing tabetic symptoms in the course of this affection which may then become prominent. In such instances, the consideration of the presence of other symptoms not belonging to tabes, the intention tremor, the disturbance in speech, and the nystagmus, raise the suspicion that in such cases the tabetic symptoms are only *part phenomena* of a multiple sclerosis, although, and this should be well remembered, both the last-named symptoms also occasionally occur in the course of ordinary tabes (not symptomatic), and also the cerebral symptoms arising in multiple sclerosis: Psychical disturbances, dementia paralytica, and apoplectiform attacks, not rarely happen in the clinical picture of tabes. Neither does the *ophthalmoscopic examination* offer any certain points of support; it is said that in multiple sclerosis only an incomplete change of colour occurs in the optic-nerve papillæ, i. e., being developed either in its inner or outer half. In the main, however, the findings in both diseases are the same, but the *functional disturbances* may prove of importance in a diagnostical respect, as these occur in sclerosis, and consist particularly in the appearance of a *central*, absolute or relative, *scotoma*. In the course of sclerosis, an improvement in the disturbances of sight is said to take place, blindness occurring in usually but one eye, and even then being transitory.

Upon the whole, it very rarely happens that we are called upon to make a differential diagnosis between tabes and disseminated cerebro-spinal sclerosis, as in sclerosis only exceptionally tabetic symptoms are prominent, and usually, besides nystagmus and disturbance in speech, the tremor occurs which gives the entire pathological picture its characteristic appearance.

Peripheral Pseudo-Tabes, Neuritis.—Greater difficulties arise in those cases of *multiple neuritis* which run their course with disturbance of sensation and with ataxia, and which, as has been previously remarked, may frequently closely resemble tabes. I must refer for the differential diagnosis between both these affections to what has been explained in the diagnosis of neuritis (which see); I shall only mention here, briefly, that in neuritis the pains, sensory disturbances of all kinds, the visceral crises, the absent tendon reflexes, and, above all, the ataxia, especially in the case of *alcoholic* neuritis, produce a tabes-like clinical picture. But it is usually not difficult, upon close examination of the case, to recognise tabes, in contrast to polyneuritis, in that in the latter the *reflex pupillary rigidity* (almost exclusively) *is absent*, the gait being more that of a paralysis (especially of a peroneal paralysis) and *atrophic muscular paralyses* with reactions of degeneration develop after some time; finally, that in multiple neuritis also

optic neuritis may occur, which is rare in the symptom-complex of tabes. Especially characteristic, however, in the tabetic process is the *progressive character* of the affection, whereas the symptoms of neuritis are susceptible of rapid improvement. This last fact must be taken into consideration if in the course of tabes, as has lately been observed, neuritis occurs, which especially by the transitory character of its consequences shows itself as a complication of the tabetic process, which is not liable to show improvement. Furthermore, also, the consideration of the ætiology in the individual case, the acute development of the disease, in connection with infectious diseases, and above all the observation of the important ætiological factor in *"peripheral pseudo-tabes,"* the *abuse of alcohol,* render the diagnosis more easy. Some prominent secondary phenomena which occur in the picture of alcoholic neuritis, such as the tremor, and the specific colouring of the psychical phenomena in drunkards, finally also the good results obtained by the withdrawal of the alcohol in questionable cases, direct the diagnosis correctly.

Finally, tabes shows some symptoms which resemble those of another affection, Friedreich's *ataxia,* an affection which was formerly looked upon as a variety of tabes, lately, however, has been separated from tabes as an individual affection. We must therefore describe this affection separately.

HEREDITARY ATAXIA—FAMILY ATAXIA—FRIEDREICH'S DISEASE

The title *"hereditary ataxia,"* which is also called, after the discoverer of the malady, *"Friedreich's disease,"* includes various affections in which an *hereditary deficient capacity of the central nervous system,* especially of the spinal cord, the medulla oblongata, respectively of the cerebellum, occurs, in consequence of which, during the time of growth of the body, a functional weakness occurs in certain directions, which becomes prominent. The affection, as a rule, attacks several children of the same parents (family ataxia) and is usually observed in the first decade of life, or during the period of puberty, apparently without cause, or in connection with recovery from some infectious disease, a marked psychical emotion, masturbation, etc.

Clinical Picture.—According to the involvement of the spinal cord, or of the cerebellum in particular, the morbid picture varies somewhat, so that lately two varieties have been described, the usual Friedreich's ataxia (also called "spinal") and the cerebellar hereditary ataxia. *The phenomenon which dominates the clinical picture is a markedly developed ataxia.* This shows itself, at first, in an uncertainty of gait; the patients walk with the legs widely separated, and sway markedly, in that the trunk and head continuously balance forward and backward, so that the gait resembles that of a person under the influence of alcohol. If the patients close their eyes, this uncertainty sometimes increases; this, however, in contrast to tabes, is by no means always the case. The ataxia does not only show itself in walking, but also in sitting and standing ("static" ataxia), and not only in the lower, but also in the upper extremities, in grasping for objects which

are held before them and so on. As further signs of ataxia, there are usually noted disturbances of speech, in the form of slow, scanning, explosive manner of speech and nystagmus. A further symptom of hereditary ataxia, also occurring in tabes, is an *absence of the patella tendon reflex,* whereas *sensory disturbances* are absent, or at most only insignificant; especially do the patients not complain of paræsthesia or pain. There are also absent, in contrast to tabes, disturbances of bladder, of the optic nerve, reflex pupil disturbance, and the visceral crises. Also in the *motor* sphere, for a long time, no alterations are noted; the strength is maintained and the muscles show no marked atrophy. Conspicuous, however, is a *hyperextension of the toes, especially of the great toe, with an equinus position of the foot.* The vertebral column in most cases shows a deviation, in the form of a *kyphosis* or *scoliosis.* Frequently the patients complain of *vertigo.* This in general is the typical clinical picture; but deviations also occur and a rigid adherence to individual portions of the symptom-complex is not advisable in the diagnosis, especially as the affection has by no means been determined with certainty in all of its phases up to the present time.

Nature of the Disease.—If we compare the previously mentioned disturbances of function with the anatomical findings, there are noted in the spinal cord, besides glia proliferations, wide-spread degenerative, atrophic changes, affecting distinct tracts: Primarily disease of the posterior columns, especially of Goll's columns, simultaneously disease of Clarke's columns, and the cerebellar lateral column tracts belonging thereto, as well as of Gowers's bundles, and finally also in the region of the pyramidal lateral column tracts. It occurs comparatively often that the posterior roots have also been found affected. Besides diseases of the fibre tracts previously mentioned which have been included as a "combined system disease," Schultze was the first to notice an *exquisite smallness of the spinal cord and of the medulla oblongata,* and this observation has since then been frequently confirmed. The inhibition of development of the spinal cord and of the medulla oblongata perhaps furnishes the key to the explanation of the difficulties still offered in the symptoms of the affection. It is possible that such a poorly developed central nervous system is insufficient for the requirements which arise in the course of time and (in the sense of Edinger's compensation theory) degenerates in those tracts which most frequently are utilized during life, especially in those tracts serving the equilibrium of the body, the tendon-reflex tracts, and certain other sensory and motor tracts. This degeneration would then be due to a relative overexertion, or that a further exhausting condition, as an intercurrent infectious disease, etc., is added to it, and the ball set in motion. Satisfactorily to explain the individual symptoms is, for the time being, impossible.

Cerebellar Form.—From the *spinal* form, a *cerebellar* form may be differentiated, as already remarked, by the clinical material at our disposal at present, in which the inhibition of development is limited to the cerebellum, as proven by various autopsies. In such cases, the swaying of the trunk in walking and standing is especially marked, but is not increased by closing the eyes; the upper extremities, only late in the disease, becoming ataxic. With this there are disturbances in speech, not rarely

also intention tremor, choreiform movements and vertigo. *In contrast to the spinal form, the patella tendon reflex is retained or even exaggerated,* sensory disturbances and trophic changes are absent, especially equinus and scoliosis, whereas optic atrophy, rigidity of the pupil and nystagmus occur in the cerebellar form.

Combinations of other forms certainly occur, and in cases of "hereditary ataxia" the diagnosis must be so formulated that, upon the basis of the symptoms described, there should be added whether the cerebellum or the spinal cord are more involved in the special case.

Differential Diagnosis.—Mistaking hereditary ataxia for other diseases of the nervous system is possible. From *tabes* this disease differs, as has already been mentioned, and not only the cerebellar but also the spinal form of the disease, very markedly—the latter, in contrast to tabes, shows absence of the disturbances of sight, the rigidity of the pupil, the bladder disturbance, and the visceral crises, also marked sensory disturbances and pains, whereas both affections have only in common ataxia and the disappearance of the tendon reflexes.

More difficult is the differential diagnosis between family ataxia and *multiple sclerosis,* with which it has quite a number of symptoms in common, namely, the uncertainty in movements, the oscillations of the head and trunk, the nystagmus, the disturbances of speech, etc. Also, the onset of both affections takes place during the period of early childhood, their course being specifically chronic. However, there are nevertheless important differential points, especially on the part of the spinal form, regarding the increase of the patella tendon reflexes, the spastic pareses, the changes in the optic nerve and the disturbance in the function of other cranial nerves, in the intercurrent apoplectiform attacks, constrained laughing and crying, and in the psychical alienation—morbid phenomena which are foreign to hereditary ataxia.

Other forms of ataxia such as occur following the infectious diseases, especially after attacks of diphtheria, are differentiated by their acute course and their susceptibility of cure, from hereditary ataxia.

Finally, the differential diagnosis between the cerebellar form of hereditary ataxia and *other cerebellar affections* may arise. Here, the development of the disease in late life, the appearance of headache and vomiting, and eventually the symptoms indirectly dependent upon the growth of the cerebellar focus (cranial-nerve paralysis, diabetes mellitus, stasis pupil, etc.) are to be noted, symptoms which are opposed to cerebellar hereditary ataxia and are much more in favour of disease of the cerebellum of another nature, especially of a cerebellar tumour.

DEGENERATION OF THE MOTOR CONDUCTION TRACT

PRIMARY AND SECONDARY DEGENERATIONS OF THE LATERAL COLUMNS

Secondary Antero-lateral Column Degeneration.—As in the posterior columns, so also in the antero-lateral columns, columniform degenerations may occur. In many cases these are of a ***secondary nature.*** As previously explained (p. 518)

the motor chief innervation tract, i. e., the pyramidal tract, consists of two individual nerve units (cell, fibre and terminal arborization), of which one reaches from the cerebral cortex cells to the anterior horn ganglion cells, surrounding the terminal arborization of the motor column fibres (central motor neuron), the other from the anterior horn ganglion cell up to the terminal arborization in the muscle (peripheral motor neuron). Further, we have seen that the conserving influence of the motor cranial nerve cells reaches only to the terminal arborization in the spinal cord, whereas that of the anterior horn ganglion cells affects the peripheral nerve fibres to the terminal arborizations and, with this, the muscle. Any interruption of the motor tract, therefore, will produce degeneration of the peripheral parts of the fibres, and up to the termination of the interrupted nerve continuity. According to this, it is plain that *descending* degeneration in the pyramidal lateral column tract (and for a small part of the antero-lateral pyramidal column tracts) is frequently found as a secondary condition in *diseases of the brain, the cerebral peduncles, the pons, the medulla oblongata, and also of the spinal cord,* in the latter case, as soon as the affection of the spinal cord concerns the greater portion of a transverse section, thus producing an interruption of conduction from above, such as occurs in *compression of the spinal cord,* in *transverse myelitis* and in *tumours,* further also in *mechanical injuries* and the (*rare*) *hæmorrhages of the spinal cord.* In this case, degeneration occurs from the point of lesion downward, according to the seat of the distribution of the latter, unilaterally or bilaterally, in the pyramidal lateral column tract, and in so far as a pyramidal anterior column tract is present at all below the point of lesion (which usually can no longer be determined even in the lower thoracic cord), this also degenerates.

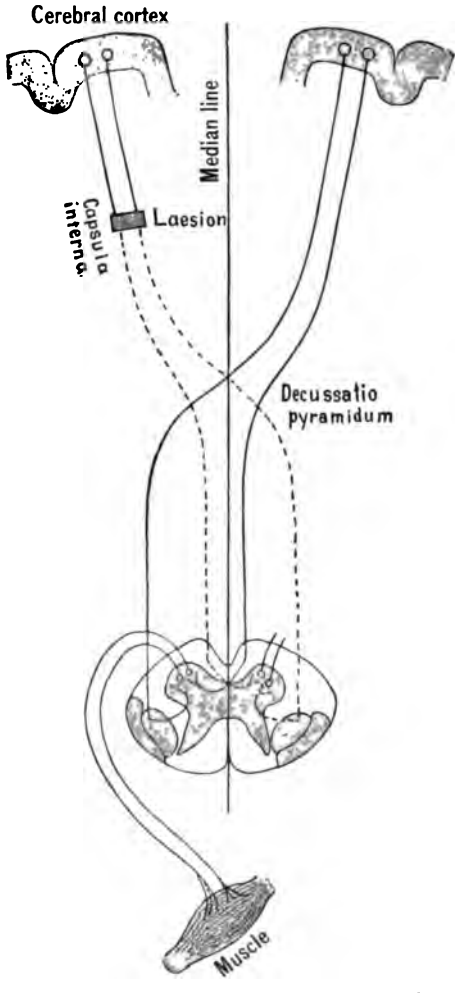


FIG. 35.—DIAGRAM OF THE DEVELOPMENT OF SECONDARY DEGENERATION IN THE PYRAMIDAL TRACT IN A LESION OF THE LEFT INTERNAL CAPSULE. .....DEGENERATED FIBRE TRACT.

Much discussion has arisen regarding the *clinical importance* of these secondary degenerations. Without further dispute it is clear that they can neither cause nor increase *paralysis,* as the paralysis is due to an *interruption of the tract of conduction.* This latter condition is also the cause of the *increase of the tendon reflexes* in such cases, in that the impulse of the will coming from the cerebrum, can no longer exert its preponderating influence upon the motor ganglion cells (see p. 523). *Secondary degenerations are therefore, in a clinical respect, conditions without importance.*

If, however, the pyramidal lateral columns are affected in the course of a spinal-cord affection, by contiguity, or are exclusively *primarily* affected by the degeneration, a clinical picture appears which has been designated spastic spinal paralysis.

SPASTIC SPINAL PARALYSIS

Spastic Spinal Paralysis—Primary Lateral Column Degeneration.—By this we understand a symptom-complex occurring in paralysis or paresis, with marked increase in the tendon reflexes and the occurrence of reflex spasms due to this cause. The latter changes arise in all active and passive movements, and in turning the extremities, give the *gait of the patient* an especially conspicuous stamp: The patients raise the spastically paretic, stiff legs with much difficulty in the knee joint, the feet remaining upon the floor, they are slowly shoved forward, and on account of the spastic reflex contraction of the muscles of the calf of the leg, the tip of the foot is brought down first; walking occurs by small steps, and gives rise to the appearance as if the patient were walking upon stilts ("*spastic-paretic gait*"). Occasionally paresis is but slightly developed compared with the increased tendon reflexes and spasms, so that the gait in these cases becomes *purely spastic*. Every attempt to bend the stiffly extended legs fails on account of the reflex muscular spasms which follow.

This picture of spastic spinal paralysis is seen as a part phenomenon in some diseases of the medulla not localized to distinct areas of the spinal cord. In compression of the spinal cord, in myelitis, in multiple sclerosis, etc., spastic spinal paralysis is usually prominent, if the process, in keeping with its distribution and with the stage of development in which it happens to be, especially affects the pyramidal tracts.

In other cases, the pyramidal lateral columns are *primarily* affected by unknown causes (similar to the posterior columns in the case of tabes) and appear to be degenerated in a greater area of distribution through the entire length of the spinal cord. In nearly all instances of this character, besides degeneration of the pyramidal lateral column tracts, other portions of the spinal cord were also found affected at the autopsies, such as the cerebellar lateral column tracts and Goll's columns. In such cases, during the life of the affected patients, besides the symptoms of spastic spinal paralysis, there occur also disturbances of the bladder, ataxia and mild sensory disturbances. These cases are summarized under the name of "*combined system diseases.*" If we judge impartially a great number of autopsy findings of diseases of the spinal cord, the impression is certain to arise that the diagnosis of these "combined system diseases" in a certain number of cases is of very slight value. In an organ in which, as in the spinal cord, in the smallest possible space, there are crowded together nerve-fibre tracts producing many different functions, localizing diseases affecting a single column tract are possible, and are represented by such types as tabes, poliomyelitis, etc.: But it is also easily understood that degeneration of other columns may also occur without this being at all unusual and that a new disease need not be assumed on this account. I should like to advise, instead of setting up new special disease types, to designate the disease *a poteriori*, and to the name of the principal affection if necessary there may be added the name of the secondary condition, as for example: Degeneration of the pyramidal tract with simultaneous affection of the posterior columns, etc.

The future will show which special types the combined system affections properly give rise to, and whether they are separate diseases or not. Lately the beginning has been made to separate such affections from the general symptom-picture of spastic spinal paralysis.

1. *Hereditary, respectively, Family Spastic Spinal Paralysis.*—This affection occurs in certain families (as it appears especially if the parents are closely related to one another) in several members of the same family during the time after puberty (usually during the third decade of life) and is characterized by the previously described symptoms of spastic spinal paralysis, by an increase in the tendon reflexes, an increase of the rigidity of the lower extremity, and the spastic paretic

gait, without muscular atrophy. Sensory disturbances are absent, and the bladder function is intact. The affection is probably due to a pathological slow development, respectively a feebleness of the pyramidal tracts.

2. *Spastic Spinal Paralysis not Hereditary.*—This occurs partly in the *newborn*, especially after severe, premature birth; probably a poor development of the still incompletely formed pyramidal tract or aplasia of the motor centres play a part—(*Little's disease*), it sometimes occurs in persons having passed the mid period of life. The affection then develops, usually, in connection with a chronic intoxication (with lathyrus, a plant of the species of leguminoses, which occasionally is mixed with flour, further by flour from diseased maize—pellagra—or after infectious diseases, acute articular rheumatism, diphtheria, influenza, etc.). The most important cause of spastic spinal paralysis, however, as is also the case in tabes dorsalis, is *syphilis*. Since Erb, in 1882, has given this opinion upon the basis of clinical investigation, a great number of observations confirming this view have been made, so that lately it has become usual to describe a special form of spinal paralysis as:

3. *Syphilitic Spastic Spinal Paralysis.*—The kernel of the morbid picture is the well-known symptom-complex of spastic spinal paralysis; only there appears to be present an affection of the bladder, retention of urine and later incontinence, these being almost constant symptoms; also impotence frequently occurs. In spite of marked increase of the tendon reflexes but very slight muscular tension can be demonstrated; also only slight disturbances of sensation are noted. An increase of the thermic reflexes has been noted by Kowalewsky among the symptoms of syphilitic spastic spinal paralysis, this is said to be an especially prominent symptom. The development of the disease is very gradual, the course, a chronic progressive one; but just in this form of spastic spinal paralysis, improvement, even cure, may occur. Anatomically the affection is probably due to a myelitis of the dorsal cord, especially originating in the small vessels of the posterior part of the lateral column, the posterior horns and posterior columns.

As is obvious, the separation of the previously mentioned varieties depends principally upon the ætiology; as an anatomical starting-point a *primary affection of the central motor neuron in the pyramidal lateral columns* may be assumed, to which, according to the individual case, a further degeneration in the posterior horns, cerebellar lateral column tracts, and posterior columns is added.

In the affections which now follow, there is an exclusive *degenerative affection of the motor neurons*, be it of *both neurons simultaneously* (*amyotrophic lateral sclerosis*), be it of the *peripheral neuron* (including the muscles) in its various parts: The anterior horn cells (*spinal muscular atrophy*) or of the peripheral nerves (*neurotic muscular atrophy*), or finally of the end apparatus of the peripheral motor neuron, the muscles (*dystrophia muscularis*). This diagrammatic, anatomically founded separation of individual disease forms, belonging together, cannot be strictly carried out in all cases; but it will be well, at least in general, to adhere to the mentioned principal varieties.

An exception to this is *amyotrophic lateral sclerosis* which is now to be described, in that in this disease, with a regularity almost conforming to a law, the affection is limited to the pyramidal tracts and anterior horns (i. e., therefore, to parts of both nerve units which collectively form the chief motor innervation tract) and which remains almost exclusively limited to the pyramidal tract, even if it does not affect the spinal cord alone but also the posterior, middle, and anterior brain in its course. In keeping with this, amyotrophic lateral sclerosis presents a sharply defined, typical morbid picture.

AMYOTROPHIC LATERAL SCLEROSIS

We shall most clearly and quickly understand the symptoms of this disease, which are of especial importance, if we first study the autopsy findings which occur in amyotrophic lateral sclerosis.

Morbid Anatomy.—The pyramidal tracts of the spinal cord are in a condition of degeneration, primarily both *pyramidal lateral column tracts*, less constantly the pyramidal anterior column tracts (which in individual cases are not implicated at all, or to a very much slighter extent than the lateral column tracts), simultaneously with atrophy of the *motor ganglion cells of the gray anterior columns*. The ganglion cells are occasionally but very slightly affected, relatively this is noted most markedly as it appears in the outer portions of the anterior columns. Rarely does the process remain limited to the spinal cord; but rather very usually also above the cord in the *medulla oblongata* and in the *pons* degeneration is noted along the course of the pyramidal tract and atrophy of (analogous to the anterior horn ganglion) the *motor nucleus* of the cranial nerves (hypoglossus, facial nerve, etc.) upon the floor of the fourth ventricle. Not always does the process find its limitation here, in some cases it follows exactly the direction of the cerebral pyramidal tract, through the cerebral peduncles, the internal capsule, yes, even into the central convolutions or the paracentral lobes, i. e., the *entire course of the chief motor innervation tract is affected—from the ganglion cells of the cerebral cortex to the anterior columns and beyond these to the periphery*; for the peripheral nerves, the direct continuation of the anterior horn ganglion cells, as may be expected, also take part in the degenerative process, and their fibre disappearance has also been determined with certainty. Above all, however, the muscles show the character of *degenerative atrophy* with preponderance of the fat and connective tissue above the more or less marked disappearance of the muscular substance.

Besides the fibres and cells of the chief motor innervation tract, the commissure cells and commissure fibres also degenerate, further in some cases, also fibres of the boundary stratum and of Goll's columns. But these may be looked upon more as secondary findings; the brunt of the affection consists in the degeneration of the cortico-muscular conduction tract, i. e., *both* motor neurons in the various portions of their course are injured.

Diagnostic Symptoms.—The clinical symptoms of the disease correspond very closely with these pathological changes, so that the diagnosis as a rule, does not give rise to difficulties, as the explanation of the phenomena present can be given in a satisfactory manner. Whereas *sensation* in all of its phases remains normal, the *motor sphere is severely damaged*. Primarily, there is difficulty in movement, pareses and spasm in the extremities, *usually at first in the upper, and only after half a year, and later, also in the lower extremity*, giving rise to the spastic paretic gait. *The tendon reflexes are greatly increased*. To these symptoms, the clinical expression of the alteration in the pyramidal columns, there is now added, especially in the upper extremity (in the lower extremity at first not at all, later only to an insignificant degree), a very marked muscular atrophy, beginning in the muscles of the hand, the ball of the thumb and of the little finger, and farther on, affecting the interossei, the extensors of the forearm, the deltoid and triceps of the upper arm, *en masse and comparatively rapidly*. The atrophy affecting simultaneously the larger muscle masses is the result of the anatomical changes in the motor ganglion cells of the anterior horns. That the latter are not affected alone, is proven by the pareses and the increase of the tendon reflexes preceding these conditions, and as shall already be remarked here, is absent without exception in progressive muscular atrophy (in which the pyramidal tracts are not affected under any condition).

It is undoubtedly conspicuous, at first sight, that on the other hand, in amyotrophic lateral sclerosis, this *increase in the tendon reflexes*, in spite of the atrophy

differential diagnosis to the next chapter, I shall mention in advance that the presence of an increase of the tendon reflexes and of spastic phenomena, besides the paresis, form the important principal points between both diseases, in that the increase of the tendon reflexes and spasms are always absent in progressive muscular atrophy.

ANTERIOR POLIOMYELITIS

In a number of diseases, an affection of ganglion cells of the anterior horns is the anatomico-pathological cause which determines the clinical picture. Whereas in amyotrophic cerebro-spinal (lateral) sclerosis just described the chief motor-innervation tract is completely affected or at least from the posterior brain downward to the peripheral distribution of the nerves to the muscles, being found in a condition of degeneration, the *affections which are to be discussed now are principally limited to the peripheral motor neuron,* i. e., to the ganglion cells of the anterior horns and to the nerve fibres going off from them. Degeneration of the peripheral nerves, degenerative atrophy of the muscles, diminution or complete absence of the tendon reflexes, are the obvious results of the diseased peripheral motor-fibre tracts. These consequences arise whether the point of attack is in the anterior horn or more peripherally situated in the nerve tract, and in keeping with this, also certain cases of peripheral neuritis affecting exclusively the motor-nerve fibres belong to this category of diseases (neural muscular atrophy).

By their course and by the clinical picture which they present, *acute* and *chronic anterior poliomyelitis* markedly differ from one another. The *acute* form occurs particularly in children in the first years of life, rarely in adults; the *chronic* form on the contrary affects almost exclusively adults, and shows certain peculiarities in its development and course, so that we have become accustomed to describe one of these varieties as a special disease and to designate it *spinal progressive muscular atrophy.* Even if this diagnostic procedure is well founded, *practical points* are opposed to this, all the more as by the term chronic anterior poliomyelitis in the wide sense by no means all progressive muscular atrophies can be subsumed. But, instead, certain cases of progressive muscular atrophy must be considered as *neuritides* (*neural muscular atrophy*) affecting especially and exclusively the motor nerves, and again other cases must be referred to degenerative affection of the terminal apparatus of the motor neurons, of the muscles (*dystrophia muscularis*). In keeping with this, when considering the diagnosis of chronic anterior poliomyelitis we shall devote a special chapter to the consideration of progressive muscular atrophy.

ACUTE ANTERIOR POLIOMYELITIS—POLIOMYELITIS ANTERIOR ACUTA INFANTUM, SPINAL (ESSENTIAL) INFANTILE PARALYSIS

The clinical picture is very characteristic, easy to diagnosticate, the explanation of the phenomena upon the basis of the anatomical changes in the spinal cord is a very satisfactory one. The disease usually begins sud-

denly, most frequently with high fever (105° F. and over), severe affection of the entire nervous system and of the general health (with headache, pain in the extremities, somnolence, convulsions, in mild cases with anorexia, lassitude, restless sleep). After cessation of these *initial symptoms*, which may last from hours to weeks, usually only a few days and which sometimes may be entirely absent, the persistent chief symptom occurs, the *paralysis*.

The *intensity and distribution of the paralysis* in the individual case varies greatly. At first being greatly distributed, the paralysis confines itself to both legs or to the arm and leg, as well as to one half of the trunk (hemiplegia spinalis), the arm of one side and the leg of the other side, or even to but one leg, or to one arm (monoplegia spinalis), or finally affecting but certain muscle groups of the extremity. The localization and limitation of the paralysis depends principally upon the extent of the morbid process, and upon the amount of ganglion-cell groups which are permanently and completely damaged by the latter, whereas the temporary paralyses may be looked upon as a *transitory* damage of the ganglion cells by the infectious cause of the disease.

Character of the Paralysis.—The *paralyses are constantly characterized by special phenomena which must be closely observed in the diagnosis.* The paralyses are exclusively *flaccid* in character, never accompanied by spastic phenomena in the paralyzed muscles, and after a few weeks show a rapidly increasing *atrophy* (rarely being masked by a marked fat deposit). The paralyzed limbs cannot be actively moved and so long as antagonistic contractures do not occur, may be recognised as *flaccid* dead masses, which upon being raised fall passively without resistance and also, especially on account of the flaccid condition of the joints, may be placed in any position. The *reflexes* are *obliterated*, the cutaneous reflexes as well as the tendon reflexes. The *electrical contractility* of the paralyzed secondarily atrophically degenerated nerves and muscles (from the middle of the second week on, after the paralysis) is, as in peripheral motor paralysis, altered, i. e., DeR may be noted in the paralyzed atrophic muscles at any time. The *skin* is cool, marbled, occasionally scaly, œdematous, and covered with cold perspiration. The *sphincter functions* appear to be *intact, sensation* is *not interfered with*—all phenomena the cause of which in the disease depends principally upon an affection of the anterior columns and is limited there, and requires no further explanation after what has been previously stated.

In the course of time, it is seen that the atrophied extremity, in all of its parts (bones, fasciæ, vessels, etc.), except the cutaneous fat which is disproportionately greatly developed, is more or less retarded in its growth. The difference in length, between the normal and atrophied extremity, is then usually several centimetres (up to 20).

Paralysis of Muscles functionally Belonging together.—In an exact determination of the distribution of the paralysis of the extremity, it is found that distinct muscle groups are affected by the paralysis with a certain degree of regularity, in the forearm usually the extensors of the hand, with the exception of the supinator longus which is also supplied by the radial nerve, are paralyzed and atrophied, whereas in other cases, the last-named muscle simultaneously with the biceps and brachialis internus appears paralyzed; in the upper arm the deltoid is especially affected by the paralysis. If the lower extremity is the seat of the paralysis, there are

to be noted, besides well-distributed paralysis, the intact condition of certain muscles. For example in the atrophic paralysis, in the course of the crural nerve frequently the sartorius, in the course of the perineus nerve the tibialis anticus, is spared. Since we know that in a nerve root those nerve fibres leave together which are intended for synergistically acting muscles, and that the completeness of these fibres is in keeping with common ganglion-cell groups in the anterior horns, in certain segments of the spinal cord (compare p. 519), this fact is not to be wondered at, i. e., it is easily understood that the appearance of a paralysis of a certain motor nerve, apparently sparing certain muscles supplied by the same nerve, may occur in the picture of paralysis.

Secondary Contractures.—By the action of antagonistic muscles that are not paralyzed, by their power and other subordinate external conditions, there are not rarely formed, especially from the second month on, contractures which, upon superficial examination, may give rise to difficulties in the diagnosis; for instance in the lower extremity there develop *clump-foot, flat-foot,* and *equinus positions,* the appearance of the *genu recurvatum,* etc.; further, by partial paralysis of the muscles of the back, curvature of the vertebral column may occur: kyphoses and scolioses, also loose joints—especially in the shoulder, by paralysis of the deltoid. In special cases, the inflammatory process, usually localized to the anterior horns, may also distribute itself to other parts of the spinal cord, especially to the posterior horns, to the lateral and posterior columns.

Differential Diagnosis.—The *differential diagnosis* occasionally, but very rarely, gives rise to difficulties. The *acute* course of the disease, with its rapidly developing and afterwards *stationary* paralyses, excludes affections of the cord which may arise with a similar general result of disturbances (paralyses, muscular atrophy, etc.), but which are from the onset chronic and progressive. However, a confusion of spinal infantile paralysis is possible in cases in which *acute diffuse myelitis* develops in early childhood, the differential diagnosis of which will be discussed later on; only this much shall be mentioned now, that in these cases disturbances of the functions of the bladder and rectum occur, sensory alterations and bedsores are present, apart from the fact that reactions of degeneration and muscular atrophy are almost exclusively absent. Upon close observation, therefore, confusion of both affections cannot occur; but the differentiation of spinal infantile paralysis from certain *peripheral paralyses* and individual varieties of *polyneuritis* may give rise to great difficulty.

Obstetric Palsy.—In this connection we must first regard the so-called *"parturition paralysis"* (compare p. 491) in which, similar to certain varieties of anterior poliomyelitis, synergistic co-ordinating muscles of the arm appear simultaneously paralyzed (deltoid, biceps, brachialis internus and supinator longus). There is present in this peripheral paralysis an important symptom of poliomyelitic paralysis and it is obvious, as the symptoms of both affections are naturally the same (muscular atrophy, DeR, etc.), that in cases of poliomyelitis anterior, in which accidentally the degeneration is limited to this muscle group, the differential diagnosis as such cannot be made from the clinical picture. But upon considering the ætiology it is easily accomplished, in that the paralysis is due to trauma, especially during the act of birth, therefore the paralysis in the affected

child is noted at the time it is brought into the world. The diagnosis becomes more certain if, besides the motor paralysis, simultaneously the cutaneous sensation is also influenced, which, however, is rarely the case.

Neuritis.—The same difficulties arise in the differential diagnosis regarding neuritis occurring in children. *If in the initial stage of the disease the pain is absent or at least the pain in the extremity which is later paralyzed is not present, this is a diagnostic sign of importance for the presence of poliomyelitis.* Now there are certainly also cases of neuritis which are exclusively limited to the motor fibres of the peripheral nerves so that the previously mentioned diagnostic symptom is not always active. This is also the case with another symptom, which at first sight appears to be of importance in the differential diagnosis: The *exclusive paralysis of synergistically co-ordinating muscles,* which might be regarded, as we may assume, as belonging to an affection of the ganglion cell-groups of the gray substance of the anterior columns. But the hope that this would be a certain differential point between poliomyelitis and neuritis has not been fulfilled, in that also in rare cases of polyneuritis (in which the motor ganglion cells are not affected) muscles functionally belonging together are paralyzed and become atrophic. A certain differentiation therefore between both diseases under some circumstances is impossible, all the more as the same cause which gives rise to spinal infantile paralysis, an acute *infection,* usually also affects the spinal cord, occasionally even the cerebral cortex, the medulla oblongata or the peripheral nerves and in some cases also the latter, simultaneously with the spinal cord. In individual cases, in favour of the diagnosis of a polyneuritis (opposed to a poliomyelitis) on the one hand, are the relatively rapid disappearance of the paralyses, on the other hand, the appearance of ataxia, of disturbance of the bladder and rectal functions and paralyses of the cranial nerves (compare p. 504, etc.), and in such cases the presence of the marked symptoms of polyneuritis (painfulness upon pressure of the nerve trunks and cutaneous sensory disturbances) is unnecessary to arrive at a diagnosis.

Finally, in monoplegic or hemiplegic infantile paralysis the question may arise whether the source of the disturbance should be looked for in the *brain* or in the spinal cord. The decision of this question is easy, according to what has been dwelt upon previously (p. 430)—the observation of the flaccid character of the paralysis, the marked muscular atrophy, the presence of DeR, and the diminution of the tendon reflexes in the paralyzed members, allow us to exclude a cerebral affection in the individual case.

POLIOMYELITIS ANTERIOR ACUTA ADULTORUM, ACUTE AMYOTROPHIC SPINAL PARALYSIS OF ADULTS

Acute anterior poliomyelitis, according to the investigations which have been lately made, also occurs in adults (especially up to the thirtieth year of life), however it is very much rarer than in children. The symptoms produced by it differ from those described in infantile paralysis in no essential diagnostic criteria. In this affection fever and general phenomena also usher in the disease, which on an average appear to last longer than in infantile paralysis (about one week); the usual convulsions so common in the initial stage are absent in anterior poliomyelitis of adults. The paralyses which occur show all the characters of the motor form described in

infantile paralysis—the limitations to a single extremity, to synergistically co-ordinating muscle groups, the signs of DeR, the absence of the reflexes, the muscular atrophy, etc.; only the deficient growth in length of the paralyzed limbs, is of course not present. The functions of the bladder and rectum are normal as well as that of the sexual apparatus; sensation is not interfered with—the diagnosis is therefore identical with that of infantile paralysis. A number of the cases described as acute amyotrophic spinal paralysis of adults undoubtedly belong to neuritis which then unquestionably may be referred to the same ætiological cause, only showing a different localization of the virus in the peripheral motor neuron, or which may develop simultaneously with acute poliomyelitis (just as in the case of children). The diagnosis of neuritis may be made in such cases by regarding the points described in the diagnosis of infantile poliomyelitis, especially the differentio-diagnostic criteria regarding sensory disturbances, the onset with pains in the back and in extremities.

SUBACUTE AND CHRONIC ANTERIOR POLIOMYELITIS

The chronic variety of this affection occurs in two different forms: 1. As subacute, respectively chronic, anterior poliomyelitis, and 2, as chronic progressive anterior poliomyelitis (progressive spinal muscular atrophy).

The former affection, *chronic anterior poliomyelitis (adultorum) in the restricted sense*, differs from the previously described varieties of acute poliomyelitis in that its course is more protracted and that certain irritants show a chronic action from the onset (*syphilis and lead intoxication*) and, besides, the conditions which give rise to acute anterior poliomyelitis (*cold*, overexertion, *trauma*—i. e., effects which markedly shatter the nervous system in accidents, and specific *infections*, the exact nature of which has not yet been determined) must be regarded in the genesis of the affection. In keeping with its gradual development, the affection begins insidiously without prodromes, or after a prolonged initial stage in which prodromes are very indefinite and by no means marked, the disease sets in with flaccid paralyses which gradually increase in intensity and extent, and which usually are first noted in the lower extremity, later also occurring in the upper extremity, occasionally even showing an inverted course of development, and but rarely limited to a single extremity or individual muscles. In this affection, as in the acute variety, the peculiarity is noted, showing the nature of ganglion-cell disease, that muscles functionally belonging together are affected simultaneously, or are rapidly affected in succession: for example, the supinator longus becomes affected with the biceps, occasionally even before the muscles of the forearm are attacked, the abductor pollicis longus, supplied by the radial nerve, with the muscles of the ball of the thumb, after the extensors of the hand and fingers have become implicated. In the course of the paralyses, periods occur in which the disease appears to be stationary and improvement sets in; but in all cases the paralysis is accompanied with atrophy of the muscles, reduction of reflexes and with the sign of milder or severer DeR. To this there may be added fibrillary contractions and secondary contractures, the muscles of respiration may be affected, this causing the disease to become directly dangerous to life; or the process may reach the medulla oblongata producing bulbar phenomena (as also occurs in acute anterior poliomyelitis in rare cases). The *sensory sphere, the functions of the bladder and rectum in the pure form of the affection are completely intact*, bedsores do not occur—briefly, the complete picture of chronic anterior poliomyelitis, harmonizes in all points with that of the acute form.

Method of Diagnosis.—In the differential diagnosis it should first be determined that *principally the motor innervation tract is affected*—i. e., the absence of all sensory disturbances as well as implication of the functions of the bladder and rectum must be ascertained. This eliminates a great number of chronic affections of the spinal cord (tabes, poliomyelitis posterior, syringomyelia, diffuse myelitis, polyneuritis with affections of the sensory and motor nerve fibres). Now, the investigation must be directed to determine whether *both nerve units of the motor innervation tract are diseased, or only whether the peripheral one is affected.* This is easily decided by the observation of the spastic character of amyotrophic paralysis. If

this is not entirely absent but if the reflexes are increased, there can be no question of an exclusive affection of the peripheral neuron; it must then be assumed that disease of both neurons of the motor innervation tract is present, especially *amyotrophic lateral sclerosis* has occurred. If the opposite is possible and an affection of the central neuron may be excluded, only anterior chronic poliomyelitis or polyneuritis with an especial elective affection of the peripheral motor-nerve fibres is to be considered.

Polyneuritis.—As has already been mentioned and explained in the diagnosis of neuritis, an absolute differentiation of both conditions, at least in a part of the cases, is no longer possible and this lies in the nature of the case. Naturally in favour of chronic anterior poliomyelitis is the onset and course of the disease without any localized sensory disturbance in the parts of the body which are later paralyzed, the strictly typical atrophy of the muscles occurring in groups functionally acting together. However, as has already been mentioned, cases of polyneuritis occur in which these symptoms which are specially in favour of poliomyelitis exceptionally also occur. In these cases the simultaneous occurrence of symptoms which are occasionally noted in polyneuritis, such as disturbances of the bladder function or well-developed ataxia, may point in favour of the latter disease, however even these symptoms are of questionable diagnostic value, and the great effort which has lately been made to develop differential points between both affections, artificially in my opinion, is entirely unnecessary, as in these questionable cases a marked difference in the nature of both disease processes does not exist.

Spinal Progressive Muscular Atrophy.—The same is also true of the differentiation of anterior chronic poliomyelitis, respectively the subacute form, from *spinal progressive muscular atrophy*, the diagnosis of which for practical purposes only will be described in a special chapter. The course of development of the latter disease is *particularly chronic and progressive*, in which fibre after fibre atrophies; this allowing us clinically to differentiate this form of chronic anterior poliomyelitis from the other. In the latter, even if the disease develops gradually, its course in all cases is relatively quicker, and after the affection has reached a certain stage, unless the symptoms improve, it becomes more stationary. The function of the motor ganglion cells also ceases at once to a marked extent, and this damage of function is shown in a more or less well-distributed paralysis which is *followed* by atrophy of the muscles more *en masse*, whereas the atrophy of the muscles in progressive spinal muscular atrophy occurs periodically and dominates the scene so completely that the paresis appears to be a secondary consideration. Also in progressive spinal muscular atrophy, the DeR and the absence of the reflexes is developed to a slighter degree. In general the symptoms of both diseases, in keeping with their identical localization in the anterior columns, must be the same, and they are, apart from a few phases of development, as the following description will show.

PROGRESSIVE SPINAL MUSCULAR ATROPHY: POLIOMYELITIS S. POLIOATROPHIA ANTERIOR CHRONICA PROGRESSIVA

This disease depends upon a *progressive atrophy of the voluntary muscles due to an affection of the principal motor-innervation tract centrally, not going beyond the peripheral motor neuron (from the ganglion cells of the anterior horns to the termination of the motor-nerve fibres in the muscles)*.

Pathology.—The anatomical lesions are: Diminution of the anterior column, sclerotic atrophy of the motor ganglion cells, degeneration of the anterior roots and peripheral nerves, atrophy and eventually fatty degeneration of the muscles in connection with an increase of the interstitial connective tissue, intact condition of the pyramidal tracts, occasionally extension of the affection of the anterior columns of the spinal cord to the motor cerebral nerve nucleus in the posterior brain. Nothing can be noted of inflammatory phenomena in this process, so that the affection is

better designated by the name of *polioatrophy* than by the name of poliomyelitis; but in chronic poliomyelitis which we have just described, the inflammatory (*sensu strictiori*) character of the disease is by no means beyond all question of doubt. If the changes are compared which occur in the peripheral nerves and muscles in progressive muscular atrophy with those in the ganglion cells, in various cases undoubtedly the atrophy of the musculature is not in keeping with the process—i. e., a conspicuously slight disappearance of the ganglion cells of the anterior horns is noted. However, to conclude therefrom that the process is opposed to our previous experiences, that it is an *ascending* one from muscles to the spinal cord, *is unnecessary*, and on that account, as I believe, is not permissible at present.

The Course of Atrophy in the Muscles.—The appearance of the disease is always typical, the diagnosis usually easy. The onset is characterized by a *diminution* in the size of certain muscles and *muscular weakness* going hand in hand with this process, the atrophy appearing first in the small muscles of the hand, those constituting the *palms of the thumb and little finger*, so that their roundness completely disappears. The muscles which particularly show the affection in the thumb are the adductor, opponens, and the abductor pollicis brevis; the extensor pollicis longus is not affected and shows its full activity in opposition to the weak, atrophied muscles of the ball of the thumb, so that both joints of the thumb appear stretched. If there is a well-developed steady approach of the thumb to the second metacarpal bone, a position of the thumb results which reminds us of the "ape-hand." To the atrophy of the muscles of the palms of the thumb and little finger, very soon an *atrophy of the interossei and lumbricales is added*; occasionally this may precede the former changes. This gives rise to deep furrows between the metacarpal bones and a flattening in the palm of the hand, and the development of the "claw-hand" occurs, which has been described under paralysis of the ulnar nerve (compare Fig. 23). Quite important (because an insight into the distribution of the process in the spinal cord is given by it) is the fact that following disease of the muscles of one hand (usually at first of the right) next, as a rule, the same muscles of the other hand are affected, and now only the muscles farther towards the trunk are attacked by the atrophy—preferably first the *deltoid*, then the muscles of the forearm (first the extensors), later those of the upper arm, in which the triceps is spared longest by the atrophy. Now atrophy of the muscles of the trunk occurs, first in the trapezius then in the pectoral muscles, the latissimus dorsi, etc. Late, if at all, the muscles of the lower extremity are affected, at first the flexors of the lower leg and the quadriceps. In the same manner, only in well-advanced stages of the disease, are the respiratory muscles attacked, especially also the diaphragm; then marked disturbances of respiration occur, which may bring on a speedy fatal termination.

From this description, the usual course of the atrophy, many *exceptions* occur. Thus in some cases the wasting of the muscles does not begin in the small muscles of the hand, but in the deltoid and thence affect the interossei; in other cases first the biceps, brachialis internus and supinator longus are affected, after the deltoid. Indications of typical further development of the atrophy in muscle groups belonging together are also found in progressive muscular atrophy.

The disturbances of function due to the atrophy of the muscles, abnormal posi-

tion of the limbs, and disorders of movement are manifold and vary greatly, according to the extent and intensity of the process; on this account, in the diagnosis of the atrophy of the individual muscles, we cannot describe the individual effects produced. In rare cases, besides the atrophy of the muscular fibres, a plentiful deposit of fat occurs between them, so that the diminution in volume of the muscles is covered by the fat, the contours are not markedly changed and the degeneration must be judged by the disturbance of function, and the electric contractility and so on. It must be emphasized that there are never great muscular masses affected *at one time*, but muscle after muscle diminishes in size, and in individual muscles the atrophy occurs from bundle to bundle and increases, as may be seen particularly plainly in the deltoid.

The *tendon reflexes are not increased* (in contrast to the condition in amyotrophic lateral sclerosis), but *pari passu* are *reduced or disappear* with the atrophy, in keeping with the disturbance of conduction in the ganglionic or motor portion of the reflex arc. The patella tendon reflexes are retained for a long time, i. e., so long as the lower extremities are not affected by the atrophy. The *cutaneous reflexes* are diminished, in so far as in their development the full contraction of the atrophied muscles is concerned.

Electrical Conditions.—The *electric contractility* of the diseased flaccid muscles varies greatly, according as to whether retained or atrophic muscular fibres are irritated. The latter usually show at first partial, later complete *DeR*; the stimulation of the nerve is preserved for a long time, sometimes even increased, to disappear in the further course of the affection. To determinate the theoretically postulated DeR, the smallest electrodes must be used, so as to touch isolately the individual degenerated muscular bundles; without this precautionary measure the sign of the altered electrical reaction would be missed.

Fibrillary Contractions.—An almost constant symptom of muscular atrophy are the isolated contractions of individual muscle bundles in the course of the atrophied bundles, designated by the name of *"fibrillary contractions."* They apparently occur spontaneously, but become quite plain by blowing upon the skin and by mechanical or electrical irritation of the muscles.

Their *causation* is not quite clear; they are not only found in patients with progressive muscular atrophy, but also in other nervous diseases and even in health, and may be present for many years without showing any further consequences. In my opinion they are of *reflex* origin. It is true that the property of conduction of the reflex as in progressive muscular atrophy has suffered, as is proved by the reduction of the tendon and usual skin reflexes, and by the microscopical examination of the peripheral nerve fibres. However, the latter has only shown a partial degeneration of the anterior roots and of the peripheral nerve fibres, and as the nerve in the stage of the declining energy shows also increased irritability and, besides, as a rule, the bulbo-cervical region is not affected in the disease, it is obvious that sensory irritations may easily give rise to reflex contractions, which are, however, not accompanied by a well-distributed complete effect, as the individual nerve and muscle fibres are for the greater part degenerated. The correctness of this assumption of fibrillary contractions as a reflex phenomenon appears to be favoured by an observation which I some time ago had the opportunity of making. It occurred in a case of myelitis with special affection of the lateral columns; spastic paresis and slight anæsthesia were the principal phenomena. *On blowing upon the skin of the thigh or with slight tapping against the patella tendon, marked fibrillary contractions*

occurred, which gradually increased to a decided tremor terminating in clonic spasm as soon as the tendon was struck with more force. In normal individuals a weak sensory irritation, such as blowing upon the skin, or the cooling of the skin produced by undressing, is not sufficient to develop even these weak reflex contractions; as soon however as the weak irritability of the reflex arc is increased up to a certain point, as occurs in nervous individuals and which may be assumed to appear in various nervous diseases, these weak sensory irritations are sufficient to produce fibrillary contractions.

Besides the symptoms already described, occasionally *vaso-motor disturbances* occur: Coldness and marbling of the skin, desquamation, and a fissured condition of the skin and nails, etc. This vaso-motor disturbance may be understood according to the latest investigations regarding certain sympathetic fibres originating in the anterior horn cells (see p. 520).

Sensory Conditions, etc.—It is of great importance to note *that the sensory conditions as well as the functions of the bladder and rectum remain entirely intact.* From the previously described course of the atrophy, from muscle to muscle, the conclusion may probably be drawn that the process affects different transverse sections of the spinal cord of one side, going over to the other and *descending* from the cervical cord.

Bulbar Symptoms.—A propagation of the disease however also occurs *upward,* in that *bulbar symptoms appear*: Anarthria, atrophy of the tongue, difficulty in deglutition, etc., the result of degeneration of the cerebral-nerve nucleus which, however, appears but late in the disease. The opposite, however, is also undoubtedly known to occur, that the signs of progressive bulbar paralysis open the scene and progressive muscular atrophy of the muscles of the trunk and extremities follows.

Differential Diagnosis.—As a rule, it is easy to prevent confusion of typically developed muscular atrophy with other diseases. Primarily there must be noted whether the muscular atrophy is *symptomatic,* due to a cessation of conduction in the motor nerves, in that trauma, etc., have caused the interruption of continuity of the latter, or whether in disseminated lesions of the spinal cord, among others, also the motor-ganglion cells are affected. In both cases, paralysis precedes the atrophy of the affected muscles, and the motor paralysis is combined, provided we are not dealing with a purely motor nerve, with severe sensory disturbances. The latter is also true of those cases of secondary atrophy the result of *neuritis,* those rare cases excepted in which progressive muscular atrophy itself probably owes its production to a multiple affection of the motor fibres in the neuritic process. (See p. 567, *neural muscular atrophy.*) Above all, however, *according to experience,* the differential diagnosis *depends upon the slow, progressive character of the atrophy and the typical, fascicular course of the same, from one muscle to the other, usually in a distinct sequence.*

From *amyotrophic lateral sclerosis,* spinal progressive muscular atrophy differs especially in the fact that in the course of the latter an increase of the tendon reflexes and spastic paresis never occurs; the course of amyotrophic lateral sclerosis is also decidedly more rapid. The same is also true of *acute anterior poliomyelitis* as well as of the *subacute* and *chronic forms.* The latter can only be differentiated, as was shown in the previous

chapter, by certain deviations in its course from that of progressive muscular atrophy.

A confusion of the disease with *syringomyelia* is possible, at least only upon superficial examination, in that, besides the muscular atrophy, the *disturbances in the sensory sphere* are nearly always most markedly developed; this at once influences the diagnosis. Also the *trophic* changes in syringomyelia are more severe and more constantly developed than in muscular atrophy, in which if they occur at all they are very rare and are subordinate to other symptoms.

Differentio-diagnostic difficulties finally arise on account of certain forms of muscular atrophy which only very lately have been separated from the spinal progressive forms, and are designated as *neural muscular atrophy*, and still another variety as *primary myopathic muscular atrophy*. We shall devote special consideration to these two forms in the following chapter.

Regarding the last *cause of progressive muscular atrophy* we are by no means clear; probably hereditary conditions play a part, i. e., an *innate weak condition of the motor system* in these affections. We see these diseases without exception, one of them to a less extent than the other, in some *families*, in various members of the same, arising with a certain degree of regularity. The degenerative disease of the hereditary weak motor system, becoming active, especially due to infections and overexertions of the muscles, as the anatomical findings have shown which are in our possession up to the present, affect the motor neurons, damaging them at one time in their peripheral parts, at other times in the spinal cord. We have already learned to recognise in this connection amyotrophic lateral sclerosis and spinal muscular atrophy as types; as types of progressive muscular atrophies which damage the peripheral motor neuron and its end apparatus, the muscles, which are influenced peripherally from the anterior horn cells, we may regard neural muscular atrophy and muscular dystrophia. In the former the peripheral motor-nerve fibres, in the latter the muscular-nerve fibres themselves are primarily affected. We may disagree whether these strict separations of individual forms from one another are justified or not, as transitions from one form into the other certainly occur, and as the differential diagnosis between the individual types is not a strict one and partly, unquestionably, appears somewhat artificial. For the present it seems to me however to be correct to adhere to the individual varieties, even if, in the diagnosis, the limitations of the same must be clear to us.

NEURAL MUSCULAR ATROPHY (PERONEAL TYPE OF PROGRESSIVE MUSCULAR ATROPHY)

This variety of progressive muscular atrophy is commonly a family affection, characterized by the localization of the slowly developing muscular atrophy to the distal parts of the extremities. In the greatest majority of cases, the *lower extremity is primarily affected, particularly the muscles of the foot* (especially the peronei, the tibiales antici, and the extensors of the toes). This produces the typical development of a bilateral peroneal paralysis: The absence of the dorsal flexion of the foot, and the extension of the toes, and the difficulty in raising the external border of the foot; the tip of the foot hangs flaccid, etc. (compare p. 494). Gradually the muscles of the lower leg, especially the muscles of the calf and the muscles of the thigh, become atrophic. Similar conditions occur in

the *upper* extremity, i. e., the small muscles of the hand are first affected—the fingers take on the claw-position, the hand, as a result of the atrophy of the interossei and lumbricales, shows deep furrows, etc. Later the muscles of the forearm, especially the extensors, atrophy, whereas the muscles of the trunk and face usually are spared and bulbar phenomena are absent; the bladder and rectal function shows no disturbance. The *electrical* reaction, in keeping with the process in the motor-nerve fibres, shows: diminution or complete absence of electrical contractility, and on the part of the muscles DeR. It is easily understood that there will be diminution, respectively a loss, of the tendon reflexes, and the presence of fibrillary contractions.

The *differential diagnosis* between neural and spinal myopathic atrophy is based particularly upon the course of the atrophy, its onset in the lower extremity, and upon the circumstance that, besides the motor fibres, frequently sensory fibres are also affected and disturbances in sensation, such as paræsthesia, pain, and diminution of sensation, are observed.

From an anatomical standpoint neural muscular atrophy is a form of hereditary chronic progressive *neuritis,* which is especially limited to the motor-axis cylinders. The ganglion cells of the anterior horn remain intact, whereas Goll's columns are occasionally found degenerated.

DYSTROPHIA MUSCULARIS PROGRESSIVA (ERB)—JUVENILE MYOPATHIC MUSCULAR ATROPHY—PSEUDO-HYPERTROPHY

This affection was formerly frequently observed, but was not sufficiently defined, neither in a clinical nor in an anatomical respect; we owe our precise knowledge of this disease particularly to Erb. The affection appears in various forms which are clinically separated from one another, they have, however, but one anatomical foundation, namely, the *localization of the morbid process to the muscles, whereas the spinal cord as well as the peripheral nerves remain intact.*

Condition of the Muscles.—Usually, and primarily in early life, *muscular atrophy of the muscles occurs in the region of the shoulder, upper arm (especially in the flexors) and in the trunk,* later, in the muscles of the lower leg. Those *parts which remain longest free from atrophy are*: The *muscles of the calves* and the small *muscles of the foot* as well as the rectus abdominis, the deltoids and the muscles of the forearms, and, what is of special importance in a differentio-diagnostic respect, also the *small muscles of the hand.* In contrast to the atrophy of the previously mentioned muscles, there develops simultaneously a *conspicuous increase in size of certain muscles,* especially of the deltoid, the supra- and infraspinatus, the brachial triceps, and the gastrocnemii, etc. This increase in volume of the muscles is partly an expression of actual hypertrophy, so that these greatly developed muscles show compensatory functional increase; but, in these muscles later atrophy also becomes prominent. In an anatomical respect there are found: Hyperplasia of the interstitial connective tissue with deposit of fat, proliferation of the nucleus of the muscles and of the connective tissue. The muscle fibres themselves show simple atrophy or even

hypertrophy, in general, however, are not fatty and retain their transverse stripes. The *electrical contractility* of the muscles shows simple diminution up to complete loss; with this, however, distinct *DeR* are absent, and, just so, are there almost invariably *absent fibrillary* contractions in the affected muscles.

Condition of the Nervous System.—The *tendon reflexes* are retained, in more marked grades of the disappearance of the muscles they may be *reduced* or entirely absent. *Sensation, sphincter function, and trophic conditions of the skin are normal.*

Pseudo-Hypertrophy.—In some cases of juvenile muscular atrophy the "hypertrophy" of the muscles is predominant, i. e., the development of the interstitial fat hyperplasia is so marked, compared to the "atrophy" of the muscles, that a muscular-(pseudo) hypertrophy becomes the principal external manifestation of the affection. As already remarked, the calves and the deltoid, etc., show this condition most prominently, they appear as thick masses of muscles, especially in a stage of the affection in which the muscles of the trunk, arm and shoulder already show decided atrophy. The gait and carriage of the patient is very characteristic : The gait is waddling, the vertebral column in the lumbar portion bent forward, the patients (children) cannot sit down correctly and rise with great difficulty, they "climb up upon their own legs," so that they first rest upon the four extremities, then place the arms upon the knees and gradually rise from this position.

FIG. 36.—TYPE OF PSEUDO-HYPERTROPHIA MUSCULARIS. (After Duchenne.)

The *"infantile atrophic form of muscular dystrophy"* therefore is differentiated from the usual form of juvenile muscular atrophy in that it develops in earliest childhood and in that an atrophy of the *muscles of the face* is particularly prominent. In such cases the patients—usually children of the male sex—are not able to close the eyes nor to move the lips, whereas chewing and the movement of the internal muscles of the eyes are not impaired. Simultaneously or usually later than the muscles of the face, the muscles of the shoulder, trunk and upper leg become atrophic.

The separation of the different types of muscular dystrophy from one another as individual diseases is unnecessary, as the transition of individual forms into one another and the identity of the same, in general, remains. This only gives rise to confusion in making the diagnosis. In my opinion, it is sufficient, in an individual case, to add the prominence of pseudo-hypertrophy, or the affection of the muscles of the face, to the diagnosis.

It is characteristic of this affection, in all its forms, that, as a rule, it has a *family tendency,* respectively, that heredity plays a most important

part in its genesis. Further, that it almost always develops in childhood, or at least begins at puberty. The designation of progressive muscular atrophy as *juvenile* myopathic muscular atrophy is therefore certainly justified.

Differential Diagnosis.—There are no difficulties in the diagnosis of the disease; it can only be confounded, and this upon very superficial examination, with the spinal, respectively neurotic, progressive muscular atrophy, from which it must under all circumstances be distinctly separated, as in myopathic dystrophy the findings in the nervous system have up to the present been, generally, negative. Moreover, in this latter affection, anatomical changes in the muscles are found which are the result of a hereditary predisposition, causing a disturbance of nutrition of the muscular fibres which, during the period of marked growth, develop atrophy. But also the clinical phenomena vary greatly in both diseases; the following differential table may facilitate the diagnostic separation of both affections.

SPINAL PROGRESSIVE MUSCULAR ATROPHY	MYOPATHIC PROGRESSIVE MUSCULAR DYSTROPHY
Onset of the disease in the small muscles of the hand, rarely in the deltoid; the atrophy in general advancing from the periphery to the trunk; muscles of the face remaining free.	Onset in the muscles of the trunk, region of the shoulder and upper arm, the dystrophy in general advancing from the trunk to the extremities. Muscles of the face in some cases especially markedly implicated.
Muscular hypertrophy *absent.*	*Muscular hypertrophy present* besides atrophy.
Fibrillary contractions are very usually present.	Fibrillary contractions almost invariably absent.
The atrophic muscles show partial or complete *DeR.*	The atrophic and pseudo-hypertrophic muscles show diminution of the electric contractility; however, *DeR are scarcely ever present.*
The affection may develop at any age; however, before the thirtieth year.	The disease begins in earliest youth, i. e., almost always before the twentieth year of life; it is particularly a *juvenile* disease.
Complicated by bulbar symptoms (which follow atrophy of the muscles of the trunk and extremities, occasionally preceding) is not very rare.	Bulbar symptoms are invariably absent.

Common to both affections is the absence of disturbances of sensation and of the sphincter function, further—and in contrast to *amyotrophic lateral sclerosis*—the diminution or disappearance of tendon reflexes and the absence of spastic pareses.

POSTERIOR POLIOMYELITIS

In cases in which the *posterior* portions of the gray substance are affected, alterations in the vaso-motor innervation, analgesia and thermo-anæsthesia, eventually also a hyperæsthesia affecting the tactile sense, as well as sensory ataxia (Clarke's columns), and reflex disturbances, are to be expected. The symptomatology of poliomyelitis posterior is by no means determined clinically with certainty. A portion

of these theoretically pustulated morbid phenomena form an affection of the spinal cord in which the gray substance, the anterior as well as the posterior portion, is more or less exclusively affected, this is *syringomyelia*, in fact this is occasionally the nucleus of the entire pathological picture.

SYRINGOMYELIA—CAVITY FORMATION IN THE SPINAL CORD—GLIOSIS SPINALIS

Syringomyelia is especially due to the formation of cavities in the spinal-cord substance; in a pathologico-anatomical respect there have been distinguished in general from syringomyelia, *hydromyelia*, a condition in which, by faulty development or also now and then by circulatory disturbances and engorgement which occur in the posterior cavity of the skull due to tumours, the central canal of the cord remains wide or becomes so. As a rule this causes the adjacent spinal-cord substance to be uniformly pushed aside, giving rise to the absence of all clinical symptoms. Yes, the latter condition may apparently be the case if the nerve elements of the gray substance *gradually disappear* due to the further enlargement of the cavity formation and the postulated functional disturbances are supplied by the compensatory action of the still normal portions of the gray substance. In other cases the clinical results of hydromyelia are identical with those of *syringomyelia*.

The latter affection is due to a decay of *glia masses*, which develop in the central portion of the spinal cord or in the region of the posterior sæptum, and to a greater or lesser extent reach into the gray and white substances, especially into the posterior horns. The new formation gives rise to a cavity due to the disintegration, communicates, according to its formation, with the central canal and the surrounding cells; occasionally a second cavity is formed alongside. Sometimes syringomyelia may appear as the result of trauma, from hæmorrhages of the central cord substance (central hæmatomyelia), in that a secondary formation of larger cavities occurs due to the hæmorrhage. The symptoms which arise in the affection must vary according to the seat and distribution of the tumour and cavity formation; however, in most cases at least, a certain uniformity is present, so that recently a definite clinical picture has been determined which is of value in the diagnosis of the affection.

Important Symptoms Diagnostically—Sensory Disturbances.—If, as is usual, the affection appears primarily in the region of the central canal of the cervical enlargement and involves the *posterior gray matter*, we note (1) in the upper extremity, in the region of the neck and trunk, *disturbances in the temperature and pain senses* (thermo-anæsthesia and analgesia), eventually also *paræsthesia*, whereas the *tactile sense and muscle sensation are but slightly, if at all, altered*. The phenomenon is not difficult to explain if we reflect that the sensory muscle fibres form the principal constituent of the posterior columns, therefore, in an affection of the posterior gray matter of the cord, they are not implicated, the tactile fibres, although they enter the gray substance, they leave it again, very probably without coming in contact with cells (in the border stratum of the lateral columns), whereas the conduction of the temperature and pain senses most likely *only occurs in the gray substance by the action of the ganglion cells of the posterior horns* (see Fig. 30). A pathological process which, at least in its onset, affects particularly the gray substance, will therefore damage the latter tracts and primarily cause analgesia and thermo-anæsthesia.

According to our knowledge regarding the direction of the tracts for the vaso-motor innervation, it is easily understood that the disappearance of the gray substance gives rise to (2) *vaso-motor disturbances.* As a matter of fact, in cases of syringomyelia, well-distributed trophic, secretory and vaso-motor alterations of the skin and the deeper tissues have been noted. Vesicles, wheals, cutaneous œdema, bedsores, arthropathies, joint deformities, fissures in the hands, paronychia, phlegmons, distortion or hypertrophy of the fingers, etc., so that the clinical picture may almost resemble *lepra mutilans,*[1] also anomalies in the *secretion of sweat,* especially hyperidrosis, occur as symptoms of an implication of the gray substance of the cord. The *cutaneous reflexes,* in individual cases, vary greatly: Occasionally they are normal, sometimes reduced or even entirely absent. The palpebral fissure and pupils have rarely been found narrow, the functions of the *rectum and bladder,* as might be expected, have frequently been noted as disordered; rarely are there changes in *co-ordination.*

Motor Disturbances.—Whereas an anatomical lesion has been determined for the previously described symptoms, a localization of the process to the posterior commissure, the posterior horns, and eventually also to parts of the posterior columns, an atrophy of the *anterior horn,* has been found to correspond to the sensory, trophic and reflex disturbances (this may be noted in such cases post mortem) which give rise to (3) the *very common flaccid amyotrophic paralysis.* At one time the muscles of the upper extremities, at another the muscles of the trunk and but rarely those of the lower extremity are atrophied, giving rise to the clinical picture of progressive muscular atrophy with its symptoms (furrows in the spaces between the bones of the midhand, disappearance of the ball of the thumb and of the little finger and development of the claw-position of the hand). The electrical examination, in the various cases, shows DeR, but very rarely is there no marked disturbance of the electric contractility. If the process spreads in a transverse direction, it depends whether the pyramidal columns or the posterior columns are more affected. In the former cases, paresis and spastic phenomena occur as in amyotrophic lateral sclerosis (but with sensory disturbances!); in the latter case, symptoms resembling tabes arise, i. e., disappearance of the tactile and the muscle senses, the relatively intact condition of which, as a rule, is present, at least primarily, which is in marked contrast with the highly developed analgesia and thermo-anæsthesia. If the cavity formation develops upward, *bulbar symptoms* occur (hemianæsthesia, paralysis of the recurrent laryngeal and hypoglossus, nuclear facial paralysis, abducens paresis, polyuria, melituria, etc.),

---

[1] Such cases were described as a special disease, *Morvan's disease*; besides analgesia and thermo-anæsthesia, there are present tactile hyperæsthesia, fissures, and as a most marked symptom, felon of the fingers, which runs a painless course and leads to disorganization of the end phalanges. A separation of these cases from the clinical picture of syringomyelia is unnecessary. On the other hand, the symptoms in *lepra* differ from those of syringomyelia, in that, in the former affection, a *multiple disease* of the peripheral nerves occurs due to the infection with the bacillus of leprosy which shows itself in swollen nerves, painful upon pressure, peripheral facial paralysis, etc.

which may even arise as complications late towards the termination of life. If the gliomatous development is limited to *one* side of the spinal cord, the morbid phenomena are more or less unilateral.

Differential Diagnosis.—From what has been said it is obvious that the clinical picture of syringomyelia varies greatly, and that the diagnosis frequently gives rise to great difficulty. In keeping with the anatomical location of the process, the sensory disturbances or the muscular atrophy will be more prominent and the clinical picture will resemble either tabes or muscular atrophy; in other cases, as has been mentioned, syringomyelia runs a course resembling amyotrophic lateral sclerosis. If a differentiation of the disease in question from the previously mentioned spinal-cord affections is to occur, there must be in the clinical picture, *as a nucleus, a symptom-complex which the expression of an affection of the gray substance of the spinal cord,* presenting itself particularly in the form of partial paralysis of sensation (analgesia and thermo-anæsthesia with simultaneous relatively intact condition of the tactile and muscular senses), severe vaso-motor disturbances and muscular atrophy, in which, to a more subordinate degree, or as late symptoms, hyperidrosis, narrowing of the pupils and lid space, disturbances of the functions of the bladder and rectum and of co-ordination occur. If this typical symptom-complex is prominent in the clinical picture, we are justified in making a diagnosis of syringomyelia, even if in the course of the affection, with the propagation of the process, phenomena arise which more closely resemble other affections of the spinal cord.

That certain *tumours of the cord substance* give rise to similar symptoms is obvious. In this respect we may only assume the probable presence of tumours if marked irritative phenomena take place and the course of the disease is a rapid one, more rapid than in the case of syringomyelia in which the disease is very protracted, lasting for many years.

DIFFUSE MYELITIS (ACUTE AND CHRONIC MYELITIS, TRANSVERSE MYELITIS)

In contrast to the affections of the spinal cord which we have considered up to now affecting certain anatomically and functionally related portions of the cord, in the affections which now are to be considered there are anatomical changes which implicate the longitudinal and transverse portions of the cord diffusely, unilaterally or in irregularly disseminated areas. Regarding the diagnosis, it is characteristic that it is impossible to refer the clinical phenomena to an *isolated* affection of individual portions of the spinal cord that supply certain physiological functions.

The diagnosis of *diffuse myelitis,* which shall be considered now, depends upon a number of *functional disturbances affecting simultaneously the motor, sensory and further the vaso-motor, trophic and reflex tracts in the spinal cord,* in which one or the other of these tracts may be particularly damaged.

Motor and Sensory Disturbances.—Regarding the *motor* symptoms, they are almost always particularly prominent. Irritative phenomena (contrac-

tion and spasm) are not rare; usually from the onset the signs of weakness occur. These soon increase to paralysis, and in the diffusely distributed process in myelitis it is self-evident that *both* halves of the body are affected by the paralysis (*paraplegia*). Usually the *lower* extremity is exclusively paralyzed, the *upper* extremity with the lower only when the myelitic changes have their seat above the thoracic cord. If the process is more developed in one half of the spinal cord than in the other, an unequal intensity of the paralyses of the extremities will result. If the myelitis affects especially the gray substance in its anterior portion, atrophy occurs in the flaccid paralyzed muscles, with signs of diminution of the electric contractility in the sense of DeR. This, however, is exceptional and only occurs in those muscles the innervation of which is supplied by the region of the myelitic lesion. As a rule, the nutrition of the muscles is not interfered with, in so far as the distribution of the myelitic process is a moderate one; outside of slight quantitative changes in the electic irritability the reaction of the paralyzed parts is normal to the electric current.

The *sensory disturbances* are almost always *insignificant*; severe pains are absent in the clinical picture of uncomplicated transverse myelitis,[1] but in a simultaneous meningitis and spondylitis they may be prominent, giving rise to girdle pains and radiating pains of the extremity. The diminution of sensibility, as might be expected, is not markedly developed, but only hypæsthesia may be demonstrated. This probably is due to the fact that the conduction of sensory irritation is distributed to a very large territory comparatively (posterior columns, the greatest portion of the gray matter of the cord, as well as the lateral and anterior columns). Only in the late stages of the disease, or from the onset in a very diffuse myelitis, do severe sensory disturbances occur, such as complete anæsthesia, the development of which may be preceded by partial paralysis of sensation, slow perception, etc. Exceptionally, also hyperæsthesia and hyperalgesia are noted. Under all circumstances, the distribution of alteration of sensation is to be tested in each case with exactness, as by this means we are enabled to determine the upper boundary of the myelitic affection.

Condition of the Reflexes.—The *condition of the reflexes* is also important in the diagnosis. If the myelitic area of the spinal cord occurs in the entire transverse section, but only in a limited longitudinal portion, the reflexes which occur in the reflex arcs below the lesion are not only retained, but, on account of the simultaneous interruption of the inhibiting reflex influences coming from above, are *increased*. If, as is very frequently noted, the reflexes diminish more and more in the further course of the affection, this is a good sign if simultaneously the paralytic phenomena are also reduced. It is a bad sign, however, if the improvement of both con-

[1] Exceptions occur: In a case of myelitis following influenza, which I observed for months, before the autopsy was held, severe pains were present for a long time, being the most prominent symptom of the affection, whereas the autopsy showed simple myelitis, without the merest trace of a disease of the vertebra or of the meninges.

ditions does not go hand in hand. In the latter case it must be concluded that the myelitic process has distributed itself farther downward, especially into the region of the affected reflex arc, and has diminished the property of conduction or has abolished it entirely. After what we know regarding the position of individual reflex arcs, which may at least be assumed as very likely, with an increase of the *patella tendon reflexes,* the arc of which is situated in the upper portion of the lumbar cord, it must be concluded that the myelitis has its lowest extent in the upper portion of the lumbar cord, and with a simultaneous increase of the tendon reflexes in the upper extremity, that a cervical myelitis is present which does not reach to the dorsal cord. In cases in which the tendon reflexes are increased, the paralysis may also assume a *spastic* character. Upon the cessation or increase of the *Achillo-tendon reflex,* in a similar manner the diagnosis of the seat of the myelitis may be determined; upon an absence of the same, an affection of the lowest part of the lumbar cord may be assumed, the region of the reflex arc of the Achillo-tendon reflex being situated in this position. The condition of the *cutaneous reflexes* is very variable; an explanation of the same, in the individual case, must be looked for in the previously described points of support (compare especially p. 523).

Trophic Disturbances.—Similar conclusions may be gathered from certain trophic disturbances according to the condition and limitation of the reflexes. If the volume of the *muscle* remains normal or but slightly reduced (in a longer duration of the paralysis, the absence of any active movement of the limbs may cause slight atrophy of the muscles (atrophy of inactivity) which are the opposite of the hypertrophy induced by activity of the same), and if the electrical contractility is maintained, it may be assumed that the portion of the spinal cord in which the central trophic activity for the affected muscles arises, especially the anterior columns of the various portions of the spinal cord are not affected by the myelitic process. If, on the contrary, paralysis with atrophy and DeR occurs in the limbs, it may be concluded: In atrophy of the lower extremities, that the *lumbar cord* is the seat of the myelitis, this myelitis affecting the anterior columns of the lumbar cord, in atrophy of the upper extremities, that the cervical cord is affected in its anterior horns or its anterior roots. If the inflammatory process is limited to the *upper portions of the spinal cord,* the lower extremity is paralyzed, besides the upper extremity on account of an interruption of conduction, but, in contrast to the condition of the upper extremities, there is seen in the lower neither rapidly advancing atrophy of the muscles nor qualitative changes in their electrical reaction. Also *trophic, respectively vaso-motor, disturbances of the skin* are frequently noted in the course of myelitis—marbling of the cold cutaneous covers, œdema, disturbances of sweat secretion, urticaria, or, as in one of my cases, a wide-distributed eruption of herpes upon the backs of both feet.

Bedsores.—Above all, and very frequently, a rapidly developing *bedsore* occurs, the origin of which, very probably, is in connection with disease of the middle portion of the gray substance, and the distribution of which depends upon external pressure, the immovable position of the insen-

sitive patients, and upon the contact, at the surface of the wound, of urine and fæces.

Disturbances in Urination.—*Disturbances in the bladder and rectal functions* are very common in myelitis. The diagnosis of paralysis of the individual factors in the function of the bladder has been described in another chapter (see p. 401). We are interested in the question whether the appearance of disturbance of the bladder and rectal functions may be utilized in the topical diagnosis of the spinal-cord affection. In this connection, we are not in possession of the required precise well-determined points of support regarding the course of the innervation tract for the bladder function, and even more so for those of the rectum, both from a physiological and clinical standpoint. The little that we know in regard to the innervation and conditions of the bladder which may be assumed as likely are as follows: The nerve fibres for the voluntary contraction of the sphincter urethræ and also for those of the voluntary inhibition of the reflex sphincter contraction arising in the brain, seem to run through the anterior columns and the posterior portion of the lateral columns. The arc for the reflex contraction of the detrusor and sphincter vesicæ lies in the sacral cord. As the reflex sphincter contraction, due to filling of the bladder, is more powerful than the reflex detrusor contraction, a myelitis, above the lumbar cord, will cause *distention of the bladder* and *retention of urine*, especially as also the interruption of conduction produces an inability in the voluntary inhibitive fibres for the reflex sphincter contraction. To these symptoms, further on then, *incontinence* is added, in that the bladder becomes overloaded and the tracts for the voluntary contraction of sphincter urethræ are interrupted. The interruption in the conduction tract of the *sensory* nerves which convey the sensation for the passage of urine, which soon after their entrance into the spinal cord ascend into Goll's columns, produces loss of sensation for the desire of urination. This causes the proper voluntary sphincter contraction and relaxation to be absent, and as the reflex arc suffers an interruption, due to the incapability of conduction in the sensory tracts, incontinence occurs as soon as the bladder gradually fills up to a certain degree, so that the elasticity of the tissues surrounding the urethra is no longer sufficient to retain the urine in the bladder. By invasion of micro-organisms into the bladder from without, which, due to the inability of the sphincter to close, occurs more readily and develops easily, causing a stagnation of urine, the bacteria exert their activity, decomposition of the urine results and *cystitis* develops, and, in the further course of the affection, eventually pyelonephritis may take place.

Disturbances in the Discharge of Fæces and in the Sexual Function.—The reflex arc for the contraction of the *anal sphincter* is situated in the lumbar cord. The mechanism of innervation, in the discharge of fæces, appears to be analogous to the voiding of urine, and the interruption of conduction of the affected nerve tracts, above the lumbar cord, also, primarily, gives rise to a *retention* of the contents of the rectum; this may, however, occur also in a myelitis, the seat of which is above the lumbar cord, due to a paresis of the abdominal press. Later, in the severe cases, an *incontinentia alvi* arises if the paralysis of the sphincter is complete. Finally, it is usual in myelitis that the *sexual function* is markedly disturbed (the centre for erection is also situated in the lumbar cord).

By observing the symptoms described it is easy, as a rule, not only to diagnosticate a myelitis, but also its seat in the various portions of the spinal cord with at least an approximate certainty. In the latter connection a brief comparison of the individual diagnostic points of support shall be given:

Lumbar Myelitis.—Paralysis of the lower extremities, whereas motility and sensation of the upper extremities remains intact; sensory disturbances of the skin in the region of the sexual organs, of the hip and of the lower extremities; generally degenerative atrophy of the muscles of the leg, qualitative changes in their electric contractility, absence of cutaneous and ten-

don reflexes in the lower extremity; paralysis of the bladder and rectum, incontinence upon moderate fullness of the bladder, trophic disturbances in the legs and in the gluteal region: Œdema, eruption of vesicles, and, above all, early severe bedsores.

Dorsal Myelitis.—Sensory disturbances of the skin, reaching to the region above the umbilicus. Motility and sensation in the upper extremities intact; paraplegia of the lower extremities, but, in contrast to lumbar myelitis, without degenerative atrophy and DeR. Increased tendon reflexes in the paralyzed legs, spasmodic contractions in the same, rigidity and later contractures of the musculature of the lower extremities. Rectal and bladder disturbances, especially retention of urine, and incapability voluntarily to hold the urine, later incontinence when the bladder has become greatly dilated; bedsores, difficulty in respiration, especially of expiration.

Cervical Myelitis.—The same symptoms as in dorsal myelitis regarding the condition of the lower extremities, the bladder, the rectum, etc. Simultaneously, paralysis of the *upper* extremities, respectively of some muscles—in diffuse myelitis simultaneously with atrophy and qualitative changes of electrical reaction of the muscles and cessation of the reflexes. In a low situation of the cervival myelitis (eighth cervical and first dorsal segments), oculo-pupillary symptoms: Contraction of the pupil and of the lid space. If the myelitis is situated in the upper portion of the cervical cord, disturbances of respiration occur, especially of inspiration, on account of paralysis of the diaphragm (second to the fourth cervical segments). With this, degenerative atrophy, DeR, and the disappearance of the tendon reflexes in the muscles of the arms are absent, whereas difficulty in deglutition, anarthria and other bulbar symptoms arise.

Still further to specialize the seat of the myelitis from a practical standpoint I do not regard as necessary.

Differential Diagnosis between Chronic and Acute Myelitis.—Whether the myelitis in the individual case is to be regarded as *acute* or *chronic,* depends upon its development, its duration, and its consequences. Chronic myelitis, which has a gradual onset, and in its entire course is characterized by a gradual, creeping onset of the individual spinal symptoms, shows occasionally acute exacerbations in the development of the affection. Nevertheless, we should only speak of acute myelitis if the principal symptoms, the paralytic phenomena, develop in a relatively short period, i. e., arising completely in a few weeks; in such cases the affection may run its course with *fever,* which will vary regarding its character and height; however, being a symptom which is of no value in the diagnosis of the affection.

If paraplegia develops suddenly, we should think primarily of a hæmorrhage into the spinal-cord substance; but it must not be forgotten that acute myelitis may also occasionally develop very rapidly—overnight—and all the clinical symptoms be markedly developed. At most, a fulminant appearance of the paraplegia (in a few moments) without any prodromal phenomena preceding the attack would favour spinal apoplexy, especially if certain ætiological conditions were present which would make the development of a spinal-cord hæmorrhage likely.

Differential Diagnosis from Other Diseases of the Spinal Cord.—Regarding the differentiation from other affections of the spinal cord, not rarely do great difficulties arise. In acute myelitis, the differential diagnosis especially between this affection and *acute spinal meningitis, multiple neuritis* and *Landry's paralysis* must be considered. The diagnosis of the latter affection will be described later on, when the differentio-diagnostic points will be carefully considered.

Spinal Meningitis.—The differentiation from acute *spinal meningitis* is in so far difficult as both morbid processes very usually occur combined, and a portion of the symptoms in the clinical picture of spinal meningitis are due to the simultaneous affection of the cord in the inflammatory process. In favour of meningitis, especially of its prominence, are: Intense irritative phenomena, hyperæsthesia, marked pains, especially upon movement of the vertebral column, stiffness of the neck and back, whereas in myelitis the paralytic phenomena, both of the motor and sensory tracts, the trophic disturbances, the paralysis of the bladder, and the increase of the reflexes are prominent.

Polyneuritis.—*Multiple neuritis* can only be considered in a differentio-diagnostic respect, if in its course the paralytic phenomena are limited to the lower extremities, and, besides muscular atrophy and DeR in the flaccid muscles, a blunting of sensation may be determined. In such cases, the question may arise whether a localized neuritis or transverse myelitis of the lumbar cord is present. In favour of the former is a prominence of the pain, the pain upon pressure on the nerve trunks, the limitation of the anæsthesia to individual nerves, further, the absence of disturbance on the part of the bladder and rectum (although these symptoms may occur in rare cases of neuritis, without an affection of the spinal cord), and especially also by the absence of bedsores. If the myelitis is situated higher up, a confusion in both affections can no longer arise, as the increase of the reflexes and the absence of muscular atrophy, with its consequences, are directly against neuritis.

Hysterical Paralysis.—*Hysterical paralysis* also occurs as a paraplegia and may simulate myelitis. Its exclusion is easy if the (myelitic) paralysis is a flaccid, atrophic one, bedsores occur and DeR can be determined. The diagnosis is more difficult if the paralysis develops in the lower extremities, without the signs of a lesion of the lumbar cord, the myelitis, therefore, having its seat farther up in the cord. The increase in the reflexes which occurs may then also, even if rarely, appear in hysterical paralysis, and just so may symptoms of paralysis of the bladder be present in hysteria. In such cases, the observation of the entire clinical picture must determine the diagnosis, i. e., the combination of actual hysterical phenomena in other parts of the body, attacks of laughing and crying, globus, hysterical vomiting, the pressure points upon the skull, vertebral column and the abdominal coverings, the limitation of the field of vision, etc., above all, however, the alteration of the psychical conditions, and the unmotived change of the nervous phenomena, usually permit us to make a diagnosis in favour of hysteria, even if occasionally, at least for a time, the opinion may waver and an exact differential diagnosis be impossible.

Great difficulties occur in the differentiation between transverse myelitis and *intramedullary tumours.* The symptoms are the same in both affections; important is the observation of the course in the individual case, in that an inequality of the phenomena from the onset upon both sides ("unilateral lesion") and especially the variation of the intensity of the phenomena favour a tumour, a simultaneous rapid increase of the spinal-cord affection being against the presence of a tumour.

Ætiology.—The *ætiology* in the individual case offers very little that is of use in the diagnosis. Marked refrigeration, severe psychical stimulation (such as vasomotor disturbances) may, as is assumed, produce myelitis, but they may also give rise to other affections of the nervous system which must be considered in a differentio-diagnostic respect with this affection. It is more likely that previous infectious diseases (enteric fever, variola, influenza, etc.) are of value in the diagnosis of myelitis; syphilis especially appears to play an important *rôle* in the production of the disease, and to give rise particularly to a partial myelitis, showing itself with the clinical phenomena of spastic spinal paralysis (see p. 554). Also disease of the urinary organs, especially gonorrhœa, not rarely gives rise to secondary myelitis by a distribution of the inflammation *per contiguitatem*, i. e., by the encroachment of the same upon the cellular tissues (in all cases much rarer due to an "ascending neuritis"). More important in the diagnosis of acute myelitis is the occurrence of a previous shock of the spinal cord (such as occurs in railway accidents—railway spine—although the result of these accidents more frequently produces functional neuroses, eventually of the spinal type, than myelitis). The observation of the possibility of a myelitis being due to the development of gas in the vessels and in the spinal-cord substance in individuals who for a long time have remained under the influence of an increase in atmospheric pressure ("caisson paralysis") or the proof of tumours which affect the spinal cord and the vertebral column are important. It is advisable, above all, carefully to examine the vertebral column regarding pain and deviation of the individual vertebræ. Should this show a positive result, the diagnosis gains in a very important direction. There must now be considered pressure paralysis or also "*compression myelitis*," affections of the spinal cord which shall be described in the following chapter.

COMPRESSION OF THE SPINAL CORD, SPINAL-PRESSURE PARALYSIS, COMPRESSION MYELITIS

As soon as the spinal cord is subject to permanent pressure from external causes (as by meningeal exudative masses, tumours of the meninges or of the vertebræ, syphilitic new formations in the vertebral canals, etc., but above all by *tuberculous caries of the vertebral bodies*) there develop the *symptoms of an interruption of conduction in the spinal cord,* which were spoken of in myelitis. These symptoms appear particularly as *paralyses* which, according to the region of the pressure, may affect the lower extremities, these and the trunk, in other cases the lower extremities simultaneously with the trunk and the arms. With this the reflexes, to a less extent the cutaneous reflexes (which may even be diminished) than the tendon reflexes in the paralyzed parts below the points of compression, are *increased,* so that, as a result, *spastic paralysis* may occur. On the other hand, occasionally degenerative atrophy of the muscles follows, their source of innervation being situated in the compressed area of the spinal cord. The *disturbances in sensation* which are to be expected in an interruption of the conduction tracts, as in the case of transverse myelitis, *are slight in*

of the patella-tendon reflexes diminishes in spite of the continuance of the paralysis of the lower extremity, eventually also marked atrophy and qualitative changes in the electric contractility in the muscles of the legs arise, or if to the resulting symptoms of compression of the dorsal cord, as is occasionally observed, paralysis of the arms or even bulbar symptoms are gradually added. Regarding the clinical unimportance of the necessary *secondary* degeneration combined with a transverse interruption of conduction, this has been previously spoken of (from the point of lesion descending in the motor tracts, and arising in the sensory tract from the posterior columns and cerebellar lateral column tracts).

Seat of the Compression.—From a diagnostic standpoint it is important to determine the probable *seat* of the lesion of the spinal cord and further on to note whether the symptoms of the same harmonize with the position of the most external determinable cause of compression, in regard to localization and distribution. In this respect the diagnostic rules which were spoken of under the diagnosis of myelitis (see pp. 576, 577) are of use. They need not be repeated here.

Ætiological Diagnosis.—If the diagnosis of a pressure myelitis is certain, we must attempt to find the special cause of the compression. Regarding the consequences of pressure upon the spinal-cord substance by *meningeal exudates,* these have been described previously; more frequent—in by far the majority of cases—pressure phenomena are due to *tuberculous-carious disease of the bodies of the vertebræ.* It should therefore never be neglected repeatedly to examine the lungs to detect a catarrh of the apices and to investigate the sputum for bacilli. Caries of the vertebral column is found most frequently in the dorsal part, less frequently in the cervical part, or even in the lumbar portion of the cord. By caseation of tuberculous new formation, more and more substance of bone is destroyed—this causes a dislocation of the vertebral column posteriorly, as the spinous processes in the diseased part of the spinal column stand out prominently, so that an acute-angled kyphosis, a Pott's deformity, arises. Due to this cause and also to the collection of masses of pus in sacs, which are subperiosteally situated, compression of the cord occurs. Only after caries of the vertebra has been excluded should the rarer causes of compression be considered, primarily then, *carcinoma of the vertebra.* The great severity of the pains, continuing for a long time, due to proliferation of the carcinoma in the vertebral bones, and also to compression of the posterior nerve roots which cannot escape the growing carcinoma, is somewhat characteristic. A more important diagnostic symptom naturally is the gradual appearance, externally, which may be seen and felt, of a *round* tumour of the vertebra, which may be especially diagnosticated as vertebral cancer if the affected individual is in advanced life (between fifty and sixty years of age) showing enlarged glands, or if it is possible to show the development of carcinoma in other parts of the body (in the mammæ, the œsophagus, etc.). In other cases, compression of the cord is due to neoplasms in the neighbourhood of the vertebral column, encroaching upon the spinal canal by the intervertebral openings, or from erosion of the vertebral bodies, as in the case of aneurysms, the spinal cord being gradually approached by the growing tumour.

If, in spite of the gradual development or steadily increasing phenomena of compression, and notwithstanding pressure upon the spinal cord producing pains (and especially in movement), also upon longer duration of the affection and repeated careful examination, no signs of external changes of the vertebral column, no increase in size or deviation of the vertebræ can be determined, a *meningeal tumour* should be thought of as the cause of compression, or if the phenomena of irritation of the roots are entirely absent, an *intramedullary tumour* should be considered (see Tumours of the Cord).

In rare cases compression of the cord causes, if only one side is subjected to pressure, a clinical symptom-complex which is identical with the resulting phenomena occurring in animal experiments upon a unilateral severing of the spinal cord; these are so peculiar that they must be specially described.

UNILATERAL LESIONS OF THE SPINAL CORD—BROWN-SÉQUARD'S SPINAL PARALYSIS

As soon as the spinal cord is exclusively injured unilaterally, by a compression process (tumours, etc.) or by myelitis, trauma, and other conditions, we may expect, as the result of the lesion, according to our previous physiological and clinical experiences: In the parts of the body below the point of lesion, *unilateral motor paralysis upon the side corresponding to the lesion, unilateral cessation of tactile sensation upon the opposite side.* These symptoms are self-evident, according to what we know regarding the course and direction of the sensory and motor tracts. While the motor fibres below the pyramidal decussation in the spinal cord travel downward without crossing, the pain and temperature fibres, and also the tactile fibres, at least to a great extent, immediately after their entrance into the spinal cord pass through the gray substance towards the opposite side to ascend to the brain in the ground bundles of the anterior and lateral columns. The sensory fibres which ascend in the posterior columns without crossing conduct the cutaneous sense less than the *muscular sensation*, as has been explained previously, and the latter faculty therefore is *diminished upon the side of the motor paralysis*, as well as electro-muscular sensibility. Upon this side, at least in recent cases, also *vaso-motor paralysis* may be determined, the temperature being higher than upon the anæsthetic side (compare Fig. 30). The *condition of the reflexes* is not constantly altered; sometimes being increased, at other times diminished, and may even be normal; especially the *tendon reflexes upon the paralyzed side are generally increased* on account of the absence of the inhibitive reflex fibres coming from above which have already crossed in the spinal cord.

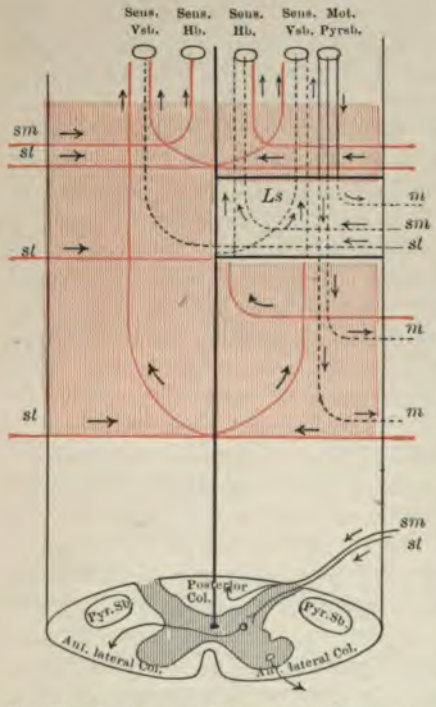


FIG. 37.—DIAGRAM TO EXPLAIN THE PRINCIPAL PHENOMENA IN UNILATERAL LESION.

Ls, Point of lesion, in red the region of increased irritability, ... interrupted ——— intact conduction: *m*, Motor and vaso-motor fibres; *s*, Sensory fibres, viz., *sm* for muscular sense, *st* for tactile sensation, *mot. Pyr = Sb*, Motor pyramidal lateral column tract; *sens. Vsb*, Sensory anterior lateral column tract; *sens. Hb*, Sensory posterior column tract.

Besides these chief alterations in the conduction of the fibres, the following less constant auxiliary symptoms which are apparently paradoxical are noted: *Hyperæsthesia*, respectively hyperalgesia of the motor paralyzed side, a more or less *small zone of anæsthetic area* above the hyperæsthetic region, corresponding to the height of the lesion, and above this a *girdle-like hyperæsthetic zone affecting both sides, disturbances in the evacuation of urine and fæces, ataxia* upon the paralyzed side, as soon as this side is again capable of motion.

The appearance of the small anæsthetic zone at the upper boundary of the region of motor paralysis is easily explained, as, with a unilateral lesion, a segmentary interruption, in keeping with the region of the lesion, occurs both in the sensory (root) fibres which enter and which have not yet crossed and in those coming from the opposite side which cross in the gray substance to the lateral column of the point of lesion. It is more difficult to explain the presence of hyperæsthesia upon the side of the motor paralysis and the narrow hyperæsthetic zone above the anæsthetic region of both sides. This condition may be most readily explained if we assume that the cord above and below the point of lesion is in a condition of increased irritation. The fibres which pass through these parts would then, while passing, become more irritable, which would show itself immediately above the point of lesion, in the cutaneous parts, as bilateral hyperæsthesia, whereas in the parts of the body below the point of lesion only one side (corresponding to the lesion) would be hyperæsthetic, because the contra-lateral side, on account of the reasons described above, must show anæsthesia. The latter is also true of the area of the sensory fibres which enter the irritated parts as they also, in their further course, pass the point of lesion. The diagrams (see Figs. 37 and 38) may somewhat more readily explain the apparently very complicated symptom-complex.

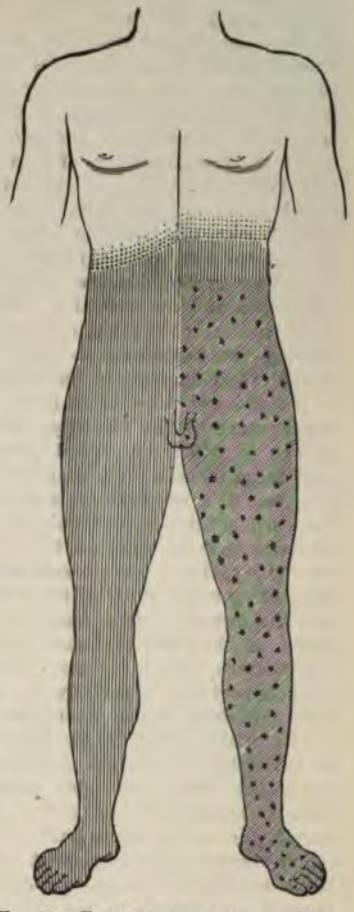


FIG. 38.—DIAGRAM SHOWING THE PRINCIPAL PHENOMENA IN UNILATERAL LESION OF THE DORSAL CORD (LEFT). (After Erb.)

The oblique shading signifies motor and vaso-motor paralysis. The vertical shading signifies cutaneous anæsthesia; the dots signify cutaneous hyperæsthesia.

TUMOURS OF THE SPINAL-CORD CANAL, TUMOURS OF THE SPINAL MARROW AND ITS MENINGES

Among the causes of compression of the spinal cord are also tumours which may develop in the canal of the cord and grow. They form a special group of spinal-cord affections, less so by their clinical picture than by their anatomical importance. The symptoms of value in the diagnosis produced by tumours in the spinal cord must vary greatly in the individual case, according to the distribution and situation of the neoplasm.

Meningeal Tumours.—The diagnosis of those tumours in the canal of the cord, which arise from the meninges, *the meningeal tumours*, present comparatively the

If, in spite of the gradual development or steadily increasing phenomena of compression, and notwithstanding pressure upon the spinal cord producing pains (and especially in movement), also upon longer duration of the affection and repeated careful examination, no signs of external changes of the vertebral column, no increase in size or deviation of the vertebræ can be determined, a *meningeal tumour* should be thought of as the cause of compression, or if the phenomena of irritation of the roots are entirely absent, an *intramedullary tumour* should be considered (see Tumours of the Cord).

In rare cases compression of the cord causes, if only one side is subjected to pressure, a clinical symptom-complex which is identical with the resulting phenomena occurring in animal experiments upon a unilateral severing of the spinal cord; these are so peculiar that they must be specially described.

UNILATERAL LESIONS OF THE SPINAL CORD—BROWN-SÉQUARD'S SPINAL PARALYSIS

As soon as the spinal cord is exclusively injured unilaterally, by a compression process (tumours, etc.) or by myelitis, trauma, and other conditions, we may expect, as the result of the lesion, according to our previous physiological and clinical experiences: In the parts of the body below the point of lesion, *unilateral motor paralysis upon the side corresponding to the lesion, unilateral cessation of tactile sensation upon the opposite side.* These symptoms are self-evident, according to what we know regarding the course and direction of the sensory and motor tracts. While the motor fibres below the pyramidal decussation in the spinal cord travel downward without crossing, the pain and temperature fibres, and also the tactile fibres, at least to a great extent, immediately after their entrance into the spinal cord pass through the gray substance towards the opposite side to ascend to the brain in the ground bundles of the anterior and lateral columns. The sensory fibres which ascend in the posterior columns without crossing conduct the cutaneous sense less than the *muscular sensation*, as has been explained previously, and the latter faculty therefore is *diminished upon the side of the motor paralysis*, as well as electro-muscular sensibility. Upon this side, at least in recent cases, also *vaso-motor paralysis* may be determined, the temperature being higher than upon the anæsthetic side (compare Fig. 30). The *condition of the reflexes* is not constantly altered; sometimes being increased, at other times diminished, and may even be normal; especially the *tendon reflexes upon the paralyzed side are generally increased* on account of the absence of the inhibitive reflex fibres coming from above which have already crossed in the spinal cord.

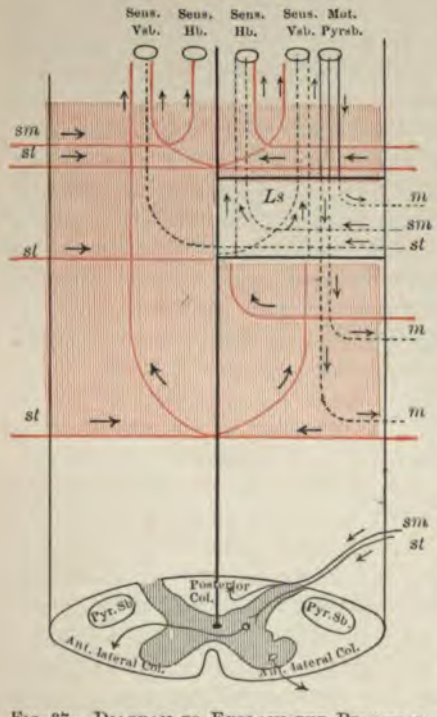


FIG. 37.—DIAGRAM TO EXPLAIN THE PRINCIPAL PHENOMENA IN UNILATERAL LESION.

Ls, Point of lesion, in red the region of increased irritability, ... interrupted ——— intact conduction: *m*, Motor and vaso-motor fibres; *s*, Sensory fibres, viz., *sm* for muscular sense, *st* for tactile sensation, *mot. Pyr = Sb*, Motor pyramidal lateral column tract; *sens. Vsb*, Sensory anterior lateral column tract; *sens. Hb*, Sensory posterior column tract.

Besides these chief alterations in the conduction of the fibres, the following less constant auxiliary symptoms which are apparently paradoxical are noted: *Hyperæsthesia*, respectively hyperalgesia of the motor paralyzed side, a more or less *small zone of anæsthetic area* above the hyperæsthetic region, corresponding to the height of the lesion, and above this a *girdle-like hyperæsthetic zone affecting both sides, disturbances in the evacuation of urine and fæces, ataxia* upon the paralyzed side, as soon as this side is again capable of motion.

The appearance of the small anæsthetic zone at the upper boundary of the region of motor paralysis is easily explained, as, with a unilateral lesion, a segmentary interruption, in keeping with the region of the lesion, occurs both in the sensory (root) fibres which enter and which have not yet crossed and in those coming from the opposite side which cross in the gray substance to the lateral column of the point of lesion. It is more difficult to explain the presence of hyperæsthesia upon the side of the motor paralysis and the narrow hyperæsthetic zone above the anæsthetic region of both sides. This condition may be most readily explained if we assume that the cord above and below the point of lesion is in a condition of increased irritation. The fibres which pass through these parts would then, while passing, become more irritable, which would show itself immediately above the point of lesion, in the cutaneous parts, as bilateral hyperæsthesia, whereas in the parts of the body below the point of lesion only one side (corresponding to the lesion) would be hyperæsthetic, because the contra-lateral side, on account of the reasons described above, must show anæsthesia. The latter is also true of the area of the sensory fibres which enter the irritated parts as they also, in their further course, pass the point of lesion. The diagrams (see Figs. 37 and 38) may somewhat more readily explain the apparently very complicated symptom-complex.

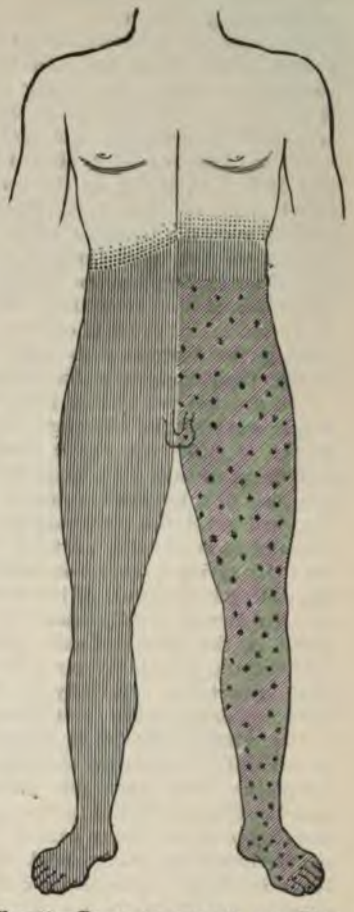


FIG. 38.—DIAGRAM SHOWING THE PRINCIPAL PHENOMENA IN UNILATERAL LESION OF THE DORSAL CORD (LEFT). (After Erb.)

The oblique shading signifies motor and vaso-motor paralysis. The vertical shading signifies cutaneous anæsthesia; the dots signify cutaneous hyperæsthesia.

TUMOURS OF THE SPINAL-CORD CANAL, TUMOURS OF THE SPINAL MARROW AND ITS MENINGES

Among the causes of compression of the spinal cord are also tumours which may develop in the canal of the cord and grow. They form a special group of spinal-cord affections, less so by their clinical picture than by their anatomical importance. The symptoms of value in the diagnosis produced by tumours in the spinal cord must vary greatly in the individual case, according to the distribution and situation of the neoplasm.

Meningeal Tumours.—The diagnosis of those tumours in the canal of the cord, which arise from the meninges, *the meningeal tumours*, present comparatively the

best foundations for diagnosis; the most frequent are those arising from the dura mater, being sarcomata. They cause marked symptoms by producing *pressure* partly upon the *meninges* and partly upon the *nerve roots* which enter and leave these tissues, partly upon the *cord substance*, which cannot escape by deviating in the narrow bony canal, the tumour pressing against it. The first symptoms precede the latter, those of compression of the cord; they consist in irritative phenomena: eccentric pains, paræsthesia and hyperæsthesia, muscular twitchings and contractures, which are followed later, often suddenly, by paraplegia, with atrophy of the muscles and DeR. According as the posterior roots are affected, *anæsthesia* occurs which shows itself as *anæsthesia dolorosa* in those cases in which the pressure of the tumour upon the posterior nerve roots is so marked that irritation from the periphery can no more pass through the principal point of pressure and reach the brain, but, from this region, an irritation may travel towards the centre with eccentric projection of fibre irritation. The compression of the cord itself may affect only a part (*unilateral lesion*), occasionally the entire transverse section of the cord, this causing the symptoms to vary, symptoms which have been previously described, so that their analysis in the individual case does not give rise to difficulty.

Of greatest importance in the diagnosis is always the observation of the gradual development of the affection, the circumstance that irritative root symptoms have existed to which are added the consequences of a transverse section of the spinal cord being more or less completely affected by compression. However, even then the diagnosis of a neoplasm, especially of one arising in the meninges and pressing upon the cord substance, can only be made with certainty in the rarest cases, in that tumours form at other parts of the body, or the symptoms of late syphilis develop, etc.; this giving a certain indication of the nature of the causative agent of compression acting in the canal of the spinal cord. Most frequent are *myxomata* and *sarcomata*, the former usually upon the inner surface of the dura and pia, the latter being found in the cellular tissue between the dura and the spinal column; tubercles, syphilomata, etc., are rarer. Determining the seat of the tumour is of great practical importance, especially on account of a possible operative interference. The rules laid down in the description of myelitis are of use here.

Tumours of the Cauda Equina and Diseases of the Conus Terminalis.—It must be especially emphasized that the *tumours of the cauda equina* which present relatively the most favourable prognosis in regard to surgical interference, produce quite a typical clinical picture: Extraordinarily severe, deep-seated pains in the sacral, and in the lower part of the lumbar vertebral column, in which under some circumstances an enlargement may be noted. Later, paraplegia may occur in the lower extremity (*paraplegia dolorosa*), with degenerative atrophy and DeR of the muscles, *anæsthesia* of the skin of the legs, the hips, the anal and genital regions, which, however, only occurs after irritative phenomena have existed for some time, i. e., intense pains in the small of the back, which may radiate into the bladder, and particularly in the area of distribution of the sciatic nerve (the posterior surface of the thigh, and the external surface of the lower leg and foot), whereas simultaneous motor irritative phenomena are absent. The character of anæsthesia, observed in caudal affections, is that of a complete anæsthesia, all sensory qualities being affected. Besides, bedsores, paralysis of the bladder and rectum, and disturbances of the sexual function occur.

The symptoms described are quite obvious as results of pressure, respectively of disease, of the *cauda equina*, if the position of the latter is considered, being in the lowest portion of the vertebral canal, i. e., that the lumbar and sacral nerve roots, which form the conus terminalis, the final portion of the spinal cord, at the second lumbar vertebra combine to form a bundle and travel downward until they find their exit in the intervertebral foramen, respectively the openings of the sacrum. In the sacral foramen the spinal ganglia which belong to the sensory roots are situated, and beyond this point they combine with the peripheral nerves coming from the anterior roots. In the cauda equina, therefore, the motor and sensory fibres run a course separated from one another, and in the manner that the *sensory* roots form *dorsal groups*, the *motor* being situated *ventrally*. Thus it is easily understood that both varieties of roots are affected one after the other, that the irrita-

tive phenomena arising from the sensory roots, in the form of very severe pains, precede the motor paralyses by some time, and that the sensory as well as the motor roots are affected upon both sides, but at the onset this is more marked upon one side than upon the other.

Conus Affection.—The *affection of the conus terminalis* may be more easily differentiated from cauda affections, and lately greater attention has been paid in a diagnostic respect to diseases of the conus. We owe our ability to diagnosticate these affections to the labours of Schultze, Köster, L. R. Müller, and others. The division of the conus terminalis from the sacral cord is an arbitrary one. According to Raynaud it consists of the three lowest sacral segments; and of the coccygeal segment, and this limitation of the conus is now very generally accepted. If we compare, with the aid of the table upon pages 527 and 528, the functions of individual spinal-cord segments, the three lowest sacral segments (and the two immediately above) would show:

	Muscles	Sensation	Reflexes
First and second sacral segment.	Outward rotators of the thighs (pyriformis, obturator int., etc.), glutæus max., muscles of the calves, tibialis anticus, peroneal musculature.	Posterior side of the upper and lower thighs (1). Outer side of the lower thigh and foot as well as bladder and rectum (2).	Plantar reflex (sacral 1 and 2) Achillo-tendon reflex. (Lumbar 5 and sacral 1). Centre for erection (sacral 2).
Third and fifth sacral segment.	Ischio- and bulbocavernosus (3). Detrusor vesicæ (4). Sphincter ani ext. and levator ani (5).	Skin of the penis, middle portion of the scrotum and urethral mucous membrane (3). Skin of the perinæum and over the sacrum (4), coccyx and anus (5).	Achillo-tendon reflex (3–5 sacral). Centre for ejaculation (3). Bladder and rectal centre. (Sacral segments 4 and 5.)

In a strictly limited disease of the conus terminalis, anæsthesia would affect the skin of the penis, scrotum and perinæum, and the cutaneous covering of the sacrum and coccyx and the rectum. Furthermore, it is characteristic of the affections of the cauda that in *disease of the conus, as a rule, no irritative phenomena precede the anæsthesia* and that it is dissociated, i. e., the sensation for tactile impression may be retained, whereas the pain and the temperature senses may be absent. Also the symmetry of the distribution of the anæsthesia and the motor flaccid paralysis upon both sides is to be expected with greater certainty in conus affections than in disease of the cauda equina, on account of the narrowness of the conus—but these are only relative symptoms and of secondary importance in diagnosis. More important is the fact that the paralyzed muscles, due to the medullary character of the disease in question, show *fibrillary contractions*, and it is still more in favour of a conus affection *if, upon advance of the disease to the upper sacral segments*, the muscles of the lower extremity, corresponding to their nuclear arrangement, are affected by the paralysis, and the reflexes also disappear one after another, therefore not only the bladder and rectal centres and eventually the Achillo-tendon reflexes disappear, but gradually also the plantar and patellar tendon reflexes diminish, after an abnormal increase has preceded their disappearance. The latter condition depends upon the

fact that the next higher segment to the one affected is found to be in a condition of greater irritability. In connection with this is also the phenomenon that, occasionally, above the anæsthetic zone a *hyperæsthetic* area may be met with, a symptom which is *directly* in favour of the medullary seat of the disease. Finally, it must be emphasized that the consecutive disturbances in conus affections usually develop very rapidly, but in caudal affections, if they are due to tumours, always very slowly.

As is obvious, it is possible under some circumstances to make a differential diagnosis between the two affections. This has a certain practical value, in that the diseases of the cauda equina, as has already been mentioned, present favourable conditions for surgical interference, whereas affections of the conus are not susceptible of improvement, nor of a surgical therapy.

Regarding the *nature of conus affections*, in the cases that have been observed up to the present it was a question of an acutely appearing hæmatomyelia or a traumatic myelitis in the lowest portion of the spinal cord, arising after the effect of great force upon the sacral region, such as a fall upon the buttocks, etc. The non-traumatic conus diseases, but apparently due to syphilis, refrigeration, or spontaneously developing, have not been confirmed by autopsy; an instance of a *tumour* limited to the conus has as yet not been observed. If the conus is compressed by a tumour situated in its vicinity, the caudal root fibres are most probably simultaneously affected.

Spinal-Cord Tumours.—In general, *intramedullary tumours* (gliomata, tubercles, syphilomata, etc.) give rise to less distinct symptoms than meningeal tumours. In cases in which tumours of the spinal-cord substance, in their growth, slowly press upon the latter, no damage in function of the nerve fibres, which are simply pressed apart, may appear, the development of the tumour, at least at its onset, may occur without giving rise to symptoms. In other cases symptoms appear that vary greatly according to whether individual columns or the entire gray substance of the cord in its anterior or posterior parts are implicated. In this manner pathological pictures may present themselves which may resemble tabes, lateral sclerosis, poliomyelitis anterior, unilateral lesions of the spinal cord, syringomyelia, or transverse myelitis; in this connection I must refer to the former diagnostic details, especially to those in the chapter on Syringomyelia. With the development of tumours the gradual accumulation of the functional disturbances and their increase from one side or the other are connected. Upon the degree of fulness of the vessels of the tumour and upon the possibility of hæmorrhages, the intensity of the phenomena and the appearance of a sudden aggravation in the pathological picture are dependent; the irritative and compression phenomena are due to the steady growth of the tumour and cannot be absent in the long duration of the process. The *last-named symptoms, due to the nature of the affection, give us at least some points of support for the diagnosis of tumours of the spinal cord*; this, however, rarely rises beyond the region of probability, and, as a rule, is only a probable diagnosis at best.

ACUTE ASCENDING SPINAL PARALYSIS, LANDRY'S PARALYSIS

The disease is characterized in the majority of cases by marked clinical symptoms. Usually it is ushered in by more or less definite *prodromes*: fever, sensory disturbances (drawing pains and paræsthesia), lassitude. After a few days or weeks, paresis of the legs follows, which rapidly terminates in complete paraplegia. The paralysis is *flaccid*, without pain; this is followed by paralysis of the trunk, the muscles of the back and abdomen, with severe disturbances of respiration, especially of forced expiratory movements, and later on paralysis of the arms follows. In the last stage of the disease, bulbar symptoms occur: disturbances in articulation and phonation, difficulty in mastication and deglutition, paralysis of the palate and paresis of the facial nerve, disorder of the muscles of the eye, inequality of the pupils, increased rapidity of the pulse, severe dyspnœa, almost to the point of asphyxia, which in the majority of cases ushers in the lethal termination (occasionally even after two or three days). The medium duration of the affection is

from one to two weeks. In rare cases, instead of an ascending course, a *descending* form of paralysis occurs.

Symptoms of Importance in the Diagnosis.—If a diagnosis of this disease is to be made and a separation from other diseases of the spinal cord and nervous affections, the individual phenomena in the morbid picture just outlined are to be noted. *Sensation* is always decidedly less altered than motion. The patients occasionally complain of paræsthesia of all kinds, rarely, however, of pain; the objective examination in the majority of cases shows *complete normal conditions of sensation.* The spinal column is not sensitive to pressure; hyperæsthesia and anæsthesia, slowing of the temperature sense and pain conduction are only noted in exceptional cases. In marked contrast to this are the *severe disturbances on the part of the motor sphere.* The paralyses do not show the slightest spastic character, they are flaccid; *in the paralyzed muscles, apparently principally on account of the rapid course of the disease, no atrophy is noted, and as a rule, no alterations in their electric contractility.* The condition of the *reflexes*, the cutaneous and tendon reflexes, varies; in general they are at least retained at the onset, later they may disappear. The *sphincters* are almost always normal; vaso-motor disturbances are only exceptionally present, especially are *bedsores* absent; marked sweating has been variously observed. Ataxia is merely indicated, the *cerebral functions* are uniformly intact. By way of illustration, a case of my own with a typical course and negative autopsy findings will be related:

Case of Landry's Paralysis.—A day labourer, female, aged twenty-six (entered the hospital August 1, 1891, died August 8, 1891), no hereditary affection, in childhood had measles, as a school-girl, pulmonary inflammation, one year ago, erysipelas. Her present affection began with *headache, vomiting, pains in the small of the back and in the legs*; appetite normal, bowels regular, slight cough, no cardiac palpitation.

The physical examination revealed normal condition of the lungs, of the heart and of the abdominal organs; the *spleen is not enlarged*, albumin and sugar not present in the urine. The skin shows no exanthem nor œdema, but petechiæ are present which may, however, be due to pediculi; strong bodily frame. Marked spontaneous pain along the vertebral column, also in the thighs which are painful upon pressure. *The pains in the vertebral column in the sitting posture are so intense that the patient cries out aloud, whereas percussion of the vertebral column does not produce pain.* Pulse 100, *no fever.*

August 5.—The legs are somewhat stiff; walking is only possible by small, stamping strides, *tendon reflexes still retained*, in lying down it is impossible to draw the legs up; left facial nerve apparently somewhat paretic. Pulse markedly increased, 150; its frequency remains between 130 and 150 up to the time of death.

August 6.—Sensibility completely intact: Tactile, temperature, pain senses and muscular sense unimpaired; the *patella tendon reflexes to-day are absent.* The paresis in the legs has markedly increased since yesterday; *pressure of the left hand is conspicuously weak.* Voluntary sitting up is impossible, cough is without force, reaction of the pupils prompt.

August 7.—Besides the left-sided facial paralysis which is now distinct, right-sided paresis of the facial nerve is also noted, lagophthalmus is present; uvula in the median line, the tongue does not deviate to either side; movement of the eyes intact, *disturbances in deglutition and speech.* To-day the *right arm is also paralyzed, upon lifting it, it falls flaccidly.* Pressure of the hand on both sides without power; *the legs* excepting slight movement in the ankle joint *completely immovable*, conspicuously cyanotic. *Electrical contractility* of the paralyzed muscle shows: normal conditions upon faradization and also to the constant current, only in the right extensor digitorum communis the ACC is greater than the CaCC.

Nourishment is only possible with the tube or by rectum. Ordered: Unguent. ciner. 4, kali iodid., 3, *pro die.*

August 8, apparently slight improvement in the general condition; but several attacks of suffocation; death occurs in one of these. Up to the end of the illness no fever was present, no disturbance of consciousness; paralysis of the sphincters only in the last days.

Autopsy on August 8, 1891, findings: oophoritis et salpingitis, suppurativa duplex, atheroma aortæ, pneumonia lobularis fibrinosa sinistra, pleuritis adhæsivæ duplex. *Examination of the spinal cord shows normal condition of the membranes and of the cord substance*; the brain also shows itself normal, the pia smooth, no œdema.

The examination, in the Pathologico-Anatomical Institute, of the peripheral nerves and of the sections of the cord which were placed in Müller's fluid gave a *negative result.*

If we review the principal pathologico-anatomical findings which have up to the present been noted in the disease in question, it is seen that the *motor elements* of the nervous system, especially the peripheral neurons, are affected. Only in very rare, exceptional cases, to which also the case just related belongs, does the examination of the nervous system give a purely negative result. Occasionally the peripheral nerves are found affected alone as a peracute polyneuritis, at other times a beginning myelitis (in comparison to the severe clinical phenomena, unimportant changes of the spinal cord, such as swelling of the axis cylinders and degeneration of the anterior horn cells), at other times again, a myelitis combined with secondary degeneration of the peripheral nerves is observed.

Nature of the Affection.—As is obvious, in Landry's paralysis we are dealing with a morbid process showing various localizations in the nervous system, especially in its motor portions. The observations up to this time point to the fact that toxines cause the disease, partly those of the well-known *infectious diseases* (enteric fever, variola, *syphilis*, etc.), partly a *peculiar* infectious virus the nature of which however is still unknown.

The diagnosis of acute ascending paralysis, which now has a sharply defined *symptom-complex*, is in general easy, and may be made with certainty. If the presence of the affection has been determined as certain, the diagnosis now has for its object the detection of the *ætiological factor in each individual case.* This is, however, the realm in which the main difficulties of the diagnosis are encountered. If the history in the affected case develops no points of support for the assumption that a previous infectious disease has exerted its toxic action in the form of a paralysis, it may be assumed that a peculiar infectious disease is present, showing itself by an acute ascending paralysis. There is a certain justification for this, in that, in some cases, without there being a proof of any known infectious disease having preceded the condition, the well-developed picture of a typical infectious disease was present, and at the post mortem there were found enlargement of the spleen, swelling of the mesenteric glands, etc.

MULTIPLE FOCAL AFFECTIONS OF THE SPINAL CORD (AND BRAIN)—ACUTE DISSEMINATED MYELITIS MULTIPLE MYELITIS

The disease is rare, characterized by disseminated, occasionally of smaller, sometimes of larger, inflammatory areas in the spinal cord, also simultaneously appearing in the medulla oblongata, in the pons, and in the hemispheres; in the main, however, the spinal cord is affected. The clinical picture may resemble *acute ataxia* or *paraplegia.*

In the former case, well-developed *ataxia of the upper and lower extremities* is present, in which however the ataxia of the legs, as in the case of tabes dorsalis, is not prominent; simultaneously there is present a slowness of movement and slight paresis. With this, as in the case of multiple sclerosis, *nystagmus* and *intention tremor* are noted, which may also show themselves in the head and in the tongue; especially conspicuous are the *disturbances of speech* appearing as monotonia and scanning speech. On the part of the cranial nerve changes may occur: Difficulty in hearing, optic neuritis, etc.; also the intelligence and the frame of mind of the patient is frequently altered. Sensation is normal or nearly so, as well as the functions of the bladder and rectum; the reflexes are unchanged, eventually the tendon reflexes may be increased. The disease begins acutely, runs its course in paroxysms

and may, as it appears, even after a long time give rise to relapses, in that recovery has apparently resulted, or it may terminate as a multiple sclerosis.

If the affection runs its course as a *paraplegia*, the principal symptom, which may often begin unilaterally, is the paralysis of the leg (occasionally of an atrophic, sometimes of a spastic character), to which there is added later paralysis of the arms and trunk, and eventually bulbar symptoms, bedsores as well as paralysis of the bladder and rectum.

As is obvious, the first clinical picture shows the characters of multiple sclerosis, the second, that of a diffused myelitis, and the differentiation of these diseases is always difficult. In favour of the presence of disseminated myelitis is the *acute character of the ataxia* and above all the ætiology of the individual case, in that disseminated myelitis, as is well known, arises after trauma, especially however after acute infectious diseases: Enteric fever, variola, erysipelas, influenza, tuberculosis and others, and follows certain intoxications. Of differentio-diagnostic importance for the *paraplegic form* of the disease is primarily and perhaps exclusively the unilateral onset of the paralysis.

If recovery does not take place, the disease may gradually terminate in multiple sclerosis. The following chapter will be devoted to a consideration of this affection.

CHRONIC DISSEMINATED MYELITIS, SCLEROSIS CEREBROSPINALIS MULTIPLEX, MULTIPLE SCLEROSIS OF THE BRAIN AND SPINAL CORD

Anatomical Changes.—The disease is characterized by a "*multiple*" development of irregularly disseminated, more or less numerous foci in the brain and especially in the spinal cord (especially in the cervical part), which in opposition to the tissue of the central nervous system which has remained normal, stand out like small islands ("*insular sclerosis*"), consisting of proliferating glia tissue, in which beside the degenerated nerve elements, there are regularly found well-preserved axis cylinders which have only been denuded of their myelin sheath. Secondary degenerations develop to a conspicuously slight degree, evidently because the relatively narrowly limited foci have not completely destroyed the tracts which functionally belong together, and that they are not progressively isolately affected. Besides, as has already been mentioned, in the foci the axis cylinders almost always remain at least partly intact. In the peripheral nervous system also sclerotic changes are found. Besides the disseminated small foci, occasionally a larger focus or diffuse disease of the central nervous system is noted.

In keeping with the irregular dissemination of the sclerotic foci in the nervous system, *there can be no question of a uniformly sharply defined clinical picture*. On the contrary, the affection is especially characterized by its protean character, and according to the localization of the foci, by the absence of this, and at another time of that, functional disturbance. In the majority of cases, however, the disease appears in such a uniform clinical picture that the diagnosis is possible, i. e., as the autopsies prove, the diagnosis has been made correctly.

Diagnostically Important Symptoms.—The most constantly observed symptom is the "*intention tremor*"—a tremor of the body, especially of the upper extremities, which becomes more marked or usually only arises when the patient attempts muscular movements or intends to do so; the tremor also causes disturbances in writing, in the gait, etc. Besides the tremor of the extremities, there are also oscillations of the head which may even be noted when the head is kept quiet, probably because the muscles of the neck and nape are in action, this giving rise to the tremor. Be-

sides this, the movements of the patient show themselves as *ataxic,* to which fact Strümpell lately called attention, and in his opinion the "intention tremor" is also in many cases connected with the ataxia. The motor power of the muscles, at least in the early stages of the disease, is as a rule maintained, so that well-developed paralyses are not noted later; however, a *stiffness of the muscles,* especially during movement, occurs, which later leads to contractures. Well-developed paralyses are rare, quite common, however, are pareses which as the result of the almost constant *increase of the tendon reflexes show a spastic character,* manifesting itself in the gait of the patient in a typical manner. The electrical contractility of the paretic parts is almost always normal, only exceptionally are there indications of atrophic paralysis with DeR. The *cause of the tremor* in the disease in question, which is almost a pathognomonic symptom of multiple sclerosis, is difficult to explain. Perhaps it is frequently, as already mentioned, an ataxic symptom, and in keeping with the relatively small foci would give rise to insignificant *disturbances in the conduction of the co-ordinative tracts* (in the wider meaning of our explanation). Besides the tremor, in a certain number of cases well-developed *ataxia* occurs (in about one half of the cases); especially frequent is a clonic spasm of the muscles of the eye, with associated movements in different directions—*nystagmus*—in the form of pendular movements of the eye.

Ophthalmoscopic Findings.—With this, there are found, rarely, but then well-developed, *paralyses of the ocular muscles* (most frequently paresis of the abducens), more rarely however disturbance in the pupillary reaction or alteration in the width of the pupil, frequently there is a disturbance in the motility of the iris, *rhythmic oscillation,* in the form of a rapidly alternating contraction and dilatation of the pupil.

Regarding the *ophthalmoscopic findings,* which, at least in one half of the cases, show pathological disturbances as well as *functional alterations* in the optic nerve, the most important points have been given under the differential diagnosis from tabes.

Speech Disturbance.—A no less important symptom in the affection than the trembling is the *alteration of speech* of the patient. Speech is *slow,* as a rule *scanning,* later becoming lalling, as a result of the impossibility of the patient distinctly to pronounce certain letters. The cause of this disturbance of speech is probably due to a difficulty in innervation and conduction, partly influencing the tracts of articulation, and partly producing a slight tiredness of the muscles used in speech. Especially characteristic is the *monotony* of speech: Modulation is absent from the voice, i. e., the rapid change, as well in the height of the sound as in the accentuation of individual syllables. The reason for the last-named conspicuous change of speech is due to an insufficient innervation of the vocal cords, especially to the incapacity of bringing them into sufficient tension, allowing vivid, accentuated speech. Upon intonation, a trembling of the vocal cords occurs, so that the vowels are uttered with a tremolo. To the incapability rapidly and firmly to innervate the posterior cricoarytænoid muscles a frequently observed phenomenon is due, that is *exulting inspirations* which interrupt the transitory loud laughter. They are

due to the fact that the inspiratory stream which follows the expiratory movements in laughing meets the vocal bands which have not yet separated, setting them into vibrations which produce a tone. In some cases, as Oppenheim has observed, the laughter of the patients is not the result of cheerful impressions, but the product of spasmodic involuntary contractions of the muscles of laughter.

The *sensory disturbances* in the clinical picture are certainly less conspicuous than those of the motor sphere, but upon careful investigation are rarely absent; especially are paræsthesias and transitory anæsthesias of the hands and feet present. Just so are there rarely absent slight disturbances of the sphincters of the bladder and rectum; also morbid alterations of the sexual functions arise, upon the whole, however, they are not frequent, consisting in sexual weakness, or excessive irritability of the genitalia. *Trophic disturbances* are occasionally noted: Exanthems, arthritic swelling, etc.

Cerebral Symptoms.—As the foci, in the great majority of cases, are not limited to the spinal cord and the medulla oblongata, but are also widely distributed in the brain, it is natural that well-developed *cerebral* symptoms may also be present. The patients frequently complain of *vertigo* which usually occurs in paroxysms, in the form of "rotary vertigo"; further in many cases there are *apoplectiform* (more rarely *epileptiform*) attacks. After slight prodromes (headache, vertigo, etc.) or also without, with an increase in temperature and in the frequency of the pulse, there develops gradually loss of consciousness and hemiplegia, which latter may be retarded, but usually, after a brief existence, again completely disappears. Not only hemiplegia, but also paraplegia, hemianæsthesia and paralyses of individual cranial nerves may develop with the apoplectiform attacks. These insults in connection with the paralyses are probably the result of small fresh inflammatory areas in the course of the disease. In a certain number of cases the *psychical* condition is altered, varying in form and intensity: There are conditions of depression, partly also of exaltation, in other patients there is unmotived laughing and crying, loss of memory, imbecility, or actual dementia. As an anatomical substratum of the last-named clinical phenomena, besides the insular sclerosis there is found diffuse sclerosis in the brain.

If changes arise in the *peripheral* nerves, especially in the *cranial* nerves: The optic nerve, the nerves of the muscles of the eye, the trigeminus, acusticus, etc., this develops symptoms of paralysis on the part of the affected nerves, giving rise to amblyopia, ptosis diplopia, ageusia, difficulty in hearing, etc.

Diagnostic Value of the Individual Symptoms.—In an affection, the special anatomical character of which depends upon an irregular distribution of small foci in the central nervous system and in the peripheral nerves, it is obvious that the phenomena of the disease in individual cases cannot be quite the same, but that according to the seat of the focus an irregular, variable, clinical picture must result. If cognizance is only taken of one or the other symptom in the diagnosis and, in the absence of the same, at once the probability of multiple sclerosis is denied, it would

be very precarious. On the other hand, it might appear as if it were an exceedingly difficult undertaking to diagnosticate with certainty a disease which, according to the nature of its manifold clinical pictures, varies so much, and to separate it from other affections of the nervous system with a certain localization of the morbid process—in cases in which the sclerosis accidentally implicates the same territory as other diseases. In fact, the clinical picture of sclerosis is occasionally identical with that of transverse and multiple myelitis, or, if sclerosis occurs with vertigo and headache, an apopletic attack and hemiplegia follows, it may resemble a brain tumour, syphilis of the brain, embolism or cerebral hæmorrhage. If the sclerosis is exclusively concentrated to but few individual portions of the spinal cord, the symptom-complex may simulate a "system disease." If, for example, especially the lateral tracts and anterior columns are affected by multiple sclerosis, the clinical picture may be identical with that of amyotrophic lateral sclerosis. Similar conditions are also true of the localization in the posterior column—in which the clinical picture resembling tabes, pains, ataxia, etc., also is present—or in which the localization occurs in the pons or medulla oblongata—the disease then running a course simulating chronic bulbar paralysis. Multiple sclerosis may also set in with atrophy of the muscles of the hand, and in this stage may simulate progressive muscular atrophy.

Special Diagnosis; Differential Diagnosis.—In spite of this, the diagnosis of multiple sclerosis in the greatest majority of cases may be made with certainty. As soon as the usual *ensemble* of phenomena is present: *Intention tremor, spastic paresis, nystagmus,* and (eventually partial) *optic atrophy,* the peculiar *disturbance of speech,* the secondary importance of the alterations of sensation compared with those in the motor sphere, the *vertigo,* the *apoplectiform attacks* and finally the signs of *psychical alienation and weakness,* this permits us to make a diagnosis of cerebro-spinal multiple sclerosis with certainty. The diagnosis is more difficult if the various foci are exclusively isolated and limited to certain portions of the spinal cord, resembling lateral sclerosis or progressive muscular atrophy, etc., and the typical symptoms: The intention tremor, nystagmus, etc., are absent, or but slightly indicated. Here the differential diagnosis is aided by the observation of the circumstance that, in keeping with the irregular distribution of small foci, besides the symptoms of the affection, individual parts of the central nervous system, upon close examination, show secondary symptoms, which do not belong to the typical picture of the system disease, for example, to lateral sclerosis. Besides, the cases of pure spinal-cord sclerosis are quite rare, there is usually found with the spinal-cord symptoms, almost without exception, this or that sign of an affection of the brain: Vertigo, disturbance of intelligence, etc., however, it must not be forgotten that monoplegia and hemiplegia dare not be regarded as cerebral symptoms, but that they may appear as a result of a unilateral development of the foci in the cord substance. In some cases, besides the weakness in movement, the tremor and disturbance in speech, the psychical alterations are so prominent that the affection resembles dementia paralytica.

The exact observation of the *onset* and *course* of the affection also may, under some circumstances, furnish the key to the diagnostician for the recognition of the frequently observed affection. The disease may follow, as has been proven with certainty, trauma, especially after a severe concussion of the body, further, after severe refrigeration and infectious diseases; occasionally we are able to trace back the onset of the affection to the earliest childhood of the patient. In such cases, from youth on, there existed weakness of the legs, a slight tremor or else some other nervous symptom which was easily overlooked until later, when more prominent phenomena appeared. In other cases the affection is manifested by insignificant symptoms: Vertigo, or optic neuritis, to which only later disturbance in speech and nystagmus are added. *Characteristic of multiple sclerosis above all is the propagation of the process,* which is rarely a creeping, progressive one, but rather usually increasing by attacks, in the form of sudden aggravations and apoplectic insults. The pathological phenomena which arise thereby, as a rule, improve, to reappear in fresh attacks, giving place to permanent disturbances. Briefly, the change in intensity of the symptoms, the intercurrent improvement and aggravation, give the disease a peculiar aspect. In this connection, sclerosis resembles *hysteria,* with which it has also in common the irregularity of the symptoms in general. Confusion of both diseases is, therefore, possible; in favour of hysteria are the anæsthesia, the spasm, the results of suggestive influence on the morbid phenomena, and the negative anatomical findings in the optic nerve.

Differential Diagnosis from Paralysis Agitans, etc.—If the *tremor* dominates the situation, multiple sclerosis may be mistaken for other forms of tremor, especially for *paralysis agitans.* However, this is only possible upon superficial examination; apart from the fact that paralysis agitans almost always occurs in elderly persons, multiple sclerosis affecting younger individuals, and in its primary stage may reach back to earliest childhood, the character of the tremor is entirely different. In paralysis agitans there are uniform oscillations, especially in the hands and arms (in which the movements of the affected parts are involuntary); these are absent in the head, further, are found especially during rest, and are not increased by active movement, on the contrary, they frequently cease—altogether in marked contrast to the tremor in sclerosis, which desists in rest, is produced by voluntary movement, and especially affects the head. Besides, tremor is only one of the characteristic symptoms of paralysis agitans. The muscular rigidity (without increased tendon reflexes) "propulsion" and "retropulsion" and the peculiar condition in which the body is held in this affection, are of similar importance in the diagnosis. On the other hand, if sclerosis is present, upon careful investigation usually other symptoms, which have been described, are present: Nystagmus, disturbance of speech, etc. From other forms of tremor, that variety occurring in sclerosis, under some circumstances, cannot be differentiated. *Mercurial tremor,* according to my experience, presents itself occasionally exactly like the tremor of sclerosis, as an exquisite intention tremor which may increase to severe shaking spasm. The two affections, however, cannot be confounded,

if we are not too superficial in our examination, i. e., not only observing one symptom, the trembling, but simultaneously also considering the erethism, the general pathological condition, etc., and above all also the ætiology.

Cases with Negative Anatomical Findings.—It must also be mentioned, finally, that in rare cases, in which during the life of the patient the symptoms of multiple sclerosis were more or less completely developed, post mortem no demonstrable anatomical changes in the nervous system are found, or a combination of disease of the spinal cord and brain is met with, not conforming to the usual type of multiple sclerosis but representing a more diffuse sclerosis, and showing, besides, marked meningeal changes. In the latter case we speak of an *atypical sclerosis,* in the former of a *pseudo-sclerosis,* i. e., a neurosis running its course resembling multiple sclerosis, which, according to experience, may follow an infectious disease.

ANÆMIA, HYPERÆMIA OF THE SPINAL CORD, SPINAL-CORD HŒMORRHAGE, MYELOMALACIA

By way of supplement the *circulatory disturbances* of the spinal cord shall be briefly described.

Anæmia of the Spinal Cord.—There is no doubt, as we know for two hundred years, that a sudden compression of the abdominal aorta in a short time may give rise to paralysis of the lower extremity, respectively of the hind legs, as experiments have shown (Stensen's experiment). This paralysis must, as Schiffer has shown, be due to a sudden *anæmia* of the lumbar cord. If we have reason to suspect that an apoplectiform paralysis of the lower extremities has been preceded by thrombosis or embolism of the abdominal aorta (disappearance of the crural pulse), we may explain the sudden appearance of paraplegia, loss of sensation and paralysis of the sphincters, in the meaning of the experiment just described. But how very rarely will such a diagnosis be justified! Pareses, anæsthesia, paræsthesia, disturbances of reflex stimulation, etc., phenomena which are observed in anæmic individuals, often are referred to anæmia of the spinal cord, especially if improvement has followed the use of iron. Such assumptions are not diagnoses, and it is not worth while to consider them further.

Hyperæmia of the Spinal Cord.—The same is true of the diagnosis of *hyperæmia of the spinal cord,* the result of congestion and engorgement. The symptoms supposed to be due to hyperæmia: Lancinating pains in the periphery, paræsthesia, anæsthesia, contractions, pareses of the extremities, etc., are very questionable as the result of circulatory changes in the spinal cord, even if these symptoms are of a transitory character. In the latter case, we may think of circulatory disturbances in the spinal cord as hyperæmic; there can however be no question of an actual diagnosis.

Hæmorrhage into the Cord.—The diagnosis of *hæmorrhage of the cord, of "spinal apoplexy," hæmatomyelia,* rests upon a better foundation. The presence of blood, by the bursting of a blood vessel of the spinal cord, may affect the longitudinal or transverse substance of the spinal cord ("tubal hæmorrhage"), especially in the gray substance, being distributed there, but on account of the narrow space it may only show very limited dimensions. The immediate results are *sudden paralyses of the extremities,* which usually affect a transverse section, more rarely giving rise to a unilateral lesion, resembling syringomyelia, or an affection of the conus. This may produce, possibly due to the sudden tension in the membranes of the spinal cord or to compression of the posterior roots, marked pains in the vertebral column and in the periphery. The other symptoms: disturbances of sensation, the conditions

of the sphincters and reflexes, the succeeding muscular atrophy, etc., depend upon the seat and distribution of the hæmorrhage, and require no further explanation. If the hæmorrhage arises in portions of the cord which are close to the medulla oblongata, sudden death may occur, due to a lesion, respectively compression, of the respiratory centre.

The diagnosis of spinal apoplexy should only be made with the greatest reserve. The sudden onset of the disease, the apoplectiform appearance of paralysis—the most important support for the diagnosis—are also noted in other affections of the cord, especially *now and then* in acute myelitis. Only when certain *ætiological* conditions simultaneously are in favour of the diagnosis of spinal-cord hæmorrhage: a fall, a severe blow upon the back, fracture of a vertebra, the existence of a marked hæmorrhagic diathesis or of atheroma, a little more certainty in the diagnosis is permissible. It is better not to attempt to make a differential diagnosis between meningeal and spinal hæmorrhage, even if the prominence and continuance of irritative phenomena, stiffness and pain of the vertebral column, incompleteness of the paralysis of the motor and sensory nerves, are more in favour of meningeal apoplexy. To determine the *seat* of the hæmorrhage in the spinal cord in the individual case is rarely difficult.

Myelomalacia.—*Myelomalacia*, due to embolism or thrombosis, occasionally gives rise to phenomena which resemble a circumscribed myelitis, when larger necrotic areas the result of occlusion of the vessels have occurred (capillary emboli may appear without giving rise to symptoms). This disease therefore cannot be differentiated from myelitis. The diagnosis, however, may be made with a certain degree of probability, if, as the result of endocarditis or in the course of marantic conditions, symptoms of a rapidly developing myelitis occur, in which it can be shown that coagula have been dragged into the circulation, and no other causes can be determined for the appearance of these symptoms.

DIAGNOSIS OF DISEASES OF THE MEDULLA OBLONGATA AND THE PONS

WHEREAS the anatomical and physiological differences of the spinal cord are relatively simple, the structure and function of the posterior parts of the brain, medulla oblongata and pons, are much more complicated. Anatomical investigation finds great difficulties here, and still greater ones are encountered by physiological research. It is, therefore, not strange that the explanation of pathological phenomena due to diseases of the oblongata and of the pons is still without the required certainty. With the following exposition of the anatomico-physiological conditions of this portion of the central nervous system we shall attempt to give a brief review of what is considered in this chapter as demonstrated or, at least, likely in this respect.

ANATOMICO-PHYSIOLOGICAL INTRODUCTION

Anatomical Structure of the Medulla Oblongata.—The structure of the transverse section of the spinal cord disappears more and more at the upper end of the cord in that the central canal is gradually forced back, to pass, about at the middle of the medulla oblongata, into the sinus rhomboideus, in that, further, the individual component parts of the transverse section of the cord, which up to this had retained their regular order, assume a different form, and that new structures arise in the medulla oblongata (the olives, the cerebral nerve nuclei, etc.).

Structural Conditions at the Height of the Pyramidal Decussation—Pyramidal Decussation.—If we proceed from below upward, the *pyramidal lateral column fibres* at the boundary between the spinal cord and medulla oblongata pass from the lateral columns over into the anterior columns of the opposite side (*pyramidal decussation*), and associate themselves with those fibres of the pyramidal anterior column which cross in the spinal cord. Both together therefore form, above the point of decussation, a combined pyramidal column upon the side opposite to their peripheral area of distribution, the further course of which, through the pons, cerebral peduncles, internal capsule, etc., may be comparatively easily followed. By this shifting of the fibres the remainder of the anterior column (the *anterior column ground bundles*) are forced more posteriorly. In the *lateral column*, at the lower end of the medulla oblongata, a new formation appears (gray, rich in ganglion cells, greatly plicated)—the (lower) *olive*, which increases in size at its upper boundary. In the *posterior columns*, finally, at the height of the pyramidal decussation, a marked change occurs: In Goll's column and in the funiculus cuneatus, gray nuclei, which combine with the posterior horn, appear (*nucleus funiculi gracilis et funiculi cuneati*), with which probably the *sensory fibres* which ascend in the spinal cord, without having crossed, are in contact, to radiate from here between the olives

through the fibræ arciformes internæ to the opposite side (*lemniscular decussation*, situated above the pyramidal decussation), forming here the basis of the so-called *lemniscular stratum* (olivary intermediary layer). These, undoubtedly, are also entered by the sensory fibres which have crossed in the spinal cord and which ascend in the ground bundles of the anterior and lateral columns, so that now the *entire sensory fibres* unitedly ascend upon that half of the body which is opposite to their peripheral course, to the middle brain, especially to the lemniscus. As the pyramidal decussation area serves the motor fibres, that which is situated dorsally from the lemniscular area of decussation accordingly serves as the main area of deposit for the sensory fibres. The following diagrams, after Edinger (Figs. 39 and 40), show the described conditions in a clear manner.

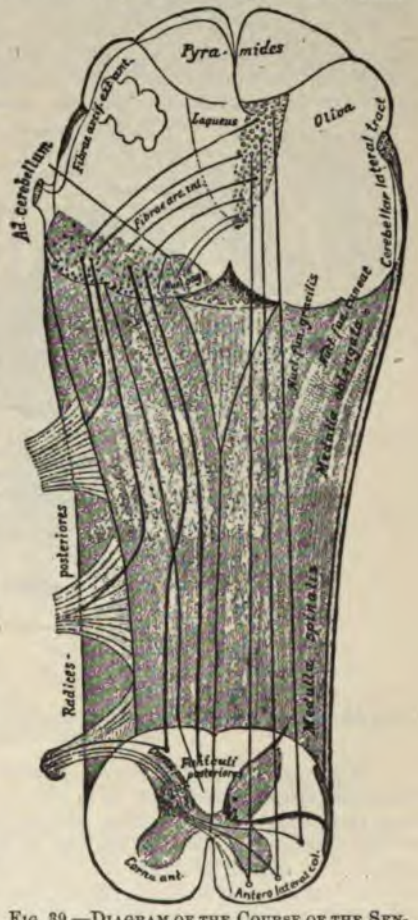


FIG. 39.—DIAGRAM OF THE COURSE OF THE SENSORY TRACT FROM THE POSTERIOR ROOTS TO THE CORD. (After Edinger.)

Transformation of the Gray Substance.—The gray substance of the spinal cord meanwhile has also materially changed in form and position. Even in the uppermost portions of the spinal cord, the anterior horns differ, more and more, from the *lateral horns*, giving origin to the root fibres of a cranial nerve, the *spinal accessory nerve*. Owing to the fact that the *nuclei of the posterior columns*, which appear in the lowest part of the medulla oblongata, coalesce with the posterior parts of the gray substance, the latter assumes a new form, differing from that which it possessed in the spinal cord, and as, farther on, the posterior columns incline more and more anteriorly and somewhat laterally, containing less and less fibres, the central gray substance and with it the central canal recede to the posterior surface of the spinal cord; finally, only the slightest layer of gray substance is found between the central canal and the free upper surface.

Fourth Ventricle.—The latter widens more and becomes the *fourth ventricle*. The *roof* of the fourth ventricle is formed by the cerebellum, from which a thin leaflet covering the sinus rhomboideus, representing the combination between this and the spinal cord, is given off posteriorly (*velum medullare posticum*), and anteriorly the *velum medullare anticum*, which, expanding between the cerebellar peduncles, as a roof forms the connection between the middle and posterior brain. In the medial portion of the velum medullare posticum, before the plexus chorioideus of the fourth ventricle which presents itself as an addition to the velum, a large opening is found, the *foramen Magendii*, forming a communication between the cavities of the brain and the subarachnoid space and allowing the forcing of cerebro-spinal fluid, in alterations of the pressure, within the cerebro-spinal canal cavity.

Situation of the Cranial Nerve Nuclei.—In the floor of the fourth ventricle, formed by the gray substance of the spinal cord, which has become flattened and thinner, the ganglion-cell collections are found which form the *cranial nerve nuclei*, in so far as they are not situated below the medulla oblongata and the pons (nuclei of the spinal

accessory nerves) or above the same (nuclei of the oculo-motor in the region of the corpora quadrigemina at the floor of the aqueduct, as well as the nucleus of the trochlear, somewhat below therefrom). The relations of the nuclei of the different cerebral nerves to the peripheral fibres and to the central distribution of the fibres of these nerves, after the general laws of neuron chain communication, have only lately become somewhat more clear; the result of the investigations relating to this subject shall, at least briefly, be referred to here:

The *nuclei of the hypoglossal* nerve appear in the medulla oblongata as ganglion cell masses in the region of the remains of the anterior horns. From these cells the peripheral fibres originate, which, combined into bundles, pass out at the height of the lower olive, laterally from the pyramids. The individual ganglion cells are

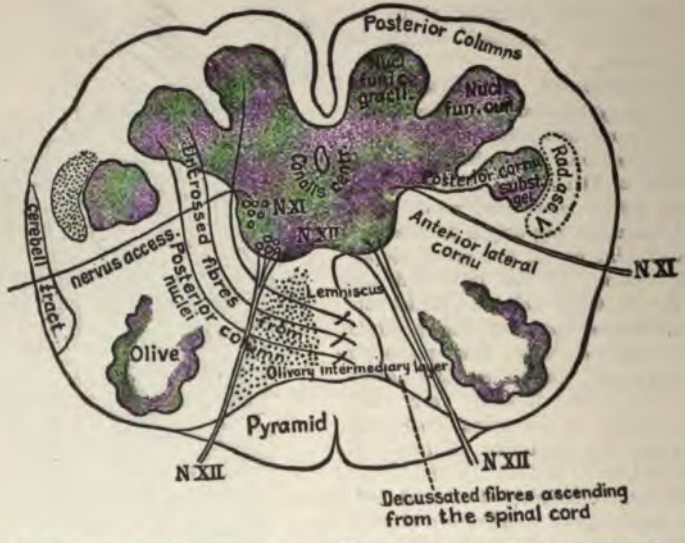


FIG. 40.—TRANSVERSE SECTION THROUGH THE MEDULLA AT THE HEIGHT OF THE ROOTS OF THE HYPOGLOSSUS NERVE. (After Edinger.)

combined among themselves by a fine network of nerves; commissural fibres, also, have been found between both nuclei of the hypoglossus, so that the combined action of the hypoglossal fibres in the act of deglutition and in speaking is anatomically plain. The central neurons, i. e., the corticonuclear tracts of the hypoglossus arise in the central convolutions of the cortex of the brain, farther on they traverse through the internal capsule and the foot of the cerebral peduncle, probably remaining between the tract of the facialis and the extremity tract downward to the pons, forming in the lower portion of the latter the most medial part of the pyramidal tract. Then the fibres decussate in the rhaphe and ventrally approach the nucleus of the hypoglossus; fibres extend directly to the latter from the dorsal longitudinal bundle (compare Fig. 41). Besides these fibres, sensory collaterals from the vagus, glossopharyngeal and trigeminus are in contact with the hypoglossal cells and may be looked upon as reflex fibres.

The nucleus of the *spinal accessory* nerve, on account of its position, represents a series of motor nerve cells, which are found in the lateral dorsal part of the anterior horn, respectively of the lateral horn (Fig. 40) of almost the entire cervical cord (from the first to the fifth cervical nerve). The root fibres arising in these cells enter the lateral column after they have run a short distance in the gray matter, towards the brain, then, leaving the spinal cord, to travel upward, so that the trunk which is formed from them, lies above the trunk of the hypoglossus.

Similarly situated is the *motor nucleus of the vagus*; only, as it is situated higher up than the nucleus of the spinal accessory nerve, the gray matter, rich in cells, which surrounds it, is almost entirely separated in a ventral direction from the remaining gray substance of the horns ("ventral" motor nucleus; nucleus ambiguus). Its neurites, which go off dorsally in arcs (compare Fig. 41), combine with the fibres of another nucleus of the vagus, the *sensory nucleus*, which lies somewhat more dorsally, and which, as it at the same time forms the end nucleus of a part of the glosspharyngeal fibres, is designated as *dorsal glossopharyngeal nucleus*. Besides, the vagus fibres are in connection with a third cell system, the *solitary bun-*

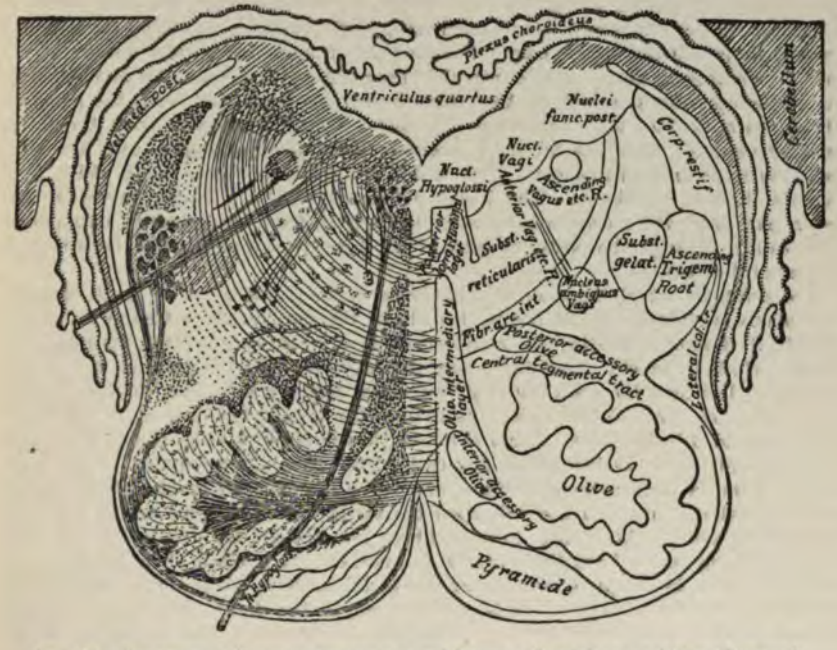


FIG. 41.—TRANSVERSE SECTION THROUGH THE MEDULLA OBLONGATA. (After Edinger.)

dles (spinal glossopharyngeal root), which consist of fine, caudally travelling nerve fibres, joined into a bundle before they approach the ganglion cells, and also of an admixture of gray matter, and which extend from the upper cervical cord to the medulla oblongata. Vagus fibres spread around the cells of gray matter in this column, whereas, on the other hand, the cells probably give off fibres, which, crossing in the rhaphe, enter the contralateral lemniscus and travel farther towards the cerebrum. If we remember that the sensory fibres of the vagus and also those of the glossopharyngeal nerve arise from the cells of their root ganglia, we note, as in the case of the sensory nerves of the spinal cord which arise in the spinal ganglia, the well-known structure of the sensory neurons, namely, peripheral neuron, peripheral nerve, original nucleus, root—sensory terminal nucleus (sensory vagus nucleus), neurite of the secondary sensory root crossing in the rhaphe and ascending to the cerebrum; nothing further is known regarding the central course of the vagus.

Exactly the same conditions are found regarding the *glossopharyngeus*, the roots and nucleus of which cannot be sharply isolated in the internal parts of the medulla oblongata, as is well known. It is probable that the fibres which terminate in the fasciculus solitarius are for the most part glossopharyngeal fibres, *the gray substance of which, therefore, is the principal end nucleus of the glossopharyngeus* (*nucleus fasciculi solitarii*). Individual fibres of the trigeminus, especially the

gustatory fibres of the lingual nerve (see Fig. 13), also enter the solitary fasciculus and its nuclear column.

The relations of the intrabulbar course of the *acusticus* have been well investigated lately, especially those of the cochlear nerve, the actual nerve of hearing. It arises in the spiral ganglion of the cochlea, and the termination of the root fibres (the posterior root of the acusticus) is in the ventral (lateral) nucleus of the acusticus, which is situated with the tuberculum acusticum ventro-laterally from the restiform body, at the caudal border of the pons. The peripheral acusticus neuron terminates in this nucleus, and a new neuron begins, the neurites of which partly encircle the restiform body laterally (as striæ acusticæ at the floor of the fourth ventricle entering deeply near the rhaphe and decussating), and then travel in an arch to the contralateral lower lemniscus, partly extend medially, forming the corporus trapezoideum which passes transversely through the caudal border of the pons. The trapezoid fibres radiate into the upper olive of the same side and also into that of the opposite side, terminating there by coming in contact with the ganglion cells of the olive (secondary acusticus neuron). The fibres arising from the latter (tertiary neuron) enter the *lateral lemniscus*; here lateral ganglion cells are situated (lateral lemniscus nucleus), from which a number of fibres leave which reinforce the lateral lemniscus. The lemniscus fibres of the acoustic nerve for the greater part enter the *posterior corpora quadrigemini*, from here, finally, a communication exists, through the posterior part of the internal capsule, with the cortex of the temporal cerebrum.

The arrangement of the nucleus and fibres of the *facial nerve* is much simpler. The nucleus is situated at the caudal margin of the pons in the reticular substance and extends 4 mm. frontally; the neurites which arise from it collect, forming thin bundles, which primarily wend their way dorsally, towards the floor of the fourth ventricle. Hereupon the root, turning two loops, forms the "knee" of the "facial nerve," and now ventro-laterally has its exit as the trunk; the nucleus of the abducens is embedded in the knee. From the facial nucleus fibres also extend to the root of the opposite side; besides, fibre fasciculi travel from the pyramids of the pons medially towards the rhaphe, *cross* there and reach the opposite facial nucleus, tracts upon which the influence of the will upon the facial nucleus attains action from the contralateral side of the brain. Finally, fibres join the arising crus of the roots of the facial nerve which have their origin in another cell nucleus than in the facial nucleus, so that, in complete atrophy of the facial nucleus, some of the facial fibres in their exit are found intact. It is assumed, as, in the destruction of the muscles supplied by the *upper* facial nerve (orbicularis palpebrarum and frontalis) in the newly born rabbit, the posterior portions of the oculo-motor nucleus of the same side are found degenerated (Mendel), that the fibres of the peripheral neuron of the upper facial nerve arise from cells in the region of the oculo-motor nucleus and pass downward in the posterior longitudinal fasciculus to the facial nucleus. Besides, the upper facial nerve appears to have its own cortical centre (in the upper parietal lobule), separated from the chief centre of the facial nerve (in the anterior central convolution), and to descend in special tracts in the corona radiata.

The *abducens nucleus* lies, as has already been mentioned, in the knee of the facial root. Here fibre branches enter, probably of sensory nerves as from pyramidal fibres, the latter apparently having previously crossed in the rhaphe. Besides, collaterals from fibres of the posterior longitudinal fasciculus terminate around the nuclear cells of the abducens. Especially peculiar, finally, is the connection of the abducens nucleus with the upper olive, whereby a combination of acusticus fibres with those of the abducens, serving for reflex acts, or co-ordination effects on the part of the corpora quadrigemina through the lateral lemniscus with the oculo-motor nerve, seem to be brought about.

The last nerve nucleus situated in the pons medulla is the *trigeminus*, the complicated structure of which and its connections with other cranial nerves has only become plain by the latest investigations.

The *motor nucleus of the trigeminus* appears in the pons frontally and somewhat dorsally to the nucleus of the facial nerve, and may be looked upon as its local continuation. The neurites of the nuclear cells form the motor root (portio minor

trigemini), which innervates the muscles of mastication. Probably it contains fibres from the nucleus of the same side and from that of the crossed side, besides fibres which arise from cells in the region of the corpora quadrigemina, laterally to the aqueduct of Sylvius, and farther caudally, in the locus cœruleus at the beginning of the fourth ventricle (see Fig. 42, Vc, middle cranial root of the fifth nerve).

According to Ramón y Cajal these middle brain fibres, which descend without crossing, in the neighbourhood of the chief motor nucleus of the fifth nerve give off numerous collaterals to it and form rich terminal plexuses around the cells of the principal nucleus. Therefore, we must probably look upon this neuron chain between the nucleus situated frontally and the more caudal chief nucleus of the fifth nerve as a motor interpolated neuron of the trigeminus, and we may, perhaps, assume with Ramón y Cajal that irritations arising in the frontal nucleus increase in the principal nucleus, and that thus an accumulation of the stimulation occurs in the chief nucleus which is so important for the energy of the act of mastication. Collaterals of the sensory root fibres of the fifth nerve also approach the cells of the motor trigeminus nucleus and transmit the reflexes originating in the former to the muscles of mastication. The communication of the motor trigeminus nucleus with the cerebrum unquestionably occurs by the pyramidal fibres. It has not been possible as yet to determine it anatomically; however, from an anatomical standpoint there is nothing opposed to the assumption that fibres from the pyramidal tract, going through the tegmentum to the rhaphe and above the latter, reach the contralateral motor nucleus. The lower third of the anterior central convolution and the adjoining cortical parts are usually regarded as the cortical field for the motor trigeminus.

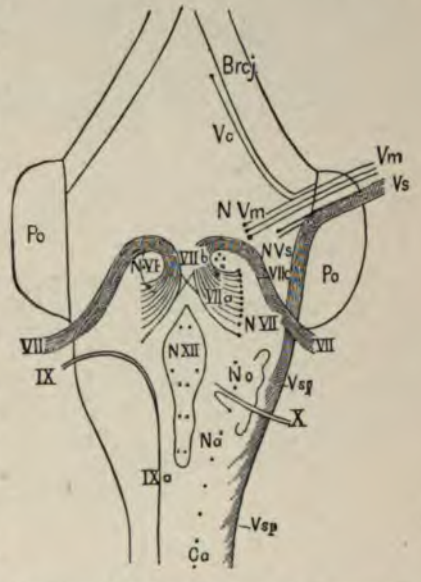


FIG. 42.—DIAGRAM OF A BASAL CROSS-SECTION OF THE MEDULLA OBLONGATA. (After Obersteiner.)

Po, brachium pontis; *Brcj*, brachium conjunctivum; *Vc*, cerebral, *Vsp*, spinal, *Vm*, motor, *Vs*, sensory, trigeminus root; *N Vm*, motor, *N Vs*, sensory, trigeminus nucleus; *N VII*, nucleus of the facial nerve; *VIIa*, *b*, *c*, root of the facial nerve; *VII*, exit of the facial nerve; *N VI*, nucleus of the abducens nerve; *IXa*, ascending glossopharyngeal root; *IX*, its point of exit; *No*, nucleus olivaris; *X*, nervus vagus (or glossopharyngeus), with the origin of some of its fibres from the nucleus ambiguus *Na*; *Ca*, anterior horn of the spinal cord; *Ca*, *Na*, *N VII*, *N Vm*, column of the original motor nucleus.

The *sensory trigeminus nucelus* also for the greatest part is situated in the pons, but extends from here, for a very long distance, caudally into the cervical cord and below the pyramidal decussation; centrally, also, the nucleus reaches still a little farther upward than corresponds to the point of entrance of the sensory root. The Gasserian ganglion with its cells is in the same relation to it as is the spinal ganglion to the posterior spinal cord nerve root, i. e., there arise, from the cells of the Gasserian ganglion, fibres which enter the fibres of the pons as a trunk, the central root fibres, whereas the peripheral trigeminus fibres extend to the opposite side. After the entrance of the roots into the pons, the majority of the fibres divide into an ascending and a descending branch. The branch ascending towards the cerebrum divides with its terminal arborizations around the nuclear cells situated frontally to the place of entrance; the descending branch travels towards the spinal column (*spinal trigeminus root*) and gives off numerous collaterals which divide around the trigeminus nuclear column situated towards the cord. From these nucleus cells, then, neurites are given off (*central sensory trigeminus tract, secondary sensory fifth*

nerve neuron), which with their collaterals, partly as reflex tracts join the motor nucleus of the hypoglossus, of the fifth nerve, and of the facial nerve (also from the sensory root fibres themselves, i. e., from the neurons of the first order, reflex collaterals go to the original motor nucleus), partly cross the middle line and, bending, ascend (being crossed) in the reticular substance, to the mid-brain, to terminate with the fibres of the upper lenmiscus in the ventral thalamus nucleus, respectively to become connected with the *tertiary cortical trigeminus tract.* Finally, communications exist between the fifth nerve and the cerebellum, and that probably both by direct fibre tracts to the cerebellum and by the fibre tracts originating in the terminal nucleus of the trigeminus and extending to the cerebellum.

The accompanying diagrams (Fig. 42 and Fig. 43) will show the location of the individual cranial nerve nuclei.

As has already been remarked, the individual columns of the spinal cord are so shifted in the medulla oblongata that the pyramidal lateral columns, entering the

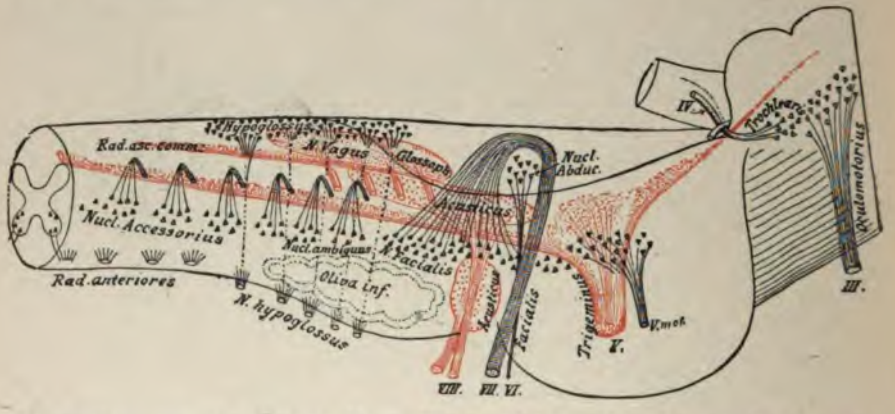


FIG. 43.—POSITION OF THE CRANIAL NERVE NUCLEI. Medulla and pons supposed to be transparent. (After Edinger.)

anterior columns, here form the combined pyramidal column, the basic fasciculi of the anterior columns being forced posteriorly and forming, with the basic fasciculi of the lateral column, a part of the lemniscus layer. Into the latter the posterior column fibres radiate more and more, whereas the area of the disappearing posterior columns is partly occupied by the newly formed posterior column nuclei, that of the lateral columns being occupied by the olives.

Corpora Restiformia.—On the other hand, the cerebellar lateral column tracts in general retain their position at the periphery, and in the medulla oblongata continue directly into the *restiform bodies*, of which they form the principal base and through which they travel, strengthened by fibres from the posterior columns to the cerebellum. The restiform body receives, however, quite a fibre mass from the *olive of the opposite side* (cerebellar-olivary tract); their origin from the cerebellum can be determined from the fact that in destruction of the cerebellum the fibres atrophy with the crossed olive. The fibres of the restiform bodies are in connection with the fleece of the cerebellum and this again with the brachium conjunctivum, the red nucleus of the tegmentum in the region of the corpus quadrigeminus and of the thalamus—a fibre complex which probably functionally belongs together, and, as has been already mentioned (see p. 543), has to do with the equilibration of the body (compare also Fig. 48).

Pons Varolii.—Cross-sections of the pons (compare diagram, Fig. 44), according to the height at which the section has been made, show various conditions. In general, as main constituents of the pons, there are presented, first, *the ventrally situated mass of the pyramidal fibre tracts and the fronto-temporal pontal fibre*

tract; both radiate from the foot of the cerebral peduncle into the pons, the former traverse the pons without crossing and reach the medulla oblongata, the latter extend into the cerebellum, crossed, after they have divided around the cells of the pontal ganglia as the principal constituents of the brachium pontis (compare p. 619); other constituents of the pontal ganglia are the fibres which originate in cells of the cerebellum and enter the pontal ganglia (therefore in an opposite direction).

Dorsally to the above-described fibre masses there are found, lying directly upon them, the tegmental portion of the pons, especially the lemniscus (compare Fig. 44); furthermore, the *reticular substance* and the *posterior longitudinal fasciculus* which separates from it, lying dorsally and consisting of anterior column basic bundles.

We designate by *substantia reticularis* a formation consisting of narrow fibres, principally of the anterior lateral column basic bundles, and of numerous nerve cells

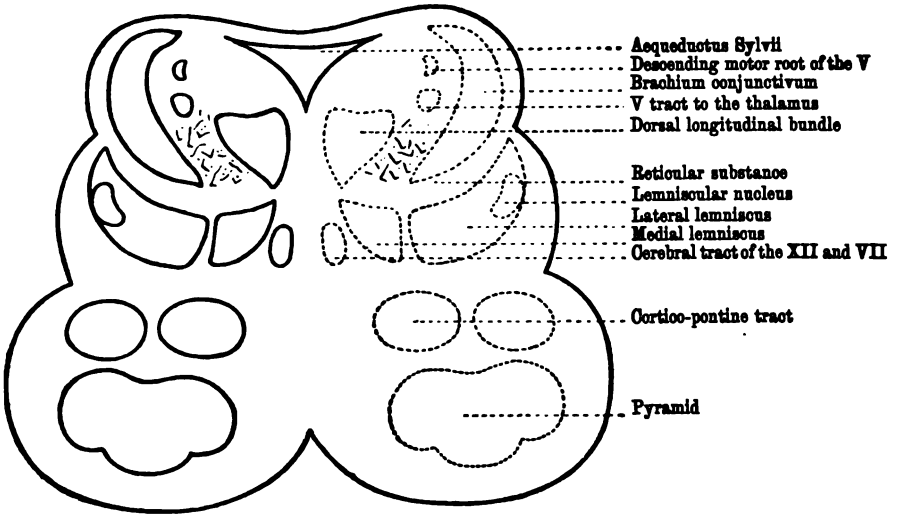


FIG. 44.—DIAGRAM OF A CROSS-SECTION OF THE PONS.

which are intermingled; the nerve cells of the reticular substance may be considered equivalent to the column cells of the spinal cord and, with their axis cylinders and their collaterals, serve associated actions.

The *posterior, "dorsal" longitudinal fasciculus* may easily be followed to the uppermost region of the corpora quadrigemina; it lies, extending as a longitudinal fibre tract, from the region of the corpora quadrigemina to the beginning of the spinal cord, ventrally from the aqueduct of Sylvius, respectively beneath the floor of the fourth ventricle on both sides of the rhaphe. The origin of the posterior longitudinal bundle is, at least in the main, found in a group of cells which are situated in the central gray matter of the cavity of the third ventricle; a great portion of the fibres of the longitudinal fasciculus also originates from cells of the oculo-motor nucleus. We may assume that the entirety of the fibres of the dorsal longitudinal fasciculus represents the communicating fibres between the individual cranial nerve nuclei which follow one another.

Both structures, the reticular substance and the posterior longitudinal fasciculus, appear to connect, in the medulla, in the pons and upwards, by their fibres and their connections with sensory and motor nerve cells, various heights of these parts of the central nervous system with each other, and to bring about reflex acts and associated actions which play the most important part especially in the functions of the medulla oblongata.

Whereas the pyramidal fibres, intended for the innervation of the extremities, traverse the pons without being crossed, the cerebral central fibres of the cranial nerves decussate, unquestionably, partly in the pons, especially those of the V, VI, and VII cranial nerves, and that immediately before they come in contact with their nuclei, i. e., therefore in the lower portion of the pons. The peripheral cranial nerve

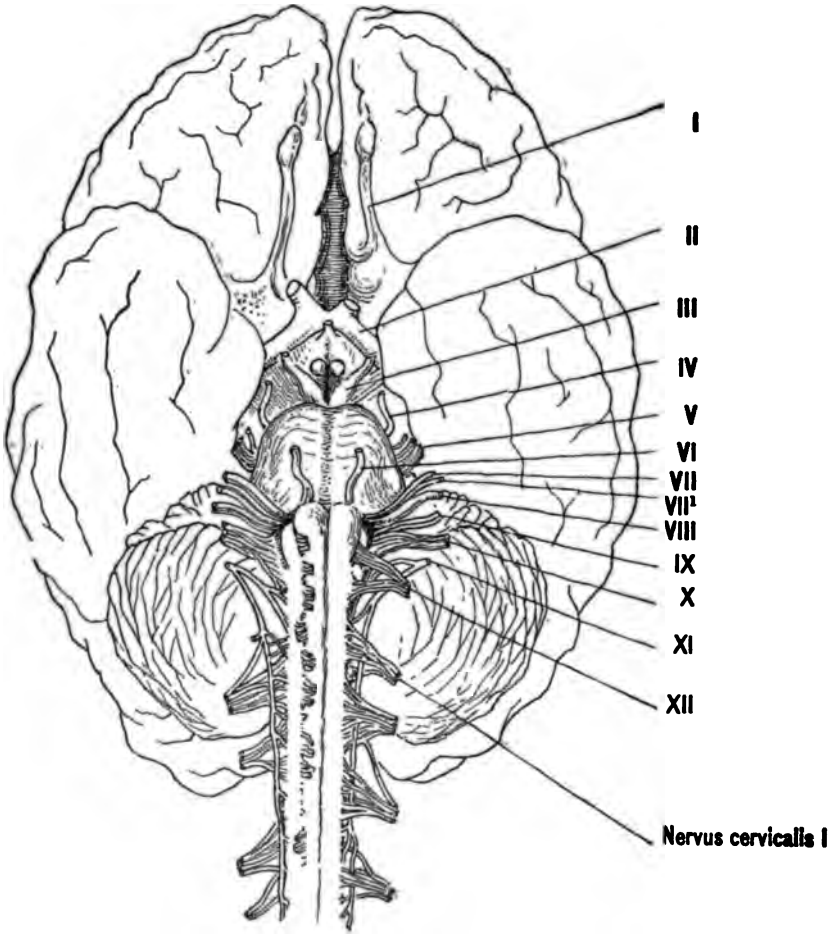


Fig. 45.—Exit of the Cranial Nerves at the Base of the Brain. (After Henle.)

fibres arising from the nucleus, collected in trunks, go off laterally from the pons in the well-known order (Fig. 45).

Functions of the Medulla Oblongata.—In a *functional* respect, the medulla oblongata serves, partly, as a conducting organ for the previously mentioned nerve tracts, and partly as the point of origin for distinct orderly *reflex movements*, the *centres* of which may be looked for in the medulla oblongata. These are the centres for the *secretion of saliva* (stimulated by irritation of the medulla oblongata), for the *act of deglutition* (sensory nerves of the palate and pharynx V 2 and 3, X—motor fibres from the vagus), for the *movements of sucking and mastication* (centripetal nerves are the sensory nerves of the cavity of the mouth V 2 and 3, IX, motor V 3, VII, XII), for sneezing, for coughing and for vomiting, for *palpebral closure* (V 1 and

VII) and, finally, for *dilatation of the pupils* (motor fibres originating from V 1, ciliary ganglion and, probably, also from the cervical sympathetic, are usually stimulated by shading the retina or by a dyspnœic condition of the blood). In the medulla oblongata, especially at the apex of the sinus rhomboideus in the region of the nucleus of the vagus, is situated the *predominating centre of respiratory movements*. This, consisting of two alternately active centres, the *inspiration and expiration centres*, is mostly irritated reflexly and should, moreover, be looked upon as automatic centre, which depends upon the condition of the blood for its irritability and is stimulated especially by a dyspnœic condition of the blood. The *centre for the inhibitory nerves of the heart* (and probably also for the accelerators) is, also, situated here, so that affections of the medulla oblongata, among other symptoms, may show also changes in the action of the heart. Further, in the medulla oblongata is situated the *chief centre for the secretion of sweat* of the entire surface of the body, and at the floor of the fourth ventricle the *predominating centre of the vaso-motors*, the irritation of which produces, as is well known, contraction of all the arteries, resulting in an increase of blood pressure, whereas its paralysis causes dilatation of the arteries and an abnormal lowering of blood pressure. If a medial portion of this vaso-motor centre is destroyed, there occurs, as the well-known Claude Bernard-puncture experiment has proved, melituria.

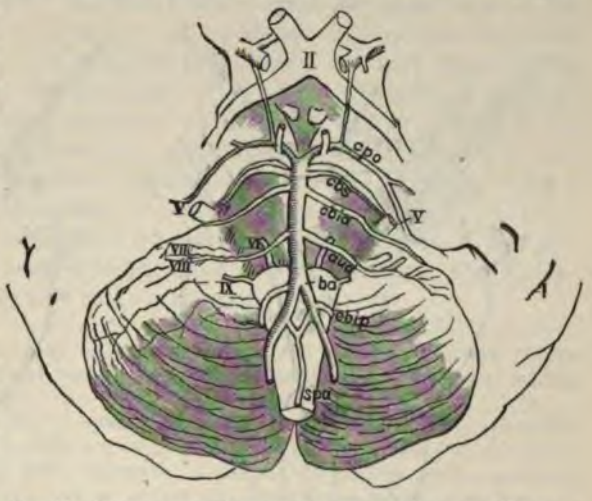


FIG. 46.—CIRCULATORY DISTRIBUTION IN THE PONS AND MEDULLA.

cpo, arteria cerebri posterior; *cbs*, arteria cerebelli superior; *cbia*, arteria cerebelli inf. ant.; *ana*, arteria auditiva; *ba*, arteria basilaris; *cbip*, arteria cerebelli inf. post.; *spa*, arteria spinalis anterior.

Finally, it must be remembered that intense irritation of the medulla oblongata gives rise to general reflex movements in the form of convulsions, so that it was assumed for some time that the medulla oblongata contains a special "*spasm centre*," the irritation of which was supposed to be dependent upon variations in the constituents of the blood, respectively upon change of gases in the same.

The blood supply in the pons and medulla oblongata occurs (compare Fig. 46) by the *vertebral arteries, respectively by the unpaired branches* arising from them, the *basilar artery* (extending above, over the middle of the pons) and the *anterior spinal* artery (extending downward over the medulla oblongata to the spinal cord). The latter artery, the anterior spinal, supplies the substance of the medulla oblongata, the nuclei of the accessorius and hypoglossus, situated in the lower portions of the medulla, whereas the nuclei of the facial, abducens and trigeminus nerves, situated in the pons, receive their arterial blood from the basilar artery. The medulla oblongata receives an additional blood supply by lateral branches of the vertebral artery: The posterior inferior cerebellar arteries and the posterior spinal arteries, which are of importance in the diagnosis of arterial thrombosis in the region of the pons oblongata, as we shall see farther on.

DIAGNOSTIC PRELIMINARY REMARKS

Clinico-Diagnostic Points of Support.—The diagnosis of *diseases of the pons and medulla oblongata* is not difficult in some cases; in other cases it is so uncertain that it is best to refrain from making a positive diagnosis. Even with great readiness and care in diagnostic localization, we run the danger of making very incorrect diagnoses, especially in assuming a focus

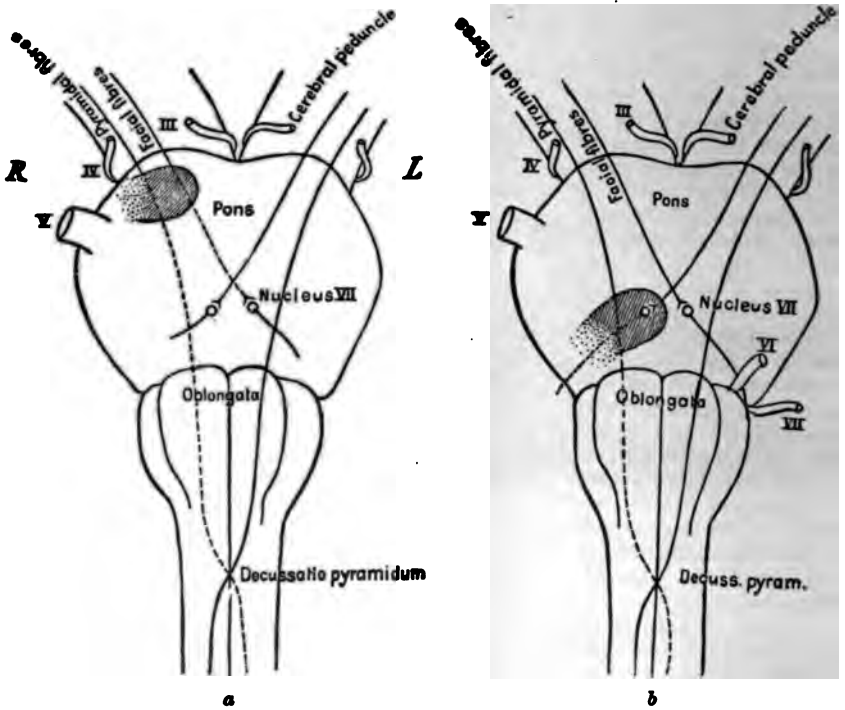


FIG. 47.—DIAGRAM TO JUDGE THE CONSEQUENCES OF PONS FOCI; —— PRESERVED, ------ DEGENERATED. FIBRES.

in the cerebrum, whereas a disease of the pons is present. This is self-evident if we consider that lesions which are situated in the uppermost portion of one half of the pons, i. e., above the decussation of the cranial-nerve fibres and their nuclei (compare Fig. 47, *a*), must produce the same symptoms as occur in cases in which, higher up, likewise uncrossed correlated tracts of the extremity and cranial-nerve tracts are affected. In both instances hemiplegia of the contralateral side of the face and of the extremities (eventually also of the tongue, very rarely only paralysis of the extremities or only facial paralysis) will be the result, with which also hemianæsthesia upon the paralyzed side will be found in combination. Such examples of pontal affections occur; then a diagnostic differentiation

of the latter from focal disease in the brain, especially in the corpus striatum, is no longer possible.

A distinct direction, however, is given to the diagnosis if the paralyses show another character than those arising in the corpus striatum, especially if symptoms occur which are directly due to disease of the pons or medulla oblongata. The characteristic form of paralysis occurring in diseases of the pons, especially in affections of the lower portions (see Fig. 47, *b*), is the so-called *alternating, crossed* (Gubler and Millard), *in which the extremities* (and eventually also the tongue) *appear paralyzed upon the contralateral side, the facial nerve upon the same side as the lesion.* The appearance of this form of paralysis becomes clear from observation of Fig. 47, *b*. The paralysis of the facial, in such cases, affects all branches of the nerve, also the upper twigs, and in the area of paralysis the symptoms of peripheral paralysis (DeR, etc.) appear. Besides the facial nerves there may be paralyzed, *alternatingly,* in lesions of the pons and particularly of their nuclei or peripheral fibres: The *abducens* and the *trigeminus,* the latter in its motor or sensory fibres and nuclei, i. e., there may be paralysis of the muscles of mastication and anæsthesia of one side of the face, both being crossed with paralysis of the extremity. Also alterations in taste and hearing now and then occur.

The *disturbance in taste* may be explained from the eventual affection of the glosso-pharyngeal nerve nucleus, respectively of the central fibres of taste arising from it and travelling upward.

The *disturbance in hearing* is easily explained if we reflect what a plentiful opportunity there is for an implication of the acusticus cells and fibres in the lower part of the pons (compare p. 604), especially of the ventral nucleus of the acusticus (at the lateral border of the pons) or of the trapezoid fibres which arise from it, the fibres of the lateral lemniscus and the cells of the upper olive and of the nucleus of the lemniscus! According to the seat of the lesion in the pons, there will occur partly unilateral, partly contralateral or even bilateral deafness.

As soon as the tegmentum of the pons, especially the lemniscus, is affected, *disturbances of the sensation of the contralateral side* appear, as the lemniscus, especially the medial one (see Fig. 50), contains the crossed central sensory tracts. Lesions situated in the medulla oblongata cause disturbance in the muscle sense if the intermediary olivary layer is affected, whereas an implication of the more laterally situated fibres of the medial lemniscus gives rise to crossed anæsthesia of the trunk and extremities. Lesions of the pons by foci of the medial lemniscus and the reticular substance may produce crossed anæsthesia and ataxia. It has already been mentioned that there may also be anæsthesia of the face of the opposite side, i. e., corresponding to the side of the lesion by an affection of the nucleus of the trigeminus or of its root fibres. If, finally, not only in diseases of the medulla oblongata but also in affections of the pons, *difficulty in deglutition* and *dysarthria* are observed, this may be explained in the following manner, that the cerebral fibres of the spinal accessory and hypoglossus have been damaged in the pons. *On the other hand, paralysis of the oculo-motor and trochlearis have nothing to do with diseases which remain limited to the pons,* as the latter nerves arise in the region

of the corpora quadrigemina and with their trunks come forth above the pons.

Besides the symptoms of paralysis of the extremities and those portions of the body supplied by the individual cranial nerves, there must be further considered in affections of the pons oblongata the implication of the previously mentioned centres in this position of the central nervous system, giving rise to disturbance in the act of mastication, in movements of the tongue, to dysphagia, vomiting, dysarthria, arrhythmia, increase or diminution of cardiac activity, respiratory disturbances (dyspnœa, Cheyne-Stokes's respiration, singultus), eventually even with a clear mind and with but slight cranial pressure and vaso-motor disturbances. Finally, in disease of the pons, especially in pontal hæmorrhages which occur suddenly, general *spasms* have been observed; clonic and tonic spasms, localized to individual muscles, also occur and trismus, especially in affections of the pons. In rare cases the symptoms of *ataxia* appear, which is not to be wondered at considering the structures that are affected (especially the corpora restiformia, cerebellar lateral-column fibres, posterior-column fibres and the fibres originating in the olive). With these symptoms *vertigo* is usually combined, which under some circumstances may increase to such an extent that actual constrained movements arise.

Differential Diagnosis between Diseases of the Pons and Medulla Oblongata.—Whether the pons or medulla oblongata are affected separately or, as is frequent, both at the same time are the seat of disease, may be determined in the individual case with great probability. In favour of an affection of the *medulla oblongata* is the distribution of the motor-extremity paralysis to both halves of the body, in which the most varied pictures of an implication of the four extremities are noted: Paralysis or paresis of all four extremities, hemiplegia, possibly also the rare so-called "*hemiplegia cruciata*," i. e., paralysis of the arm upon one side and of the leg upon the other side. The implication of both halves of the body and the appearance of these various modifications of paralysis may be explained by the very crowded condition of the pyramidal-tract fibres in a narrow space and their decussation at various heights. The diagnosis, however, only attains a firmer support in diseases of the medulla oblongata by the appearance of disturbance in the function of the cranial-nerve nuclei and centres which are situated in the medulla, i. e., respiratory and circulatory disturbances, as well as of paralytic phenomena in the distribution of the vago accessorius and of the hypoglossus (*difficulty in deglutition, aphonia, dysarthria*) *with a simultaneous absence of paralytic symptoms in the area of the innervation of the facial, abducens and motor portions of the fifth nerve.* The latter limitation of the diagnostic value of dysphagia is necessary, as functional disturbance on the part of the vagus and hypoglossus also occur in diseases of the pons, as has already been mentioned, but they are then combined with paralytic phenomena in the area of innervation of the V 3, VI and VII. *If the paralysis of the hypoglossus is combined with atrophy of the muscles of the tongue and DeR. this is in favour, if there be other symptoms of disease of the medulla also* (dysarthria, hemiplegia cruciata, glycosuria, etc.), *directly of disease of the medulla*

oblongata. Sensory disturbances are of less importance from a differentio-diagnostic standpoint, as hemianæsthesia occurs in affections of the pons and medulla uniformly, and not even crossed anæsthesia of the face is pathognomonic of affections of the pons, in that it is also observed in bulbar affections (in so far as the descending spinal root of the fifth nerve is affected) (compare Figs. 42 and 43).

In favour of *disease of the pons,* in contrast to disease of the medulla, are: *Well-developed alternating paralysis* of the facial, fifth and abducens, in so far as they show the characters of a peripheral paralysis as far as this may be determined in the area of these nerves (muscular atrophy, reactions of degeneration, absence of reflexes, etc.). The diagnosis is aided if, besides the above symptoms, ataxia occurs and if, in instances in which the affection appears abruptly, general convulsions arise.

Conjugate Deviation of the Eyes.—Frequently in pontal affections, apart from the usual picture of paralysis of the abducens (strabismus convergens), a peculiar position of the eyes has been noted—a turning of the eyes (and of the head) to the opposite side from the lesion. There exists, therefore, for example in a conjugate movement of the rotators of both eyes which move them towards the left side, a lesion in the right half of the pons, in contrast to the conjugate deviation of the eyes in focal affections in the cerebral areas above the pons, in which the eyes appear to turn towards the side of the lesion, so that, therefore, in the example assumed, in a rotation of the eyes towards the left, a lesion of the left cerebral hemisphere would be expected; the eyes in the latter case turn "towards the lesion," in a unilateral pontine affection "away from the lesion." This condition is, as is assumed, due to disease of a certain centre in the pons, which is situated in the region of the nucleus of the abducens, or in the nucleus itself and acts in the manner that the right centre of the movement of the eyes turns to the right, the left dominating that which turns towards the left. Perhaps this is also in relation with a firm connection of the abducens with the cells of the oculo-motor for the internal rectus of the opposite side, which is produced by fibres in the dorsal longitudinal fasciculus, so that in lesions of the abducens of the right side the right abducens and the internal rectus of the opposite eye are paralyzed, both eyes therefore turn towards the left—away from the lesion. Regarding conjugate deviation of the eyes in affections of the cerebrum this will be referred to later on.

Diagnostic Differentiation from Basal Tumours.—Finally it must be also considered that localized focal affections in the region of the pons medulla at the base of the brain may produce similar phenomena, such as intrapontine and intrabulbar lesions, in that from compression in the pons and medulla oblongata paralyses of the extremities may occur, due to pressure of the cranial-nerve trunks which find exit there, and these may give rise to alternating paralyses in their area of innervation. The symptoms of these paralyses of the peripheral-cranial nerves are the same as those of paralyses of the nucleus or root situated in the pons and medulla oblongata. Theoretically, we should require that, in the latter, occasionally the cranial-nerve tract of *both* halves of the body should be affected (whereas a paralysis of the trunk which is separated by the entire width of the pons and medulla oblongata by *one* tumour is not conceivable without death having occurred previously), and on the other hand, under all circumstances, not all, but only some individual, fibres arising in the nucleus should be affected by the paralysis—assumptions which, as experience

teaches us, are in reality not so absolutely correct that they may be utilized in a differentio-diagnostic respect. Certainty cannot be attained in this respect in the differential diagnosis; however, at least probable diagnoses are allowable in that in most cases, as soon as, from an ætiological standpoint, certain evidence is at hand (syphilitic periostitis, etc.). After an explanation of these general diagnostic rules, the special diagnosis of the individual affections of the pons and medulla oblongata (with the exception of progressive bulbar paralysis) may be brief.

HÆMORRHAGES OF THE PONS AND OF THE MEDULLA OBLONGATA

The diagnosis is based upon the sudden apoplectiform appearance of phenomena which point to an affection of the pons or medulla oblongata.

Hæmorrhages which affect the medulla oblongata alone, are in general very rare; usually the pons is simultaneously affected. As the important centres of respiration and circulation, which are necessary for the existence of life, have their seat in the medulla oblongata, death occurs almost immediately, or after but a few hours, following hæmorrhage into the medulla. There can be no question of a diagnosis in such cases, as sudden death also occurs in apoplectiform attacks, in hæmorrhages which affect the pons or the ventricles of the brain (naturally in the latter instance by the immediate effect upon the medulla oblongata). The conditions are different if the hæmorrhagic area be small, or if we have to do with capillary hæmorrhages in the medulla; then the clinical picture of a "bulbar paralysis" may occur (eventually with paralysis of the extremities, crossed hemiplegia, glycosuria, etc.) which, on account of its sudden appearance, has received the name of *acute apoplectiform bulbar paralysis*. According to experience, even after the appearance of the disease in this form, we should think less of hæmorrhage than of an embolism or thrombosis, which appear with similar phenomena (as in hæmorrhage) in the vessels of the medulla oblongata, the diagnosis of which is possible upon the basis of the clinical picture which is to be described later on.

Pontal hæmorrhages (with or without an affection of the medulla oblongata) are more frequent. With or without a sudden onset severe paralyses occur which affect partly all four extremities (however, only in cases which rapidly terminate in death), partly the contralateral side of the body. With this *general epileptiform convulsions* may appear; these are neither constant nor characteristic of pontal hæmorrhage, as they also occur in hæmorrhages of other parts of the brain or into the meninges, not even being rarer in these conditions. If an apoplectiform *hemiplegia* has been demonstrated, we must observe the presence of *alternating cranial nerve paralysis*, of respiratory disturbances, difficulty in deglutition, myosis, difficulty in articulation, etc., briefly the characteristic symptoms which occur in lesions of the pons-medulla region. It must, however, not be forgotten that most of these symptoms may arise from remote action if the seat of the hæmorrhage is in other parts of the brain. Important for the diagnosis, therefore, under all circumstances is it to note the severity of the general phenomena in the individual case; the slighter they are and the more they improve, the more readily may the existing focal phenomena be utilized for the local diagnosis. In the latter connection, the determination of alternating paralysis gives the most important support for the diagnosis of pontal hæmorrhage (in which the cranial-nerve paralyses show the character of peripheral paralysis), only secondarily are conjugate deviation of the eyes, pupil disturbances and dysarthria of value.

The Process of Softening in the Pons and Medulla Oblongata—Embolism and Thrombosis.—The occlusion of the vessels supplying the medulla and pons, which gives rise to softening of these areas, may occur through embolism or thrombosis. The former, as the result of cardiac lesions, almost exclusively affects the left verte-

bral artery, from which secondarily the occlusion of the basal arteries occurs. This artery, as its lumen is wider than that of the vertebral artery, is not obstructed primarily by an embolism, but most commonly by thrombosis, as this artery is frequently the seat of atheroma (most often the result of syphilis). Only in very exceptional cases is the medulla oblongata exclusively the seat of softening; usually this occurs in the pons or simultaneously in the pons and medulla.

The occlusion of the vessel may occur suddenly, and now the clinical picture of *apoplectiform acute bulbar paralysis* arises. This may give rise to a paralysis of all four extremities, or the extremities of one half the body and occasionally in the manner that the paralysis primarily occurs in one, and a few days later in the other half of the body. Further, there appears, according to the vessel which is affected, an absence of function on the part of the individual cranial-nerve nucleus, especially of the hypoglossus, the vago accessorius, or, in occlusion of the basilar artery, also of those nuclei situated in the upper part of the pons, namely that of the facial, abducens and trigeminus. In keeping with this there are found: Disturbances in articulation as the result of weakness of the muscles of the lips, tongue, and palate, aphonia, weakness or spasm of the muscles of mastication, increase in the pulse rate and respiratory difficulties. The implication of the cranial nerves may be developed besides the paralysis of the extremities, or, in rare cases, may appear alone. Besides the motor paralytic phenomena, there may also be seen paralysis of sensation; such an instance, without motor paralytic phenomena, was noted in the trunk and extremity with alternating paralysis of sensation of the face, besides difficulty of deglutition and a change in the voice, by Senator, in which softening of one half of the medulla oblongata, as the result of thrombosis affecting the inferior posterior cerebellar artery, was present. Also disturbances in hearing have been noted from time to time, the mind (at least in those cases which are not rapidly fatal) *is not influenced.*

Differential Diagnosis.—If the bulbar affection manifests itself acutely: Motor paralysis of the extremity, eventually paralysis of sensation, dysarthria, dysphagia, respiratory and circulatory disturbances, myosis, and paralysis of individual nuclei, the question arises whether the morbid picture may coincide with an exclusive affection of the pons or of the medulla. In this connection what was previously mentioned is of value (p. 607); in individual cases at least, probable diagnoses are allowable; secondarily, the question occurs to the diagnostician whether the clinical picture of the acute bulbar affection is due to *hæmorrhage, a thrombotic embolic process* in the course of the vertebral artery or basilar artery, or if it be due to the rare condition which has been noted but in few cases (naturally running its course with the same symptoms), *acute bulbar myelitis*, which was described by Leyden. *The decision of this question is impossible at present; scarcely a probable* diagnosis is justified in the individual case. To separate hæmorrhage differentio-diagnostically from a suddenly appearing embolus in the region of the medulla is risky and uncertain, as is also the differentiation between hæmorrhage and embolism in the vessels of the brain. Regarding the latter differential diagnosis, this will be referred to later on, especially the points which permit of a probable diagnosis in one or the other direction. In this connection the possibility of the demonstration of the source of the embolus (occurring most usually in young individuals), the simultaneous appearance of embolism in other parts of the body, and the eventual rapid readjustment of severe paralytic phenomena give the relative best point of support for the existence of embolism. After we have gone so far, the attempt to diagnosticate whether the vertebral or basilar artery has been affected by embolism or thrombosis, or even to attempt to say which branch of the vessel has been affected after what has been said, is *a priori* a futile undertaking. Setting up diagnostic rules in this respect is for the greatest part theoretical nonsense, on account of the manifold varieties of the arrangement of the vessels in question.

Among the *bulbar affections running a chronic course* the most important unquestionably is *chronic progressive bulbar paralysis.* The diagnosis of this disease can usually be made with certainty; the conditions shall be described minutely, whereas the other chronic bulbar affections, in so far as they must be diagnostically considered, shall only be mentioned supplementarily.

CHRONIC PROGRESSIVE BULBAR PARALYSIS—DUCHENNE'S DISEASE

By far the most frequent disease of the pons-medulla is glossopharyngolaryngolabial paralysis or, as it is designated now, *chronic progressive bulbar paralysis,* first described by Duchenne in 1861. The disease is concentrated to the motor-nerve nuclei which are situated in the pons-medulla, constituting a degenerative atrophy of the same, with secondary atrophy of the root fibres which go off from the nucleus of the peripheral motor nerves and their end organs, the muscles. The most marked changes are shown by the nucleus of the hypoglossus, the ganglion cells of which degenerate earliest and most intensely; in a similar manner, even if to a less degree, the nucleus of the vago accessorius and especially also the nucleus of the facial, in rarer cases finally also the glossopharyngeal and the motor nucleus of the fifth nerve, are affected. The disease may be limited to atrophy of the previously mentioned nuclear areas or—and this is the usual case—go hand in hand with an affection of the white substance especially of the pyramidal tract. In a portion of the cases, well-developed changes in the spinal cord are found, producing phenomena of progressive muscular atrophy, being a genetically analogous affection of the anterior horns. The process may also develop upward, in the tract of the motor-ganglion-cell regions, i. e., besides the abducens, also the trochlear and oculo-motor may be affected, giving rise to a complete immobility of the eyeball, with simultaneous ptosis—the picture of "*progressive ophthalmoplegia.*" As progressive muscular atrophy and ophthalmoplegia may close the scene, they may also, each individually, form the first link in the chain; in a part of the cases, finally, the affection is limited to one of the three areas and polioatrophy anterior, or bulbar paralysis, or progressive nuclear ophthalmoplegia retains a certain individuality. That these affections belong together genetically may be regarded as certain; also amyotrophic lateral sclerosis which is in such close relation with progressive muscular atrophy, may in its course show the symptoms of bulbar paralysis. The diagnosis must always take into consideration this merging of the previously mentioned morbid types into each other, they are identical in their nature, and this must be remembered in the examination of the individual case.

Disturbance of Speech—Anarthria.—The symptoms upon which the diagnosis of bulbar paralysis is based are very marked, the most prominent being *disturbances of speech*—disturbances in articulation. This is primarily due to an atrophy of the nucleus of the hypoglossus, producing a wasting and a difficulty of movement of the *tongue,* preventing the formation of certain consonants. These are the ones produced in articulation between the tongue and palate, above all l and r, further t, n, and s. As soon as the lips are affected in the progressing atrophy, the formation of labial sounds b, f, w, m, in paresis of the palate, k, ch, and n are affected, the speech becoming nasal. In an insufficient narrowing of the glottis, the formation of the vowels, audible; a, can be said most

readily by the patient, as the tongue remains immovable at the bottom of the oral cavity in its formation and the lips are simply opened, whereas in the other vowels, the tongue, lips and palate are more or less utilized in the formation. Finally, anarthria may assume such dimensions that, in place of speaking, an unarticulated grunting occurs. In this advanced stage of anarthria, the differentiation from aphasia may give rise to great difficulty; as a rule, however, there should be no doubt as to the character of disturbance of speech, if, in the examination, words are chosen, the letters of which may be expressed, even though in an insufficient manner.

Other Symptoms of Nuclear Paralysis.—Externally the disturbance of function in speaking is noted by atrophy of the tongue, which may be seen as a thin, narrow immovable tissue, at the bottom of the mouth cavity, showing fissures at its borders and back, as well as active *fibrillary contractions* (like other muscles in the course of progressive muscular atrophy). In the integral part which the tongue plays in deglutition and chewing, it is evident that disturbance of the nucleus of the hypoglossus in itself increases the difficulty in these acts. This is all the more the case, if the nucleus of the vago-accessorius, the facial nucleus and the motor nucleus of the fifth nerve are affected by the morbid process. With increasing *dysphagia* the nutrition of the patient suffers more and more. A further result of the atrophic degeneration of the nucleus of the facial and vago-accessorius is *immobility of the lips*; they are small and have become fissured, and also show fibrillary contractions. The mouth appears broader, rigid in laughing, remains open and can no longer be closed tightly, the lower lip droops; whistling and similar actions depending upon the functions of the lips are impossible. Finally the complete picture of *diplegia facialis* occurs, in which the upper branches of the facial, as a rule, are exempt from the paralysis (as may readily be understood from their anatomical distribution). The lower part of the face then assumes a mask-like expression, which is in marked contrast to the wrinkling of the forehead and the active forcible closure of the eyelids.

Atrophy and weakness in function of the *musculature of the larynx*, finally, produce, as was described in the symptomatology of multiple sclerosis, the monotonous voice and the sighing inspiration in laughing, further, hoarseness or complete aphonia, so that finally no vowel even can be spoken aloud. The vocal cords, in a laryngoscopical examination, appear immovable, closure of the glottis is absent; this causes the absence of certain movements, especially coughing. If in the last stages an atrophic degeneration of the motor nucleus of the fifth nerve occurs, this produces a marked *disturbance in mastication*. This act is already greatly hindered, as has been remarked, by paresis of the hypoglossus and facial, as the rolling of the bolus towards the surface of the teeth, in such cases, is incompletely performed by the orbicularis oris and buccinator, as well as by the movements of the tongue. Naturally, the disturbance in mastication is only complete if not only these acts in chewing are absent but also those produced by the innervation of the fifth nerve which move the jaws vertically and transversely, i. e., the necessary muscular movements in chewing; in such cases atrophy of the muscles of mastication does not remain

absent. Rarely are other motor nuclei as those mentioned, such as the nucleus of the abducens, affected.

The *atrophy of the muscles* in progressive bulbar paralysis is dependent upon a lesion of the peripheral neuron, especially due to a nuclear affection; this is proven by the fact that the electrical test of the atrophic muscles shows *DeR* and that the *reflexes have disappeared.* To determine the former condition there is often great difficulty, for the same reason as in progressive muscular atrophy, as the retained muscular fibres, being side by side with the atrophic fibres, conceal the reactions of degeneration. The disappearance also of the tendon reflexes (affecting the paralyzed muscles) is not always plain; on the contrary, in rare cases an increase may be noted, analogous to the condition in amyotrophic lateral sclerosis. It must be supposed in these cases that a lesion has occurred in the neighbourhood of the nucleus ("central") affecting the reflex inhibiting fibres, besides incomplete degeneration of the nuclear ganglion cells. The disappearance of reflexes is also shown in that touching the base of the tongue, the velum of the palate and the larynx no longer gives rise to choking sensations, cough, etc., in spite of the fact that sensation in the previously mentioned structures is unaltered.

Exceptional Phenomena.—The symptoms described so far refer exclusively to the motor sphere. *Sensation and the action of the organs of sense are always intact*; nor are symptoms of weakness of the sphincters of the bladder and rectum, or secretory and vaso-motor disturbances met with. In some cases the secretion of large amounts of saliva has been noted, so that an increased stimulation of the reflex centre for ***salivation,*** situated in the medulla oblongata, must be considered. Naturally this marked increase of secretion which occurs in rare cases, must not be confounded with the seemingly existing "salivation" which is very usual in bulbar paralytics. The saliva, in these patients, flows continuously from the mouth without there being an increase of the salivary secretion as soon as the inability to swallow and to close the lips has reached a certain grade of development. The *increase in the pulse frequency* which is occasionally observed is without doubt the result of a paresis of the cardiac vagus centre, as well as the usually rare *respiratory difficulty* (irregular, superficial respiration) which occurs late; they are to be looked upon as the consequence of an affection of the predominating centre of respiratory movement situated in the medulla oblongata. *Intelligence* remains ***undisturbed*** to the end.

Differential Diagnosis.—To diagnosticate a well-developed progressive bulbar paralysis occurring in the manner just described is very easy. Nor is it difficult to recognise the affection as a "secondary" bulbar paralysis, i. e., as a part phenomenon of other diseases of the spinal cord which occasionally develop bulbar symptoms. This occurs in the course of ***anterior poliomyelitis, respectively progressive muscular atrophy, of amyotrophic lateral sclerosis*** in which not only the peripheral but also the central neurons are affected, i. e., in which the entire tract of motor innervation is degenerated; further, in the picture of *sclerose en plaques* and exceptionally also in other affections of the spinal cord. The decision becomes com-

plicated at most in regard to the diagnostic separation of cases of multiple sclerosis which run a very atypical course, as in certainly four fifths of the cases the implication of the medulla oblongata is marked in the morbid picture. However, as a rule, the circumstance that, besides the bulbar symptoms, intention tremor, nystagmus, apopletic attacks and psychical alienation occur, gives the proper direction to the diagnosis.

If these diseases, which eventually run their course with the symptoms of a secondary bulbar paralysis, are excluded in the individual case, we must reflect whether we are dealing with a primary bulbar paralysis or with another morbid process concentrated to the pons-medulla. The bulbar affections which run an acute course: *Acute bulbar myelitis, hæmorrhage* and *embolic-thrombotic processes* in the pons-medulla, may at once be differentiated from bulbar paralysis by their rapid course. Besides, these affections are characterized, in opposition to progressive bulbar paralysis, by usually unilateral and especially plainly alternating paralyses, sensory disturbances and the apoplectic insult which usually ushers in these affections.

Greater difficulties occur in the differentiation of other *chronic* bulbar affections, especially of tumours and aneurysms producing pressure upon the pons-medulla, certain varieties of bulbar paralysis which compress the medulla oblongata: The so-called pseudobulbar paralysis and progressive ophthalmoplegia. We must briefly describe these affections, in which we shall particularly consider the special diagnostic points.

PSEUDOBULBAR PARALYSIS—BULBAR PARALYSIS OF SUPRANUCLEAR ORIGIN—PARALYSIS GLOSSOPHARYNGOLABIALIS CEREBRALIS ET CEREBROBULBARIS

Pseudobulbar Paralysis.—Repeatedly (primarily by F. Jolly) bulbar symptoms have been observed: Dysarthria and dysphagia, sighing inspirations during laughing, etc., i. e., paralytic phenomena in the region of innervation of the facial, hypoglossus and vagoaccessorius in cases in which the autopsy surprisingly showed absolutely no changes in the pons or medulla oblongata; on the other hand, in various areas of the cerebrum small pathological foci were noted. The microscopical examination of the pons-medulla in a portion of these cases has even shown a positive result (especially small areas of softening as the result of distributed atheroma of the cerebral arteries or of syphilitic endarteritis), in which macroscopically normal conditions appeared; nevertheless, there still remain rare cases in which, in spite of well-developed bulbar phenomena, the cerebrum is affected. In these latter cases (*cerebral pseudobulbar paralysis*) it must be assumed that the cerebral tracts for the voluntary innervation of the muscles of articulation and deglutition are interrupted. As might be expected in the cerebral form, muscular atrophy, the fibrillary contractions in the paralyzed muscles, the alteration in the electric contractility in the sense of DeR and at least partially the disappearance of the reflexes, are absent. Besides, there point to the cerebral origin of the affection in the individual case: The intercurrence of apoplectic attacks, simultaneous hemiplegia, above all psychical disturbances and eventually aphasia. Further, especial weight has been laid in these cases upon the absence of respiratory and circulatory disturbances.

In those cases, in which, besides the areas of softening found in the brain, upon microscopic examination similar conditions were present in the medulla oblongata (*cerebrobulbar form*), the well-known disturbances in speech and deglutition that occur in the usual form of bulbar paralysis, as well as paralytic phenomena in the course of the facial, vagoaccessorius and of the motor portion of the fifth nerve (disturbances in respiration, increase in the pulse rate) are also seen. With these phe-

nomena there appear, however, certain symptoms especially dependent upon the areas of softening in the brain: Paralysis of the extremities, hemiplegia and hemiparesis (with increase of the tendon reflexes), aphasia, psychical disturbances (apathy, loss of memory, constraint affective movements), changes in the optic nerve.

PROGRESSIVE NUCLEAR OPHTHALMOPLEGIA

Ophthalmoplegia Progressiva.—As has already been mentioned, *progressive ophthalmoplegia* may be separated from progressive bulbar paralysis, as an individual disease, as soon as the former attains a certain individuality and does not arise as the result of progressive bulbar paralysis. The diagnosis of the disease is easy, as all the muscles which move the eyeball are paralyzed. With this, as a rule, the paralysis of the muscle levator palpebræ superioris is less markedly developed, the sphincter pupillæ and the muscles of accommodation are unaffected. However, in some cases both last-named muscles may also appear paralyzed; then there is added to the ophthalmoplegia externa a so-called *ophthalmoplegia interna* or *interior*, which, however, may also occur by itself. The cause of the affection is a *nuclear disease*, an implication of the nucleus of the abducens and trochlearis as well as of certain nuclear groups of the oculo-motor in the gray substance in the floor of the fourth and third ventricles. If the affection increases anteriorly in the floor of the third ventricle, i. e., if the nuclear region of the *sphincter pupillæ* and of the *ciliary muscle* is affected, *ophthalmoplegia interna* is simultaneously present. In this sense it has received the name *polioencephalitis superior chronica*, "anterior bulbar paralysis." As bulbar symptoms occasionally occur, so also may the symptoms of ophthalmoplegia be noted in the clinical picture of multiple sclerosis, tabes, etc.

Lately an ophthalmoplegia has been repeatedly observed due to alcoholism, in which multiple hæmorrhages and granular cells have been found surrounding the gray substance in the third and fourth ventricles. This would also place "acute alcoholic ophthalmoplegia" in the category of nuclear affections; it will be analogous to the usual acute bulbar paralysis and could not be looked upon as a purely peripheral neuritis. However, the purely peripheral nature of the affection must be accepted in cases of ophthalmoplegia in which the trunks of the nerves of the muscles of the eye are accidentally implicated by basal, especially meningeal or peripheral, neuritic processes.

FUNCTIONAL ASTHENIC BULBAR PARALYSIS—BULBAR PARALYSIS WITHOUT ANATOMICAL LESIONS—MYASTHENIA GRAVIS PSEUDOPARALYTICA

Affections showing a similar clinical picture to bulbar paralysis in which the autopsy shows no anatomical changes in the nervous system occur; they are therefore separated from the usual cases of bulbar paralysis as a *morbus sui generis*. Whereas this affection may also terminate in recovery, there are present, besides the symptoms of bulbar paralysis (dysarthria, dysphagia, difficulty in mastication, facial paresis, etc.), also weakness of the muscles of the trunk, of the extremities and of the eyes, as in progressive ophthalmoplegia. Especially noteworthy is the transitory character of the paralysis, principally the phenomenon that *weakness rapidly increases during the use of the affected muscles, so that the latter appear more tired than paralyzed*. The speech of the patient at the onset is plain, but even after a few sentences it may become inarticulate and can no longer be understood. Just so, the gait may at first be good, but upon continuous walking tired conditions occur rapidly, so that the patient walks slowly and with difficulty and finally cannot walk at all. Sensation is intact, also the condition of the reflexes is usually normal, rarely diminished or increased. Vaso-motor and trophic disturbances, psychical alterations and difficulty in voiding urine are absent. Intercurrently attacks of difficulty in respiration and tachycardia may occur, which may cause the death of the patient by suffocation.

The differential diagnosis must primarily consider *the ease with which the muscles used in speech, mastication, etc., tire during their employment,* also the condition of the general musculature, as the disease in question only shows functional disturbances, almost never giving rise to degenerative atrophy, the muscles showing normal electrical reactions. If the tired sensations are especially marked in the muscles, the peripheral motor neuron of which originates in the medulla oblongata and in the cerebral trunk, we are justified in speaking of a functional *bulbar paralysis.* As, however, the exhaustion of muscular activity as a rule spreads more and more to the entire motor sphere, the disease has lately been designated as *myasthenia gravis pseudoparalytica.* Jolly, who chose this designation, was also the first to note a characteristic electrical condition of reaction in the easily tired muscles. If such muscles are tetanized by means of the induction current, there appear slight contractions following one another which, however, become less marked successively, or, upon a longer continuation of the current, a uniform decrease in prolonged action of the current and gradual diminution of the tetanized condition ("myasthenic contraction"). This reaction of Jolly's is by no means pathognomonic of functional myasthenia as it is occasionally absent in the disease, sometimes even occurring in bulbar paralysis with an organic lesion, being typically developed. We do not know the cause of myasthenia; probably it is due to an intoxication.

COMPRESSION OF THE PONS AND MEDULLA OBLONGATA

Compression of the Medulla Oblongata.—The clinical picture of bulbar paralysis may be the result of slow pressure upon the pons-medulla which occurs especially from disease of the bones in the surroundings of the elongated cord and of the pons, tumours in this region and especially from aneurysms of the vertebral and basilar arteries, etc. It is especially characteristic that, due to the increasing pressure upon the pons-medulla and the nerves which find their exit there, the bulbar phenomena which arise (dysarthria, dysphagia, respiratory disturbances, etc.), develop very gradually, as in other chronic affections of the pons-medulla, or the phenomena occur paroxysmally, steadily increasing in severity, *however, being combined with irritative symptoms or ushered in by them and that besides the bulbar symptoms other phenomena occur which are dependent upon the growing pressure in the cavity of the skull. Such irritative phenomena* caused by the nerves which are accessible to pressure are: Spasm and neuralgic pains in the face (VII, V), tinnitus aurium, retardation of the pulse, etc. To this are added difficulty in deglutition and disturbance of speech, phenomena of paralysis of the muscles of the tongue and face, and, from pressure of the motor and sensory nerve tracts of the extremity, also paresis and anæsthesia in the latter, which according to the position and the growth of the pressure process are not rarely unilateral at first, later becoming bilateral. Also alternating hemiplegia may occur with increased irritability of the reflexes and spasm in the paralyzed extremity, with flaccid paralysis, muscular atrophy, and DeR of the muscles of the face. The increased pressure in the cavity of the skull shows itself by the development of the engorgement papillæ [choked disk], by headache, vomiting, epileptiform attacks, etc.

By the course and variety of the disturbances, the consequences of compression are fairly well differentiated from progressive bulbar paralysis, so that the diagnosis of this condition can at least be made with probability. Now the question arises in the individual case regarding the special *cause of the compression.* In this connection it may be stated in general that a gradual increase of the symptoms of compression is in favour of tumour, aneurysm, or caries with abscess formation. The latter condition is combined with circumscribed pain in the vertebra, febrile phenomena and tuberculosis of other organs, so that this affection may be readily differentiated from the development of tumour and aneurysm. A markedly developed choked disk, severe pain in the bone, and intense compression phenomena are more in favour of a tumour. It cannot be decided whether the tumour presses externally against the pons-medulla, or develops internally, although we might expect in general that, in extrabulbar tumours, the compression of the individual cranial nerves

which have their exit here or of the pyramids (spasm or paresis of the extremity) should precede or be more developed than the disturbances on the part of the bulbar centres.

Well-developed atheroma of the arteries, especially acute vascular changes in the eye-ground, on the other hand, are more in favour of the presence of an aneurysm (in the course of the vertebral artery). If aneurysm may simultaneously be demonstrated in the carotid or even if, as Möser once had the opportunity, a vascular murmur is heard between the mastoid process and the vertebral column, the bold diagnosis of an aneurysm of the vertebral artery, respectively of the basal artery, has at last some foundation. The diagnosis of aneurysm of the vertebral artery can never be more than a probable one, even under the most favourable circumstances.

DIAGNOSIS OF DISEASES OF THE CEREBELLUM

ANATOMICO-PHYSIOLOGICAL INTRODUCTION

THE anatomical and physiological conditions of the cerebellum are by no means cleared up completely as yet. In the following description, only the most important facts which are to be regarded in pathology have been classified. The *cerebellum* represents an integral part of the *roof of the posterior brain* (pons and cerebellum) and covers the fourth ventricle which runs downward into the central canal of the spinal cord and upward into the aqueduct of Sylvius of the middle brain. It consists of the *mid portion* (*vermis cerebelli*) and both *hemispheres*; the cortex of the cerebellum which consists of gray substance covers the white central medullary substance of the cerebellum as a central *medullary nucleus*, continuing towards the periphery and dividing into branches, which become finer and finer (the picture which represents the totality of the medullary branches with their cortical layer in a longitudinal section is known as *arbor vitæ* (Fig. 48). The medullary nucleus is principally formed by three masses of medullary substances which radiate into the cerebellum, the so-called *cerebellar peduncles* (brachium cerebelli), the fibre masses of which form numerous combinations with the cortex of the cerebellum and with the gray masses situated in the interior of the cerebellum. The most important of these cerebellar nuclei are the *roof nuclei* (nucleus tegmenti) and the *corpus dentatum s. ciliare*, which is surrounded by a lace-work of closely woven fibres, the *fleece*. The cortex itself contains numerous fibres of varied origin, which are in contact with the nerve cells of the cortex in the well-known form of neuron chains of which the Purkinje's cells are the largest.

Of greatest importance in the recognition of the function of the cerebellum is the knowledge of the formation of the cerebellar peduncles and of their fibre masses, and for this reason a more minute description is necessary.

Origin of the Cerebellar Peduncular Fibres.—*The upper anterior cerebellar peduncles* ("*brachium conjunctivum*" pedunculi cerebelli ad corpus quadrigemini s. cerebrum) connect the cerebellum with the cerebrum (converging upward, with the expanded anterior medullary velum between them). The fibre masses which for the most part form the peduncles are derived from the corpus dentatum, go to the contralateral optic thalamus and are here in connection with the fibres of the posterior part of the parietal brain. Beneath the *red nucleus* of the tegmentum, the principal mass of the fibres cross at about the height of the anterior corpus quadrigeminum with the fibres of the peduncles of the other side (peduncle decussation, "tegmental decussation"), there is therefore a *connection between the cerebellum, the red nucleus and the thalamus of the opposite side and an indirect connection with the contralateral parietal brain* (centripetal part of the cerebello-cerebral reflex arc, see Fig. 48).

The *middle cerebellar peduncles* ("*pontal arms*," pedunculi cerebelli ad pontem), the thickest of the three peduncles, connect the cerebellum with the pons and indirectly with the cerebrum. The pontal arms are formed of fibres which originate centrifugally from the cortex of the frontal brain, as well as from the temporal and occipital lobes, pass through the internal capsule and the crus of the cerebrum,

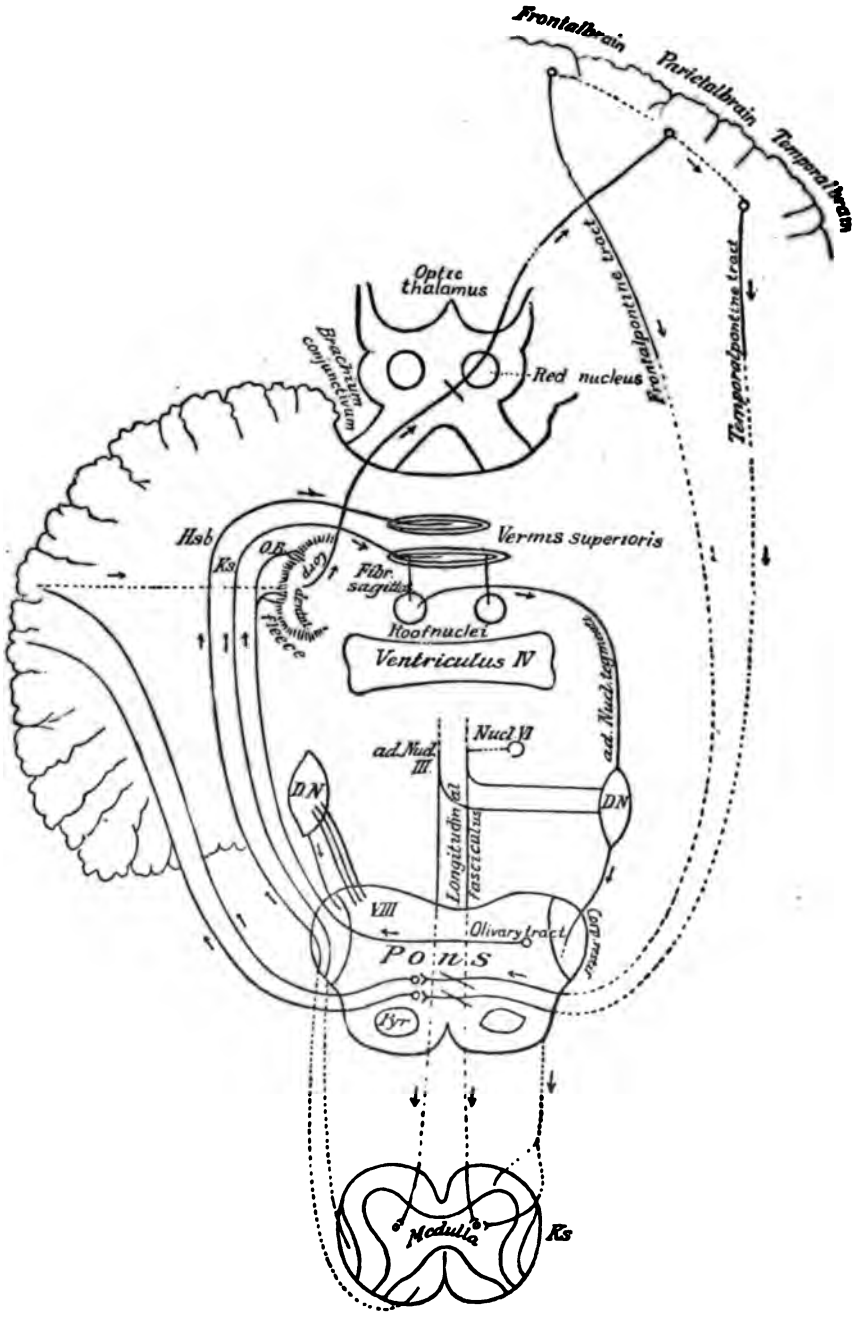


FIG. 48.—DIAGRAM OF CEREBELLAR FIBRES, ESPECIALLY OF THE TRACTS OF CO-ORDINATION.

DK, Deiters's nucleus; *Ks*, cerebellar lateral column tract; *OB*, olivary tract; *Hsb*, posterior column fibre tract.

thence entering the pons. Here they divide for the most part and cross, surrounding the large cells of the so-called pontal nucleus, and give off neurites which go to the cortex of the cerebellar hemispheres, which itself is in connection with the corpus dentatum (centrifugal part of the "cerebello-cerebral reflex arc," see Fig. 48). Other fibres originating in the cerebellum pierce the pontal arms and wend their way towards the tegmentum, ascending dorsally.

The third pair of the cerebellar peduncles finally, the pedunculi ad med. obl., the *corpora restiformia*, bring fibre masses from the spinal cord, respectively from the medulla oblongata, to the cerebellum: 1. As the principal constituent, the fibres of the *cerebellar lateral column tracts*, which, originating in Clarke's column, conduct sensory impression from the regions of the trunk of the body and perhaps also from the intestines to the cerebellum. 2. The fibres of Gowers's bundles (tractus cerebello-spinalis ventrales), which (compare p. 514) do not arise in Clarke's columns, but most probably for the greatest part in cells of the lumbar and sacral cord and which are in connection with the sensory innervation of the lower extremity. 3. Fibres from the contralateral lower olive, the importance of which is not yet understood. 4. Fibres, which originate in the sensory terminal nucleus of the spinal cord and such as originate in the cranial nerves (tractus cerebello-nuclearis). The first originate, in so far as they do not run into the cerebellar lateral column tracts and Gowers's bundles, from the *posterior column nuclei*, in which the fibres of the posterior columns themselves run, and of which a portion enter the corpus restiformia, as fibriæ arciformes posteriores, and may be looked upon as muscle-sense fibres; the latter are fibres from the sensory terminal nucleus of the V., VIII. and X. cranial nerves. Especially important are the relations of the vestibular nerve (VIII) to the cerebellum; its terminal nucleus is the dorsal acoustic nucleus which gives off fibres to the upper olive, to the cerebellum, and to Bechterew's nucleus in the cerebellum itself. Laterally from the latter, Deiters's nucleus is situated which is also in connection with vestibular fibres, from the cells of which neurites travel in various directions, forming important connections. First (see Fig. 48) caudal fibres (centrifugal) go through the restiform body to the spinal cord, to the anterior lateral columns and cells of the anterior horns of the same side. Another fibre tract runs obliquely under the floor of the fourth ventricle to the rhaphe of the medulla oblongata, joins the posterior longitudinal fasciculus, giving off ascending and descending fibres, of which the first are in connection with the nucleus of the abducens, terminating in the nucleus of the oculo-motor, whereas the descending fibres enter the anterior columns of the spinal cord, terminating in the anterior horn cells of both sides. Finally, there is a connection between Deiters's nucleus and the nucleus of the tegmentum, which are themselves connected with the cortex of the superior cerebellar worm by sagittal fibres. This forms a closed reflex arc, the centripetal tracts of which going through the vermis superior cerebelli and by the last-named sagittal fibres terminate in the tegmentum nucleus, from where centrifugal fibres carry the stimulation through Deiters's nucleus, upon various, lately recognised tracts, to the anterior columns and anterior horn cells.

Physiology.—*The functions of the cerebellum* are by no means accurately known. This however may be looked upon as certain that the cerebellum primarily serves the *purposes of co-ordination*. By the anatomical details of the course of the fibres and the connection of the fibres with certain cell groups just described, an insight, at least in certain directions, may be gathered of the complicated process of co-ordination, which unquestionably occurs in the cerebellum. The anatomical basis of this mechanism of co-ordination is formed by two reflex-arc systems (Bruce, Bruns), a *spino-bulbar-cerebellar* reflex arc and one superior to it, the *cerebello-cerebral*. The first is produced by *centripetal* tracts in the posterior columns, in the cerebellar lateral columns, in Gowers's bundles and in the communication of the vestibular nerves with the nucleus of Deiters and the nucleus of the tegmentum. Upon these tracts sensory stimulations travel from the muscles, the joints, the skin, and the semicircular canals of the labyrinth of the ear (the organs of special sense for the equilibrium of the head) to the cerebellum, and are here utilized for correcting the strength of muscular tension, the position of the extremities, of the trunk, of the head, of the eyes, etc., for purposes of co-ordination and especially for the maintenance of equi-

librium, in that the co-ordinating activity of numerous muscles, and the necessary measure of intensity of contraction of the same, may be sent by *centrifugal* tracts. These latter are the connecting fibres which have been described between the vermis superior cerebelli and the tegmental nucleus, between the latter and Deiters's nucleus, and finally between these and the nerves of the muscles of the eye as well as of the cells of the anterior horns of the spinal cord (compare Fig. 48). This spino-cerebral reflex arc which serves the purpose just mentioned, is in communication with another reflex arc which is known as the cerebello-cerebral, which is built up of the former and through which the collected sensory stimulations in the cerebellum are carried to the cerebrum and are translated into correct conditions regarding the position of the body in space, which are then capable, by centrifugal tracts through the temporal and frontal pontal fibres, to exert a controlling action upon the centre of equilibrium in the cerebellum. Whether also with this, if necessary, an action of those stimulations which are to arise in the frontal brain occurs, produced by the will in the motor cortical area in the central convolution and whether or not in the cortex of the brain the intensity of the motor impulses is regulated, is an open question and, as it appears to me, a less important one.

If this intricate apparatus of co-ordination is markedly interfered with or even interrupted at any point, as may readily be perceived, ataxia of a certain intensity and in certain directions must occur, and we will therefore find ataxia as well in diseases of the spinal cord (tabes), as also in affections of the temporal brain and diseases of the optic thalami, and this actually happens. The ataxia will however be most marked if the cerebellum itself, the central situation of the mechanism of co-ordination, is affected, or if it has become incapable of performing its functions. These conditions will be especially considered in the diagnosis of affections of the cerebellum in the following description.

CLINICO-DIAGNOSTIC REMARKS

Diagnostic Symptoms of Cerebellar Disease.—*Diseases of the cerebellum* may run their course *without producing symptoms.* This is an absolutely certain fact and has been determined upon various occasions; it is further true that this is particularly the case if only one hemisphere is affected, whereas the implication of the middle portion of the cerebellum, the worm, as soon as disease affects the greater part of it, gives rise to very peculiar symptoms which permit of a diagnosis of cerebellar disease in by far the greatest number of cases.

Cerebellar Vertigo.—Somewhat constant and characteristic is the *sensation of vertigo* from which patients affected by cerebellar disease suffer. *Usually* it occurs in the following manner: vertigo is usually absent while the body is at rest but appears to a marked degree as soon as the patient attempts to sit up or to walk; the vertigo in cerebellar affections however does not show this condition exclusively. To a great degree cerebellar vertigo is the result of a lesion of some of the fibres of the *vestibular nerve* which, as we have seen, is in connection with Deiters's and the tegmental nuclei and with the nerves which determine the position of the eyes (III and VI). If the vertigo is very marked, an expression of the involuntary attempt to compensate for the lesion of the cerebellum giving rise to disturbances in the sensation of locomotion, actual *constrained movements* may occur.

Constrained Movements.—However, the latter are by no means surely or exclusively due to the marked vertigo, as they may occur during unconsciousness and also in patients who have fully retained their mental

faculties (the usual case) without giving rise to vertigo. One of my patients (the autopsy showed a tumour in the vermis with a noticeable prominence of the right pontal arm) accurately described that, although in walking he had quite a marked sensation of circus movement towards the *right*, he *never* had symptoms of vertigo. If the *constrained movements* occur as forced rotary movements around the axis of the body, they are somewhat characteristic of disease of the *pontal arms*, especially of irritative conditions of the same (fresh hæmorrhages, inflammation, tumours). Diseases of the pontal arms are not as often accompanied with constrained movements, as with the retention of a constrained position, in that the patients retain a certain lateral position and as soon as they are brought into another position involuntarily turn to the old position (with the trunk and also with the head). Other symptoms which are observed in cerebellar affections are *nystagmus* and *pareses of individual ocular muscles*; also a peculiar constrained position of the eyes, in which the one looks downward and outward, the other upward and inward. These conditions have been described by Nonat in diseases of the pedunculi cerebelli ad pontem.

Other Symptoms.—Besides the vertigo (and possibly the well-developed constrained movements in diseases of the pontal arms), a second symptom is usually marked in cerebellar disease—*disturbance in co-ordination.*

Cerebellar Ataxia.—As the vertigo, the symptoms of *cerebellar ataxia* are also found in many other affections of the nervous system; however, the disturbances of co-ordination in diseases of the cerebellum differ somewhat from those which occur in other affections. The gait is swaying, staggering, and has been emphasized quite properly as that of a *drunken person*; the legs are placed wide apart, standing upon one leg or with the legs close together [the feet in juxtaposition] is usually impossible. The equilibrium cannot be maintained. With this, however, closing the eyes need not necessarily increase the swaying (even if it does occur); on the contrary, it may not alter the swaying or *even, as occurred in one of my cases, it may cease entirely.* In the recumbent posture the patient may carry out movements with certainty; muscle sense and sensation, as far as I may gather from the observation of others and my own, are almost entirely intact. The tendon reflexes are normal, occasionally they are absent. In cerebellar affections, ataxia is usually limited to the legs and the trunk, whereas movements in the upper extremity, at least in the majority of cases, may be apparently carried out without any disturbance of co-ordination. The explanation of the presence of ataxia, after what has been described regarding the anatomical structure of the cerebellum and its functions which most probably are due to this, has no difficulty; also the frequent absence of ataxia in cases of cerebellar disease from this standpoint, as soon as the individual case has been carefully analyzed, can be explained in a satisfactory manner. We must remember above all that the symptom of faulty equilibrium of the body in cerebellar affections only occurs, according to our present views, if the lesion affects the vermis in which the co-ordination apparatus is situated; lesions which are limited to the cerebellar hemispheres (with the exception of the corpus dentatum

and the cerebellar-cerebro reflex fibres) run their course without ataxia. Cerebellar ataxia will be all the more marked the greater the area of the co-ordinated reflex arc is affected by the disease in the individual case, especially if, besides the cerebellar spinal fibres, the connections of the nerves of the eye and parts of the cerebellar-cerebro reflex arc are also implicated. Under some circumstances, a tumour which is situated exactly in the middle of the vermis may develop without giving rise to any disturbance of equilibrium, provided it is small and if during its growth the substance of the cerebellum is only gradually compressed without being destroyed, etc. On the other hand, it is not remarkable that an ataxia resembling the cerebellar form should occur in diseases of the pons, the corpora quadrigemina and (according to an observation of Bruns) even in tumours which have their seat in the frontal brain, after it has become likely, from anatomical investigation, that all of these parts of the central nervous system more or less distant from the cerebellum are in direct connection with the functions of co-ordination of the latter.

Choked Disk.—Besides both the above symptoms, the vertigo and the ataxia, stubborn *vomiting* and severe *headache in the posterior part of the head* have been mentioned as characteristic of cerebellar affections. It is quite true that headache and vomiting are usually more marked than in other brain affections. These symptoms are absent however in cases of cerebellar disease which run their course without producing pressure and irritation (as in stationary blood and softening foci) and are not especially characteristic of other affections of the cerebellum; however, the presence of both symptoms is a support to the diagnosis. By all means, they are less valuable in my opinion as symptoms than those which arise as the result of pressure, as lesions which are established in the cerebellum, in their growth partly press upon trunks of cranial nerves which are situated there, partly upon the pons and medulla oblongata, compressing them. This then gives rise to paralyses of individual cranial nerves and of the extremities (simple or alternating hemiplegia) and bulbar symptoms, above all dysarthria, which may also occur as a direct cerebellar symptom, i. e., as inco-ordination of the movements of speech due to cerebellar disease. Also epileptiform attacks, diabetes mellitus, etc., may occasionally in this manner occur in the course of cerebellar affections. By the appearance of these indirect symptoms, being added to the direct phenomena of disease of the cerebellum, the diagnosis gains in certainty if the former by a gradual development are joined to the latter. Finally, in tumours of the cerebellum, occasionally *tremors* and *muscular weakness* occur, symptoms which are of but slight use in the topical diagnosis, and, further, the much more important choked disk; this occurs in cerebellar tumours unquestionably more constantly and earlier in the morbid picture than in the localization of tumours in other areas of the brain and in processes of intracranial increase of pressure in general.

This may be explained upon anatomical grounds in that in tumours of the cerebellum the *vena magna Galeni* is especially exposed to pressure, this favouring the development of a ventricular dropsy. The increased pressure in the subarachnoid fluid is continued to the intervaginal space of the optic nerve which is in connec-

tion with the subarachnoid space and produces arterial anæmia as well as venous stasis with œdematous swelling of the papillæ which may then be looked upon as an expression of increased intracranial pressure. The choked disk according to the course of its development is bilateral, even if it is occasionally more markedly developed in one eye than in the other. There are combined with the stasis papillæ functional disturbances; not rarely, especially in tumours affecting the cerebellum, does sudden blindness occur. The reason for this may be found in the fact that the tumour exerts direct mechanical pressure upon the *aqueduct of Sylvius* resulting in an increase of the hydrocephalus, especially the dropsy of the third ventricle; in that the floor of the latter is forced down, the optic chiasm and the optic nerve tract suffer a local pressure lesion.

Psychical disturbances are absent in the picture of pure cerebellar disease; if they are present, this is no reason, as has been especially emphasized elsewhere, to exclude a cerebellar affection, as lately, in progressive paralysis, degenerative changes have been found in the cortex of the cerebellum.

Upon the basis of the symptoms described we are capable in the majority of cases of correctly diagnosticating diseases of the cerebellum. If this is not always possible, if, especially in this category of diseases, occasionally all diagnostic points of support leave us in the lurch, it must be remembered that our knowledge regarding the course of the fibres, their connection with the ganglion cells in the cerebellum, and especially also regarding the functions of the latter, is still far from perfect. Our clinical observations also must become more exact than is the case up to the present time, and microscopico-anatomical investigations must be carried out by practised investigators or at least be controlled by them if in this realm of diseases diagnosis is to gain more certain points of support.

DIAGNOSIS OF DISEASES OF THE MIDDLE BRAIN (CEREBRAL PEDUNCLES AND CORPORA QUADRIGEMINA)

ANATOMICO-PHYSIOLOGICAL PRELIMINARY REMARKS

Cerebral Peduncles.—The cerebral peduncles which emanate from the pons upward, and which, externally and anteriorly, extend into the hemispheres, are two stalks which connect the posterior and anterior brain. They are composed of two essentially different strata, viz., the ventrally located portion (*foot*) and that part which is located dorsally, the tegmentum (*hood*).

Composition of the Foot of the Cerebral Peduncle.—Both are separated from each other by a strip of dark gray matter, the *substantia nigra* (*Sömmeringii*), which always shows very distinctly on cut surfaces, and which represents an accumulation of exceedingly fine nerve fibres and ganglion cells, the signification of which is as yet unknown. *The white fibres which form the foot of the cerebral peduncle* consist of various fibre tracts which originate in the cerebral cortex, primarily of those of the *pyramidal tract*. The latter is composed of fibre tracts which come from the central convolutions and the paracentral lobes, extend behind the knee of the internal capsule, then enter into the foot of the cerebral peduncles, the *middle third* of which they occupy (see Fig. 49), afterward pass through the pons then to appear as pyramids in the ventral portion of the medulla oblongata and thence to decussate. The pyramidal tract represents the most important motor innervation of trunk and extremities; located near this tract, medially from the same, are the fibre tracts for the cerebral motor nerves which in pons and medulla oblongata turn towards the nuclei. The foot of the cerebral peduncle contains, besides the pyramidal tract and situated bilaterally, the fibres of the pontine tract, which, emanating from the cortex of the frontal lobe (pontine tract of the anterior cerebral cortex) and from the cortex of the temporal, parietal and occipital lobes (pontine tract of the posterior cerebral cortex), extend through the internal capsule, and which, after having passed the cerebral peduncle, end in the pons and in the cerebellum in the manner which was previously discussed (see Fig. 48).

Composition of the Tegmentum of the Cerebral Peduncle Ganglion-Cell Masses.—The *tegmentum* (hood), on the other hand, contains, besides fibres, various masses of ganglion cells: Situated bilaterally, towards the median line, the *red nucleus* (nucleus tegmenti) which is permeated by medullated fibres which, belonging to the brachia conjunctiva (as previously described, p. 619, 621) extend between cerebellum and thalamus. Farther up, already forming part of the middle brain, i. e., in the basal portions of the thalamic region (regio subthalamica), we find, distally besides the red nucleus, a lentil-shaped body, containing nerve cells, the *corpus subthalamicum* (Luys's body), which gradually takes the place of the substantia nigra and (like the latter does farther down) here forms the demarcation towards the foot of the cerebral peduncle.

The Lemniscus.—The main fibre tract of the tegmentum is formed the same as in the posterior brain so also in the middle brain, by the *fillet*, the development and course of which (see Fig. 50) must be briefly explained, because it represents the most important sensory tract.

Mesial "Superior" Lemniscus.—As previously explained (p. 597), the basis of the fillet is formed in the medulla oblongata (by means of the interolivary layer) by the sensory fibres which enter through the posterior spinal-cord roots, after having ascended in the well-known manner in the spinal cord, either having crossed or not. Pontine fibres originating in the cerebellum may possibly also grow towards the fillet; adjacent to the latter we find the "central trigeminus tract" which extends from the cells of the terminal nucleus of the trigeminus towards the brain. The "mesial" fillet, which is composed in the above-described manner, can always be well differentiated and followed to the anterior corpora quadrigemina (see Fig. 50),

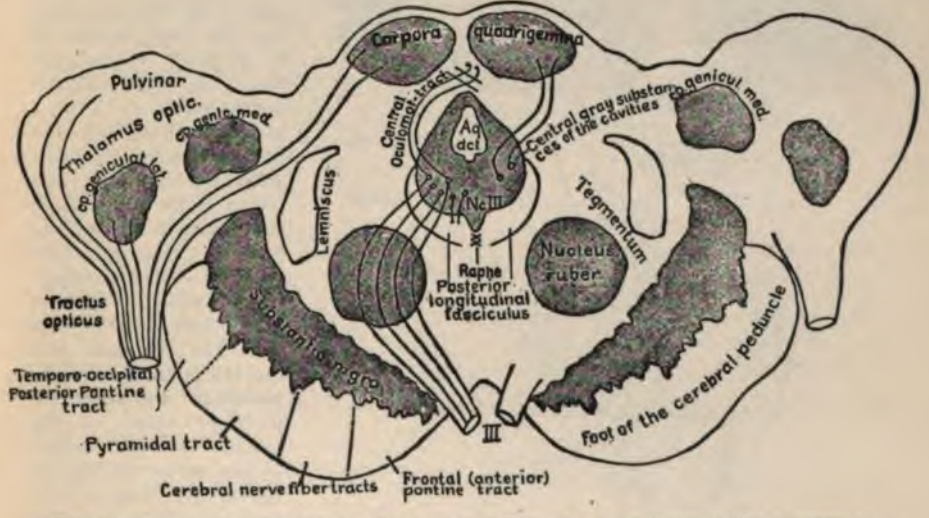


FIG. 49.—SECTION OF THE MIDDLE BRAIN AT THE HEIGHT OF THE CORPORA QUADRIGEMINA. (Diagram, in part, after Obersteiner.)

forming their deeply situated medullated layer. Farther towards the brain we find fibres of the mesial fillet which radiate towards the thalamus (see Fig. 50). The lemniscus fibres which end in the central portion of the thalamus do not continue directly towards the cerebral cortex but are connected with the sensory fibres of the anterior central convolution, so that stimulations which extend centripetally in the mesial fillet, after passing the thalamus cells, reach the cortex of the brain (*cortical fillet tract*, central [tertiary] sensory neuron). The above-described fibre radiation refers to one main portion of the fillet which is designated as "*mesial*," "*superior*," *fillet*.

Lateral, "Inferior," Fillet.—The other main portion of the fillet, the "*lateral*" or "*inferior*" portion (so called because its fibres cannot be followed as high up —into the cerebrum—as the fibres of the superior fillet) commences in the superior half of the pons, partly in ganglion-cell masses (the lateral nucleus of the fillet), which are here located, and in the "superior olive," which is situated a little more caudally at the height of the facial nucleus, and also in the corpus trapezoidus. It is situated more laterally than the mesial fillet, covers the brachium conjunctivum (in this portion of its course it is visible externally) and passes partly decussated into the posterior corpus quadrigeminum; it is, therefore, also called "quadrigeminus fillet" (see Fig. 50). According to what we have explained regarding the course of the

acoustic nerve (p. 600), the *lateral* fillet contains mostly fibres of the acoustic nerve, furthermore tracts from the terminal nuclei of sensory nerves generally, especially from the terminal nuclei of the sensory cerebral nerves.

Corpora Quadrigemina; Aqueduct.—Whereas the above-described structures represent the basis of the midbrain, the cavity of the same is formed by the *aqueduct of Sylvius* connecting the fourth ventricle with the third ventricle, the roof by the *corpora quadrigemina* which abounds in ganglion cells. The *anterior* pair of the corpora quadrigemina (the same as the lateral geniculate ganglion and the pulvinar, see Fig. 49) serves as a primary centre for the optic nerve. Optic-nerve fibres, as is well known, radiate into the anterior corpus quadrigeminum from the optic tract; on the other hand, from cells in the interior of the latter there emerge corona radiata fibres, which (together with those which come from the thalamus and the lateral corpus geniculatum), reaching the posterior third of the interior capsule, extend to the cortex of the occipital cerebrum (*cerebral, Gratiolet's radiation of vision*). The anterior corpus quadrigeminum, besides, as has just been explained, is in connection with the mesial, superior, fillet, and also inferiorly with the nuclei of the nerves of the optic muscles. The *posterior* pair of corpora quadrigemina receives the lateral fillet fibres, especially those of the acoustic nerve, and, on the other hand, by means of fibres, is in connection with the cortex of the temporal lobe.

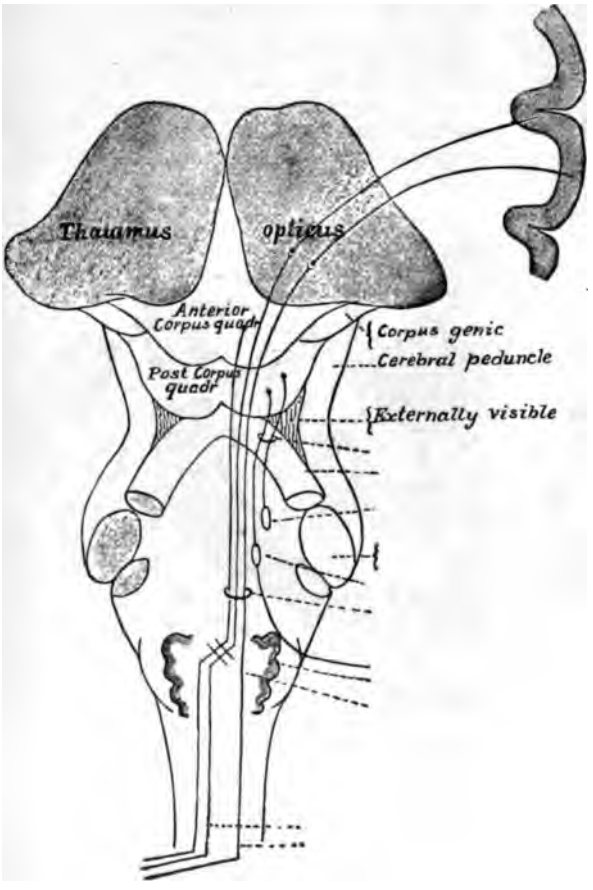


FIG. 50.—DIAGRAM OF THE COURSE OF THE LEMNISCUS. (Mostly after Obersteiner.)

Central Gray Matter of the Cavities.—The *aqueduct of Sylvius* is surrounded by a rather thick layer of gray matter—the *central gray matter of cavities*. The latter extends as a cover of the cavity walls from the soft commissure (commissura mollis) of the third ventricle through the aqueduct of Sylvius to the pyramidal decussation and contains the areas of origin of the cerebral nerves in the middle brain, namely those of the *oculo-motor* and *trochlear nerves*. Downward and externally from the central gray matter of the cavities there is differentiated a system of longitudinally arranged fibres, which stand out distinctly on the cut surface, the "*posterior longitudinal fasciculus*," the meaning of which for processes of association has already been discussed (see p. 603).

Nucleus of the Oculo-motor and Trochlear Nerves.—Regarding especially the arrangement of the *nucleus of the oculo-motor nerve*, it may be stated that it consists of a number of larger and smaller cells which are situated in the gray matter of the cavities around the aqueduct dorsally to the posterior longitudinal fasciculus within the region of the anterior corpora quadrigemina, and which send forth axis cylinders to the trunk of the oculo-motor nerve. A lateral and a medial nucleus can be distinguished. The former is the main nucleus; its cells partly crowd into the posterior lateral fasciculus. The medial nucleus is unpaired, situated in the median line, and its neurites also form those fibres of the oculo-motor nerve trunk which are situated most medially. A part of the fibres of the lateral nucleus decussates, so that the axis cylinders, passing the rhaphe, radiate towards the lateral periphery of the opposite trunk of the oculo-motor nerve. It has been demonstrated by experimental stimulation and by autopsy findings in affections of the region of the oculo-motor

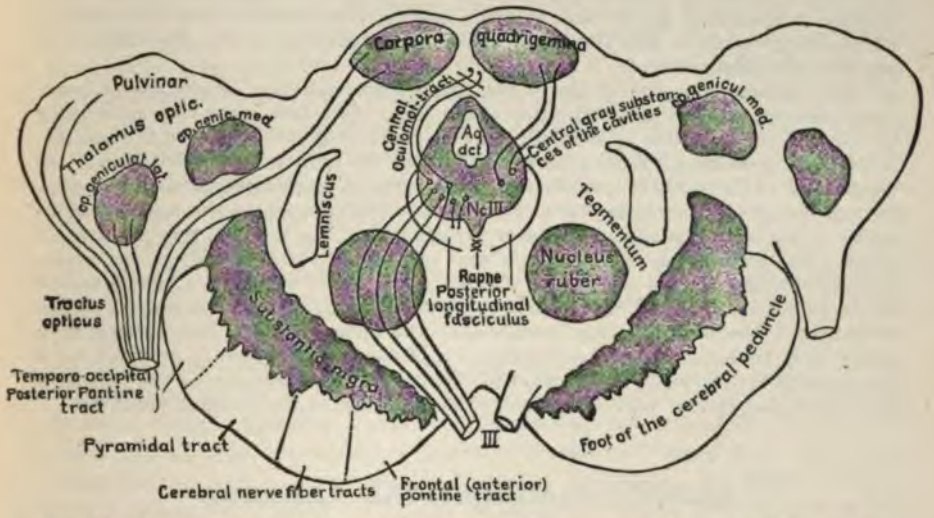


FIG. 51.—SECTION OF THE MIDDLE BRAIN AT THE HEIGHT OF THE ANTERIOR PAIR OF THE CORPORA QUADRIGEMINA. (Diagram, in part, after Obersteiner.)

nerve, that the various muscles which are supplied by the oculo-motor nerve are innervated from various nuclear cells. The cells of the levator palpebræ superioris are probably situated the most frontally and laterally, followed by those of the superior rectus, whereas the cells which supply the inferior rectus should be situated the most caudally. The intervening nuclear section might be intended for the obliquus inferior and for the rectus internus, for they obtain fibres from both nuclei. The medial nucleus, finally, is probably to be considered the point of origin of the fibres for the interior muscles of the eye. The relations of the central oculo-motor neuron are not sufficiently clear as yet. The origin in the cortex, for the levator palpebræ at least, is to be placed in the angular convolution; the transition into the peripheral neuron does not take place until the fibres which emerge from the foot of the cerebral pedunculus have decussated in the rhaphe. An association between the nuclei of the oculo-motor and abducens nerves, i. e., an anatomical basis for the synergy of the muscles which are supplied by both nerves, is brought about by the posterior longitudinal fasciculus.

The *nucleus of the trochlear nerve* presents itself as the continuation of the nucleus of the oculo-motor nerve, which is situated spinally, i. e., more towards the fourth ventricle in the central gray matter of the cavities. The original fibres of the nerve turn from the nucleus at first spinal- and dorsalward, decussate in the velum

medullare anterius and emerge near the brachial conjunctivum, to extend from there around the external surface of the pedunculus cerebri to the base of the brain. Here the trochlear nerve appears close over the pons at the lateral border of the cerebral peduncle, while the trunk of the oculo-motor nerve passes out at the mesial border.

Physiological Facts.—Our knowledge regarding the physiological meaning of the corpora quadrigemina is, upon the whole, very limited. It may be considered demonstrated that some of the fibres of the optic nerve (except those which do not extend into the optic thalamus and lateral geniculate ganglion, see Fig. 51) radiate into the anterior corpus quadrigeminus. It has been demonstrated that, upon degeneration of the optic nerves, besides the pulvinar and the lateral corpus geniculatum, the anterior corpus quadrigeminus also atrophies, whereas the posterior corpus quadrigeminus remains intact. Destruction of the anterior corpus quadrigeminus causes blindness and reflex pupillary rigidity, i. e., the reflex arc between the optic and oculo-motor nerves is interrupted; the latter fact is quite obvious, because the optic portion of the reflex arc is thus injured; besides, connections betwen the anterior corpus quadrigeminus and the nerve nuclei of the muscles of the eyes have been anatomically demonstrated.

Furthermore, the destruction of the corpora quadrigemina causes *ataxia*, especially disturbances in the equilibration of the body, which fact is not very astonishing in view of the radiation of so many sensory fibres into the corpora quadrigemina. Recently it was demonstrated experimentally that the centres for *contractions of the cardia and of the stomach* are situated in the corpora quadrigemina, so that irritation of the latter stimulates them, whereas, after extirpation of the same, apomorphine, which otherwise always produces contractions of the stomach, remains without effect. The cerebral peduncles contain also *vaso-motor* fibres, outside of the above-mentioned tracts for the conduction of sensory and motor stimulation, which extend in the *pedunculus* and the interruption of which naturally causes a suspension or disturbance of the corresponding functions at the opposite side. The cutting of a pedunculus produces contralateral dilatation of the vessels which is preceded, as a symptom of irritation, by contraction of the vessels. *Disturbances of the functions of bladder and rectum* were also observed after injury of the cerebral peduncles.

CLINICO-DIAGNOSTICAL REMARKS

Affections of the Peduncles.—Cases of affections of the middle brain are, in general, rare occurrences; their diagnosis is by no means firmly established as yet, but we are at least in possession of certain points of support which, in some cases, allow of diagnosticating the involvement of the middle brain in the affection.

Foci in the peduncles almost without exception cause contralateral motor paralysis of the extremities, of the facial and hypoglossal nerves. But it is obvious, according to the minutely explained anatomical relations, that the picture of paralytic manifestations must be quite varying according to the degree and extent of the lesion in the given case. Thus, for instance, in circumscribed foci in the foot of a cerebral peduncle, paralysis of the extremities may occur in some cases, without cerebral nerves becoming involved in the paralysis, namely in such cases in which the focus is restricted to the more lateral portion of the middle third of the foot of a cerebral peduncle; in other instances, however, if the middle third is not affected at all, no motor paralysis whatever may be present. Both facts have been observed at the bedside; but such cases obviously always form the exceptions to the rule. For, with the crowding of so many fibre

tracts into a narrow space, as is the case in the peduncles, we may almost always expect not only that the entire motor tracts of one side are affected by the disease, but that, upon the least extension of the focus, other tracts also become affected. These are especially sensory tracts, the loss of function of which causes hemianæsthesia upon the side which is opposite to the diseased area, and, furthermore, those fibre tracts which are intended for vascular innervation and the lesion which produces unilateral vasomotor disturbances. But, of course, the symptoms named so far are not characteristic of peduncular foci, because all of them also occur in affections of other cerebral regions, in which the course of the pyramidal tracts runs together with sensory tracts (as in the posterior portions of the internal capsule). Affections of the peduncles do not assume a diagnostic character until the *oculo-motor* nerve becomes affected by the paralysis. Paralysis of the oculo-motor nerve in such cases, of course, is one which *alternates* with paralysis of the extremities, it is a peripheral paralysis which sometimes may be a *total,* at other times a *partial one.* In the former case the united fibres of the oculo-motor nerve are affected in their course from the nucleus through tegmentum and foot (Fig. 51); or else the trunk itself of the oculo-motor nerve, which appears at the mesial border of the cerebral peduncle, is affected. The latter occurrence may be the consequence of an intrapeduncular tumour, in that the latter forces the trunk of the oculo-motor nerve to one side and presses upon the cerebral peduncle, but it may also be brought about by a basal process which presses upon the trunk of the oculo-motor nerve and upon the cerebral peduncle from below, thus injuring conduction. There are *no* differentio-diagnostical points of support to assume the presence of one or the other process, except, possibly, that, in intrapeduncular processes, the sensory disturbances may eventually be more markedly developed than the motor disturbances, which is impossible in basal processes. In those cases in which the crossed paralysis of the oculo-motor nerve is *partial,* i. e., in which *some* muscles which are supplied by oculo-motor branches, appear *isolatedly* paralyzed, it is the question, as is obvious from the description of the anatomical relations of the region of the oculo-motor nucleus, of an affection of groups of nuclear ganglion cells in the central gray matter of the cavities. They may be found isolatedly, as has been observed on several occasions, so that, for instance, only ptosis appears as a symptom of oculo-motor paralysis. This was present in the following case which came under my observation which, by reason of the above-mentioned diagnostic rules, permitted of the correct diagnosis *intra vitam.*

A Case of Affection of the Peduncle.—The patient, a cook (female), fifty years of age, admitted to the hospital December 15, 1885, healthy until then, about four weeks previously became affected with *headache and vomiting* and a continuous sensation of discomfort in the stomach; disturbances in the discharge of urine were absent, the sleep was poor.

Examination showed the organs of respiration and circulation to be normal, also spleen, liver and stomach. Slight somnolence was present. When asked to move the extremities, she moved only the right hand and the right foot, but not the left extremities. The left facial nerve was not affected. Reflex irritability was increased

on both sides, on the right side rather more than on the left. Athetotic movements occurred occasionally on both sides in the toes which either took place spontaneously or were brought about by touching the sole of the foot. Movements of the neck did not cause any pains; cervical rigidity was not present.

After the somnolence had persisted for several days, consciousness returned on the evening of December 19th, and the patient responded fairly well to questions which were asked. The left arm and the left leg were distinctly paretic; they could only slightly be moved and slowly. Hyperæsthesia of the left arm and leg was present. Both pupils were narrow and rigid with slight ptosis on the right side. All cerebral nerves, except the oculo-motor nerve, were functionally intact, including the trigeminus. The urine, which was scanty, was of normal specific gravity, free from sugar and albumin.

December 20th.—Consciousness was entirely undisturbed on this day; vomiting did not return. The patient was able to move the left extremities a little better than on the previous day; anæsthesia of both extremities (of the left very distinctly) pronounced. Ptosis of the right side was still present, whereas the most painstaking examination for disturbances of the function of the cerebral nerves, except the oculomotor nerve, proved negative. Neither the nerves of the eye muscles nor the fifth, the facial, the acoustic, the vago-accessorius nerves showed any deviation from normal conditions. Speech, also, was entirely undisturbed and no indication could be demonstrated of aphasia or anarthria. The ophthalmoscopical examination, which was made by Professor Michel, gave an absolutely negative result.

December 21st.—The right pupil appeared rather wider than the left on this day. Paresis of the left extremities was less marked than on the previous day, the anæsthesia was also diminished. General cerebral manifestations were absent; consciousness was perfectly clear.

December 24th.—The patient felt quite well, took nourishment and expressed a desire to be out of bed. Paralysis of the left side had receded considerably, also anæsthesia; but, on the other hand, the patient complained, unasked, of an increased sensation of cold in the left side which manifested itself also as easily determinable marked coldness of the left extremities. No ataxic symptoms could be determined in the movements of the patient.

December 26th.—Repeated vomiting again occurred in the course of the day, renewed headache, principally in the right posterior portion of the head. The pulse was strong and slow.

December 27th.—Accompanied with persistent vomiting a somnolent condition recurred, from which the patient could be aroused only by calling her. The left upper extremity was paralyzed to such an extent that the patient could not raise it; at the same time it was anæsthetic. Paresis and anæsthesia of the left lower extremity were less pronounced, and both left extremities were cold to the touch. The tendon reflexes were more marked on the left than on the right side.

December 28th.—The patient could be urged to show the tongue—no deviation of the latter was present. The pupils were unequal, the right one was wider than the left. Ptosis was present on the right side. Deglutition was not interfered with.

December 29th.—Complete unconsciousness was present. The left half of the face possibly was a little more smooth. The left arm was completely paralyzed, not reacting to pin pricks; the same condition to a slightly less degree could be determined in the lower extremity. Vomiting. Towards evening deep coma without twitching occurred during which the patient died.

The temperature had always remained normal, except on December 19th, when it rose to 101.3° F. The frequency of the pulse was increased from December 19th on, 80–100; on December 26th it rose to 105, on the day of her death to 160. The urine was always scanty (200–1000), and up to the end free from sugar and albumin.

The *autopsy showed in the right cerebral peduncle several small hæmorrhagic foci*, one of which affected the nucleus of the oculo-motor nerve (the outermost portion situated laterally above) and evidently was the cause of the right-sided ptosis; another focus was located between the fillet and the foot of the cerebral peduncle and thus had caused hemianæsthesia and hemiparesis of the left half of the body.

The same as in this instance, *vaso-motor* disturbances have been observed also in other cases of peduncular affections. But it has not yet been demonstrated whether we may count on *ataxia* and on *disturbances of the functions of bladder and rectum,* as should be expected, in a certain localization of the peduncular focus.

Finally, as to the diagnosis of *affection of the corpora quadrigemina,* i. e., of foci which are situated more dorsally than those we have just described, it is much less certain than the diagnosis of affections of the cerebral peduncles—primarily because the analysis of symptoms which can be applied diagnostically is essentially based upon tumours of the region of the corpora quadrigemina, the significance of those symptoms, therefore, is greatly restricted as to their value. However, according to the material which is in our possession so far, we are entitled to make the diagnosis of affection of the corpora quadrigemina at least with a degree of great probability.

Affection of the Corpora Quadrigemina.—Affection of the *anterior* pair of corpora quadrigemina is accompanied with suspension of the *pupillary reflex to light* and with *disturbances of vision.* Both occurrences are obvious, according to the anatomical relations (see Fig. 51); however, these symptoms, especially the disturbances of vision, can under all circumstances be made use of in a localizing diagnosis only when, in the case which is to be diagnosticated, no affection of the optic nerve is present in the periphery or when no manifestations of a general intracranial increase of pressure, i. e., choked disk and its sequences, exist. If to the above-named symptoms others are added which point to a focus in the brain, especially in the region of the corpora quadrigemina, primarily *disturbances of the oculo-motor nerve,* unilateral and bilateral paralyses and especially paralyses of isolated muscles which are innervated by the oculo-motor nerve (signs, therefore, which allow of an assumption of a partial paralysis of the nucleus of these nerves), we may presume that the central gray matter of the cavities has become involved in the disease process, and the localization of the latter upon the region of the corpora quadrigemina thus gains in certainty. A further symptom, which was observed in affections of the corpora quadrigemina and which was connected especially with an affection of the *posterior corpora quadrigemina,* is *ataxia.* Its occurrence is easily explained by the relations of the fillet to the corpora quadrigemina; however, the appearance of disturbances of co-ordination is so ambiguous a symptom, which may be caused by affections of the most various tracts and centres in the central nervous system, that it is only of some value in supporting the diagnosis when the signs of paralysis of the nuclei of the oculo-motor and trochlear nerves coincide with the ataxia.

In general, considering the present state of our knowledge, it is advisable under all circumstances not to go any further in the diagnosis of an affection of the corpora quadrigemina than to assume an affection of the *region* of the corpora quadrigemina upon the *coincidence* of the above-named manifestations. *The cardinal point of the diagnosis is always the demonstration of paralyses of the muscles of the eye; if these paralyses are total, it points to the fact that the focus is located more towards the*

pedunculus, whereas upon partial paralysis in the region of the third and fourth nerves we should think of localizing the process in a dorsal direction towards the corpora quadrigemina, which assumption will be supported if disturbances of vision and ataxia manifest themselves at the same time.

AFFECTIONS OF THE ANTERIOR BRAIN

ANATOMICO-PHYSIOLOGICAL INTRODUCTION

Anatomical Remarks.—From the anterior wall of the *anterior cerebral vesicle* of the embryo grows bilaterally the secondary anterior brain in the shape of both hemispheres, which, enlarging considerably, cause the remaining portion of the original anterior brain to appear located posteriorly and interiorly. This latter portion is designated especially as *middle brain* (*optic thalami*). Owing to the fact that the optic thalami emerge from the lateral walls of the vesicle of the middle brain and continue to grow, the cavity (of the cerebral vesicle), which is located between them, becomes, narrowed in a cleft-like manner, the third ventricle of the brain. The base of the middle brain, the floor of the third ventricle (at the base of the brain called tuber cinereum), everts inferiorly into the infundibulum (to the pointed end of which is attached the hypophysis), whereas the roof of the middle brain dorsally is likewise everted as (epiphysis cerebri) pineal body, which is situated between the two thalami immediately in front of the *middle brain* (*corpora quadrigemina*).

As previously stated, the growth of the hemispheres of the brain is disproportionately marked, so that they, curving at the same time posteriorly and inferiorly, almost entirely cover the other cerebral vesicles and their products. It is important that this growth does not occur quite uniformly at all places. Inferiorly and laterally the wall of the anterior brain thickens interiorly into the cavity into a powerful structure, the *corpus striatum* (trunk ganglion). Accordingly, the surface of the brain was slightly restricted in its growth at this place, it has curved inward; this is shown in the form of a deep depression of the surface, the *Sylvian fossa*, and in a portion of brain surface which has become displaced inwardly, the island (see Fig. 52). The trunk ganglion in man, *on the other hand*, is materially retarded in its growth in comparison to the hemispheres, i. e., to the dorsal portion of the wall of the anterior cerebral vesicle (tunica) which increases very markedly in size. The surface of the hemispheres forms numerous folds, the cerebral convolutions, and is covered with the cortical layer which abounds in ganglion cells. Besides the cortical layer a great many fibres develop, which either connect the various near and remote portions of the cortex among themselves ("*association fibres*"), or extend inwardly and inferiorly, i. e., towards the middle brain and that portion of the central nervous system which is situated farther downward (*corona radiata*). The *association fibres* are nerve fibres which, in the growing brain, do not develop until after birth as fibres surrounded by marrow and which serve to connect various portions of the brain cortex and which are regularly used in association processes in thinking, sensation and motion. . Important fibre tracts of the *corona radiata* on their way downward pass the corpus striatum; this occurs in such a manner that the latter is thus separated into several divisions, the internal of which represents the *nucleus caudatus*, the external the *nucleus lentiformis*; the white mass of fibres which passes between both is called *internal capsule*, which, in its course through the corpus striatum, forms two shanks turning off in an angle (knee) anteriorly and posteriorly (see Figs. 52 and 56). All the above-named structures play a prominent part in human pathology.

Of the cavity of the cerebral vesicle there have remained, besides the third ventricle already mentioned, the two cavities of the hemispheres, the lateral ventricles; the communication of the lateral ventricles with the third is brought about by Monro's foramen. If we remove the hemispheres horizontally to both sides at the floor

of the longitudinal cerebral fissure, which divides both hemispheres in their entire length, a broad white mass of marrow is exposed (centrum semiovale), which contains such fibres as extend from the cortex downward, forms the roof of the lateral ventricles and which in the centre shows the principal transverse communication of both hemispheres, the fibres of the *trabecula* (corpus callosum). If we advance anteriorly downward through the trabecula, we reach the sæptum lucidum, the median partition between the ventricles, and more posteriorly the *fornix* which adjoins the sæptum. The fornix, forming the boundary between the anterior brain and the middle brain, rises with its anterior pair of shanks from the corpora albicantia of the thalamus towards the trabecula, to end posteriorly along the border of the hemispheres in the temporal lobe, especially in the lower brain.

Commissures.—Both lateral halves of the brain are also connected, besides by trabecular fibres, by various *commissures*: By the anterior commissure (at the anterior wall of the third ventricle) between the two temporal lobes, by the commissura media and posterior between the two thalami. The commissura media passes midway through the third ventricle and represents a band of gray matter which unites the optic thalami of both sides; the posterior commissure, finally, a gray fibre tract, is situated, as foremost portion of the roof of the middle brain, under the pineal gland, between the latter and the lamina quadrigemina, and is probably connected with fibres of the posterior longitudinal fasciculus.

Corona Radiata Fibre Radiation.—As stated previously, all the fibres which extend downward from the cortex are called *corona radiata*. The fibres radiate from the cortex either into the thalamus or, past the same, into the previously described regions: The pons, cerebellum, spinal cord. The course of these fibres in the brain is of especial importance for the topical diagnosis of affections of the brain.

Thalamus and its Fibres.—The *thalamus* is entered from quite various portions of the brain, especially from the cortex of the frontal, temporal, parietal and occipital lobes, by fibre tracts which, crowded against the optic thalamus, represent the so-called *stalks* of the same. In detail they are fibres from the region of the *anterior central convolution, respectively of the parietal brain*, which end in the ventral nucleus of the thalamus. They represent the *cortical fillet tract*. They join those fibres of the mesial fillet (see p. 627) the course of which was described previously, so that we may regard this entire course of nerve fibres as a structure of sensory neurons. The caudal portion of the thalamus, furthermore, is entered by fibres from the *occipital brain* which, passing under the inferior parietal lobe, radiate especially into the pulvinar and into the lateral corpus geniculatum (*cortical corona radiata*) and are connected with the "primary centres of the optic nerve" which are located here (see Fig. 52). From the *temporal brain*, also, fibres enter through the lower stalk of the thalamus into the latter, after having formed the fillet of the cerebral peduncle jointly with the fillet of the lenticular nucleus. Thus we observe that the thalamus is the terminal location of quite various sensory tracts which either radiate from below from the caudal portions of the central nervous system, or from the latter place through neuron chains extend upward to the cortex. Finally, fibre tracts which originate in the trunk ganglion, in the putamen and in the nucleus caudatus, radiate through the capsule into the thalamus (into the corpora quadrigemina and the substantia nigra) "*radiatio strio-thalamica.*"

Pyramidal Tract, Pontine Fibres.—Of especial importance, because their course and physiological signification have been more minutely investigated, are those fibre tracts of the corona radiata which extend downward past the thalamus to the pons and into the spinal cord. The most important of these fibre tracts is the *pyramidal* tract, the principal route of innervation of the musculatures of the trunk and of the extremities. Its region of origin is situated in the upper portions of the cortex of the central convolutions and of the paracentral lobe. From here it extends to the internal capsule, at which place they become located behind the knee of the capsule (Fig. 52, *p*). From this point they reach the pedal portion of the cerebral peduncle and, farther down, in the well-known manner through pons and medulla oblongata into the spinal cord (after previous decussation into the lateral pyramidal tract, a small portion not crossed into the anterior column, from here, finally, into the anterior horn and into the anterior roots).

Located closely connected with the pyramidal tract are the *central fibre tracts of the motor nerves of the brain* (Fig. 52, *mh*), of which those of the facial and hypoglossal nerves are better known in their course. Both originate, the facial nerve slightly less ventral than the hypoglossal nerve, in the lower portion of the anterior central convolution (see Fig. 55), then pass over the lenticular nucleus and are

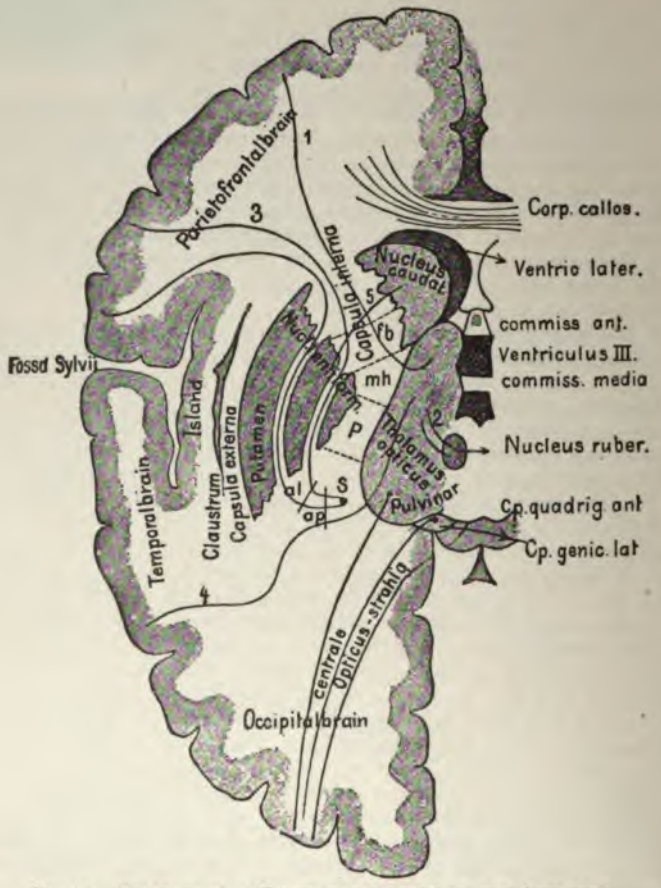


FIG. 52.—DIAGRAM OF A HORIZONTAL SECTION OF THE CEREBRUM.

fb, frontal pontine tract; *mh*, cerebral motor nerve fibres; *p*, pyramidal tract; *s*, region of the sensory tracts; *al*, loops of the lenticular nucleus; *ap* (al + 4), ansa peduncularis, loop of the cerebral peduncles; *1*, frontal thalamus fibres; *2*, fibres from the thalamus to the red nucleus; *3*, lenticular nucleus—tegmental radiation; *4*, temporal thalamus fibres (inferior stalk of the optic thalamus); *5*, fibres from the nucleus caudatus to the nucleus lentiformis.

found in the posterior shank of the interior capsule, near the knee, slightly in front of the fibre tracts of the extremities (pyramids). The following fibre tracts besides the above-named fibres of the corona radiata which pass around the thalamus and extend downward from the cortex, are determined: Fibres which extend from the *cortex of the frontal brain to the pons* (Fig. 52, *fb*) and which end here, respectively radiate farther into the cerebellum. They pass, simultaneously with the above-mentioned fibres which extend from the cortex of the frontal brain to the thalamus (Fig. 52, 1), the *anterior* shank of the internal capsule. The *posterior* shank of the latter also serves, besides the pyramidal tract, as a passageway for other central fibre

tracts, especially for a portion of the *tegmental radiation*, the fibres of which, emerging from the subthalamic region, enter into the most posterior portion of the posterior shank of the internal capsule and radiate from here, either directly or after they have permeated the inner members of the lenticular nucleus (*fillet of the lenticular nucleus*, Fig. 52, *al*), to the parietal lobe and to the posterior central convolution. Associated with the fillet of the lenticulated nucleus extend the formerly mentioned stalks of the optic thalamus, i. e., fibres which extend from the temporal brain to the thalamus. They pass transversely through the internal capsule, as does the fillet of the lenticular nucleus, and jointly with the latter they form the *fillet of the cerebral peduncle* (ansa peduncularis, Fig. 52, *ap*), which embraces the cerebral peduncle. Other fibre tracts in the posterior portion of the internal capsule are: The central fibres of the optic nerve and the central fibres of the acoustic nerve and, finally, fibre tracts which extend from the cortex of the occipital and temporal lobes to the pons (similar to the frontal pontine tract). If we consider, furthermore, that fibres which extend from the nucleus caudatus to the lenticular nucleus, and also the radiatio striothalamica (see above) pass the anterior division of the capsule, it is quite obvious to see the enormous quantity of fibres of very different origin and significance which is crowded into so small a space, also that affections of the internal capsule must be the cause of extensive interruptions of conduction and, according to the seat of the lesion, are bound to be followed by paralyses in the most varied regions of the nervous system.

Surface of the Brain, Convolutions, Sulci.—The *surface of the brain* is distinguished by convolutions and sulci which, although varying in number and form, yet, upon the whole, retain a certain regular type. The most important sulcus is the *fossa Sylvii*, the origin of which has been referred to previously. It separates the *inferior frontal convolution* (third frontal convolution) from the *superior temporal convolution* (first temporal convolution); its upper end is encircled by a convolution of the lower parietal lobe which is called the *supramarginal convolution*. Corresponding to the latter in position and shape another portion of the lower parietal lobe is demarcated farther back, the *angular convolution* which curves around the upper end of the superior temporal sulcus and which is of a more pronounced pathological significance (see Fig. 53).

At about the boundary between the lower and middle thirds of the course of the Sylvian fissure, a short distance from the same, another sulcus commences to ascend almost vertically towards the border of the hemispheres, viz., the *central sulcus* (Rolando's fissure). Adjacent to this sulcus are two of the most important convolutions of the brain: The *anterior* and the *posterior central convolutions*; usually they do not pass into each other until upon the mesial surface of the hemispheres (see Fig. 54) in the *paracentral lobule*. The posterior central convolution passes posteriorly and superiorly without boundary into the superior parietal convolution and the latter, upon the mesial surface of the hemisphere, into the *præcuneus*, which in itself represents the widened posterior upper end of the gyrus fornicatus. Posteriorly upon the mesial surface of the hemispheres the præcuneus is connected with a wedge-shaped lobule the point of which is turned forward, viz., the cuneus, which passes directly into the first occipital gyrus. Figs. 53 and 54 will facilitate the finding of the above-named most important cerebral fissures and convolutions, also of the position of the other details of the surface of the brain which we do not intend to mention. But we shall briefly refer to those facts which have recently become known regarding the finer structure of the cortex of the brain, because they are of importance to judge the physiological and pathological relations and probably will become of still greater significance in the future.

Histology of the Cortex.—The cortex of the cerebrum contains, besides the neuroglia with its cells, a labyrinth of fine nerve fibres and ganglion cells of varying calibre. The largest of the latter, the so-called "large pyramid cells," are not situated immediately below the pia, but in the deeper zones of the cortex; above and below the cortical layer containing them there are smaller ganglion cells which partly are also pyramid-shaped. From all these nerve cells offshoots arise with a characteristic course in a similar manner as has been depicted when discussing the conditions of the nerve cells of the spinal cord, i. e., two kinds of offshoots emerge from

the various cells: Dendrites and the delicate axis-cylinder offshoot ("trunk offshoot"), which in its often extensive course sends forth numerous collaterals. The latter and also the collaterals terminate in arborescent fibre formation, thus further being able to encircle nerve cells and to come in contact with them. The apposition of various neurons (cell—axis-cylinder offshoot—arborescent fibre formation) to one another renders a continuous, far-reaching nerve conduction feasible.

In the cortex of the brain, particularly, dendrites with enormously abundant side branchlets extend from the pyramidal cells to the surface of the cortex as far as

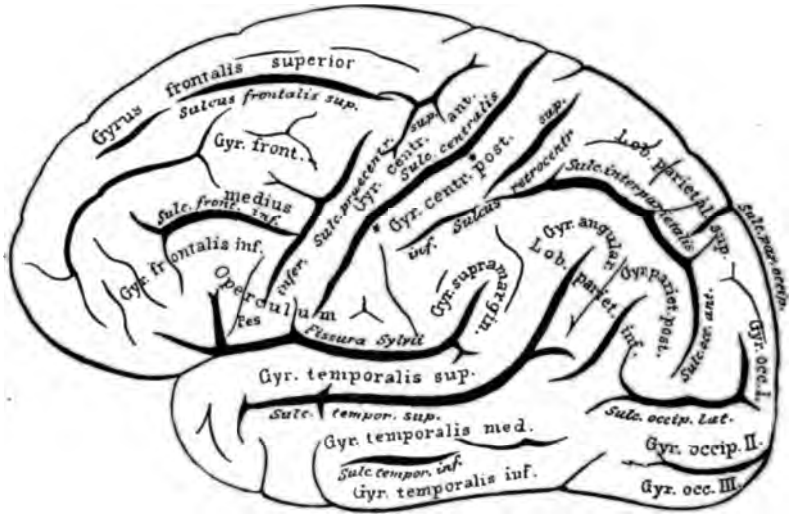


FIG. 53.—LATERAL VIEW OF THE BRAIN WITH CONVOLUTIONS AND FISSURES.

below the pia. The processes of the trunk, on the other hand, extend downward, i. e., towards the medullary layers and by fibre formation come in conducting contact with deep-seated nerve cells which are situated in the central nervous system. The deepest portions of the cortex also contain, besides the above-described ganglion cells, numerous multipolar ganglion cells, the trunk process of which, after a short course, terminates in free fibrillæ in the cortex. The most external zone of the cortex, finally, which is adjacent to the pia, is entered by fibres which are, to a great extent, surrounded by medullary sheaths and which do not originate in the ganglion cells of the cortex, but which must radiate directly from the medullary layer and originate in remote ganglion cells; they may possibly be regarded as terminal organs of sensory fibres. This most external zone of the cortex also contains, besides the last-named fibres and the dendrites of the pyramidal cells, various other ganglion cells, the axis cylinders of which usually run in a tangential direction ("*tangential fibres*"). We see thus that the cortex is distinguished by an enormous number of cell processes and fibre ramifications which come into contact with each other, and it is, therefore, more than probable that the degree of intelligence depends upon the abundance of fibres in the cortex. In fact, according to the most recent investigations we may assume that the cortex of the brain becomes more abundant with fibrés in the course of life, that the fibres, if more frequently brought into use, become covered with marrow and serve as *association tracts* to connect different cortical portions with each other and to combine our impressions among each other and to utilize them mentally. They are destroyed in certain forms of mental disturbance, as has been demonstrated at present, first the tangential fibres and later also the fibres of the deeper layers, and their place is taken by proliferated glia masses. The descending fibres in their entirety form the corona radiata, the specified composition of which has been explicitly described.

Centres of the Special Senses, Association Centres.—The findings of Flechsig, which were obtained recently, are of the greatest importance; according to them, the development of medullary sheaths regularly commences considerably earlier in certain portions of the cortex of the brain than in other cortical regions. Flechsig distinguishes three kinds of regions, according to the time of development: (1) "*Primordial regions*," in which the development of marrow forms even before birth; (2) "*terminal regions*," in which maturing of the fibres does not set in until considerably later, not before the second or fourth month after birth; and

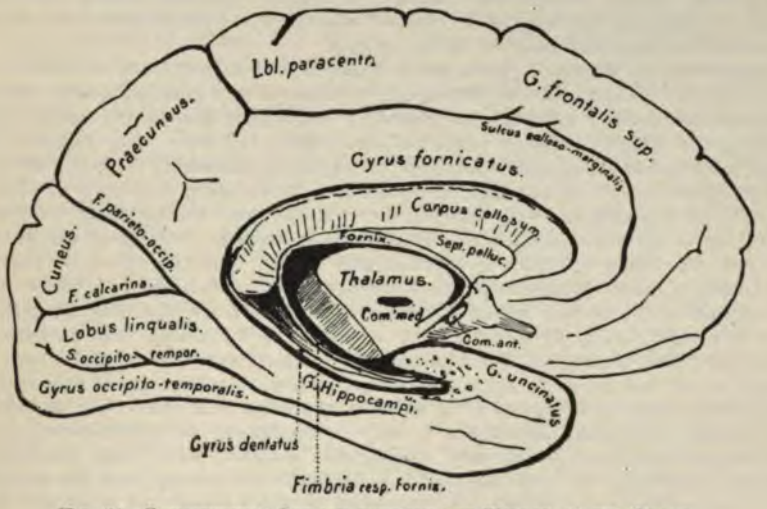


FIG. 54.—LONGITUDINAL SECTION THROUGH THE MIDDLE OF THE BRAIN.

(3) "*intermediary regions*," in which the development of marrow begins during the period between that of the primordial and that of the terminal regions. The *primordial regions* are essentially "*projection centres*," in that from them emerge well-developed corona radiata fibres, which connect the respective portions of the cortex with peripheral terminal organs (*sensory centres*), whereas in the *terminal regions* we must look for "*association centres*" (see below), in which no corona radiata develops or in which projection fibres, which may have isolately developed, are insignificant in comparison to the association fibres. Such *association centres* can surely be assumed in the first and second frontal convolutions, in the lower parietal convolution, the second and third temporal convolutions, the centre-piece of the angular convolution and in a portion of the gyrus fornicatus. *Projection centres*, on the other hand, are: The central convolutions, the first occipital convolution with the cuneus, the uncinate gyrus, the cornu Ammonis, the middle third of the gyrus fornicatus and the centre of the first temporal convolution. To these are added the early maturing portions of the intermediary regions which should also be considered centres of special sense, like the foot of the first frontal convolution, the foot and the orbital portion of the third frontal convolution, the subangular gyrus, etc. Late maturing portions of the intermediary regions are cortical portions which border upon a sensory centre and are connected with the latter by arciform fibres, "the border zones of centres of sense" (Flechsig), which should be counted as association centres, such as the insula, the præcuneus, etc.

Even if this classification of cortical regions may be considerably supplemented and changed in the course of time by further anatomical investigations and clinical experience, it remains the undisputed merit of Flechsig to have opened a new path in this field and to have established a solid foundation.

The mechanism of association is rendered possible by fibres which extend from convolution to convolution, and, furthermore, by long tracts of association fibres

which connect remote cortical regions with each other. Of these we wish to name especially: The inferior longitudinal fasciculus, which connects the temporal lobe with the occipital brain, the superior longitudinal fasciculus, the fibres of which also extend from the temporal lobe to the cortex of the parietal and frontal brains, the fasciculus uncinatus, extending below the insula forward into the most ventral portion of the frontal lobe, and, finally, the numerous trabecular fibres which radiate from one hemisphere into the other.

In spite of the enormous number of cell processes and fibre ramifications, yet *direct* communications of individual cells with others *cannot* be observed with certainty, but, instead, always only contact of separate neurons among each other, so that the general law may be proclaimed that *each individual nerve cell occupies an isolated independent position in the system.*

Physiological Facts.—A great many *physiological experiments* have been made regarding the function of the individual portions of the anterior brain, and still larger is the amount of experience gained at the bedside and at the autopsy table, according to which definitely localized small foci of disease were regularly connected with a loss of certain functions. After the fundamental discovery of Broca, that the affection of a certain area of the surface of the brain, especially of the third left frontal convolution, was followed by aphasia, had undisputably proved the connection of certain functions of the brain with certain districts of the brain, it was first discovered, in 1870, by Fritsch and Hitzig, that the cortex of the brain of animals contains certain circumscribed areas the stimulation of which causes the *contraction* of certain muscle groups. Since this time physiological investigations have not ceased, by experimental irritation or by destruction of individual territories of the brain, to elucidate the physiological function of the latter. Although we do not mean to deny that the results of experiments in this realm of investigation were not always of a uniform character and that their explanation is connected with great difficulties, it may be considered as demonstrated, on the other hand, according to the very considerable material which was furnished by anatomy, physiology and pathology during the last two decades, that the possibility of a localization of the functions of the brain actually exists and that this fundamental idea must always be considered by the clinician in the diagnosis of diseases of the brain. The latter is the more necessary, because simple conclusions, drawn from facts found in the animal upon the function of the human brain, are not quite permissible in this regard, but the foundation of our views regarding the individual functions of the normal brain must always and primarily be obtained from *clinical* observations; but these latter will be the less ambiguous the more they refer to small areas, limited to certain districts of the surface of the brain and which, according to their anatomical nature, preclude any irritation of adjacent districts. We must become aware of the fact that cases which are suitable for unobjectionable conclusions, are great rarities, and we could conclude, accordingly, that the time has not arrived as yet to proceed localizingly in the diagnosis of cerebral affections. This standpoint, however, would be wrong, in my opinion, not only because the respective material already is large enough to permit of a localizing proceeding which can actually be accomplished in a satisfactory manner in the majority of cases, which is shown by autopsies, but also because the endeavour diagnostically to connect losses of function with an affection of certain cortical regions, promotes investigation in general,—it is true, only on condition that the diagnostician, in comparing the result of the autopsy with the symptom-complex as observed *intra vitam*, retains the standpoint of logical conclusions and calm self-criticism. Accordingly, we shall only briefly mention the most important facts of physiological results obtained from animals, but we shall discuss exhaustively that material which was gained by observation of patients and which can be utilized in diagnosis.

Psycho-motor Cortical Areas.—*Electrical and chemical unilateral irritation of certain portions of the denuded surface of the brain causes*, as was first shown by the celebrated experiments of Fritsch and Hitzig, *manifestations of movements in the region of distinctly defined muscle groups of the opposite half of the body.* These regions of the cortex may be regarded as *psycho-motor cortical areas* ("cen-

tres"), i. e., as areas which, in intended movements, are especially intensely stimulated by the will; in the dog they are situated in the primordial convolutions, in the neighbourhood of the fissura cruciata. Lowering of the temperature of the cortex of the brain, narcosis due to the action of chloroform, chloral, alcohol, morphine, etc., great losses of blood, apnœa and asphyxia reduce the irritability of the "centres." Extirpation of the cortex abolishes, after a few, about four, days, the irritability of those fibres of the corona radiata which emerge from the respective area of the cortex, obviously owing to the severing of the fibres from their cortical ganglion cells; for the same reason a secondary, descending degeneration of the respective motor-fibre tract takes place from the injured area of the cortex. On the other hand, the extirpation of those "centres" does *not lead to complete paralysis* of the extremities in animals, but only to a more or less temporary weakness and awkwardness in the use of the same. This fact appears plausible if we assume that, upon a crippling of the customary means of innervation, the difficulties of innervation can be partially equalized by more extensive utilization of the remaining locomotor-fibre tracts, or that the greatest portion of the respective muscular movements takes place only reflexly during the time after the lesion. In organisms, the intelligence of which is more highly developed, in which, therefore, the muscular movements are more exclusively influenced by the will, and in which those reflexly caused movements become subordinate to the voluntary movements, paralyses which are the consequence of cortical destruction, are actually much more intense and more persistent. This is particularly the case in quadrumana and in man; here permanent paralyses may occur after total destruction of some portions of the cortex; and, indeed, in these cases especially those movements are permanently lost which are acquired slowly and which have been permanently placed under the control of the will.

The motor sphere in the surface of the human brain is represented by the two central convolutions and by the paracentral lobule (which probably are supplemented by the posterior portions of the frontal convolutions). Most basal in the central convolutions lies the cortical area of the hypoglossal and facial nerves (see Fig. 55), towards the centre than for the upper extremity and, finally, uppermost and in the paracentral lobe that for the lower extremity. According to the results which were recently obtained, especially by English surgeons, with a weak faradization of the motor sphere of the human brain surface, attempted for diagnostico-therapeutic purposes, it has even become probable that the various cortical innervation districts in man are still much more differentiated, therefore, for instance, isolated movements of the thumb can be caused by a small circumscribed area of the posterior central convolution. *In general, the motor sphere of one half of the brain governs the movements of the opposite half of the body. But this evidently does not apply to those movements which usually or always are executed bilaterally.* Thus it has been demonstrated for the recently discovered centres of the movements of eating and phonation that, upon unilateral irritation of the same, the movements of eating, respectively the adduction of the vocal cords, always occurs bilaterally. If, conversely, a unilateral extirpation is made of the phonation centre which is situated in the most anterior portion of the anterior central convolution at the foot of the third frontal convolution (Fig. 55), i. e., if the cortical region for the adduction of the vocal cords (a cortical region for the abduction is not found as yet) is destroyed, *no* paralysis of the vocal cords takes place, because the centre of the other half of the brain interferes vicariously for the extirpated one.

The conscious, voluntary irritability of the motor tracts is dependent upon the intactness of the cortical centres, whereas, it is true, as is scarcely necessary to emphasize, the motor centres which directly influence the *peripheral* nerves, are located farther down in the central nervous system, i. e., correspond to certain masses of ganglion cells which are interspersed from the middle brain down into the spinal cord. These ganglion-cell masses are superiorly connected with the respective cortical centres by medullary radiation arranged in a certain direction (linking of the central and peripheral motor neurons).

More pronounced irritation of the motor areas of the cortex causes convulsive muscular contractions, in fact actual *epileptic attacks*, and, indeed, the convul-

sions take place first in that portion of the body which, according to experience, belongs to the region of innervation of the respective (irritated) centre, and only then, progressing locally, they pass from centre to centre without omitting any intervening portion. Later on the spasms pass from the contralateral to the other half of the body and here, also, take place in the regular order. If a cortical area is extirpated experimentally, that portion of the body which is innervated by the latter will be passed over by the spasm in an epileptic attack.

Psycho-sensory Areas of the Cortex; Centres of Special Senses.—The investigations of H. Munk and others have taught, furthermore, that, besides cortico-motor,

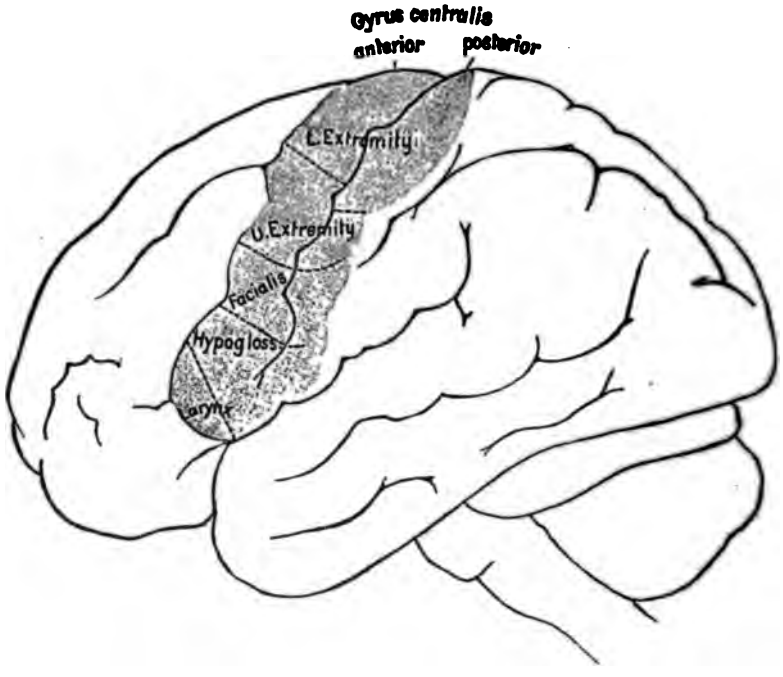


FIG. 55.—MOTOR SPHERE OF THE BRAIN CORTEX IN THE CENTRAL CONVOLUTION WITH THE VARIOUS CORTICAL CENTRES.

cortico-sensory centres should also be assumed to be present in the cortex of the brain. The destruction of cortical areas representing such centres is the cause that an irritation of sensory nerves which originates at the opposite side is no longer observed at all or can no longer be *explained* in the proper manner. If the latter is the case, we speak of "psychical blindness," "psychical deafness," etc.; i. e., the images of optical or acoustic memory have been lost in consequence of cortical lesions, so that the respective impressions of the special senses can no longer be associated with former impressions and thus not be utilized psychically. The *cortical acuity of vision*, connected with the retina and with the primary opticus centres by Gratiolet's radiation of vision, may with certainty be ascribed to the *occipital brain*, and, indeed, the first occipital convolution should be considered as the sense centre of the visual sphere, i. e., as the first place of reception of visual impressions. The *sense centre for impressions of hearing* should be looked for in the first temporal convolution, whereas the localization of the centres for *tactile impressions* and also that of the sensations which bring about the conception of position should be referred to the central convolutions, and portions of the gyrus fornicatus may possibly serve as centres for the sensations of pain.

As **association centres** for these various individual qualities of sense convolutions may be assumed which are adjacent to the respective sense centres ("border zones"), thus for the muscular and tactile senses those regions which border upon the central convolutions; association centres for the sense of hearing are the second and third temporal convolutions, for the sense of vision undoubtedly the second occipital convolution. The fact that the association centres connect the sense impressions among each other, enables the human brain to utilize the latter mentally; their destruction is followed by disturbances of association, and thus alterations and defects of the entire sensory system and of the intelligence may be expected.

It is of some importance pathologically that Landois and Eulenburg have discovered in the cortex of the brain, within the region of the central convolutions, *a thermic centre*, the destruction of which causes a rise of temperature of the contralateral extremities (possibly also an increase of heat production), which persists for days or months, whereas the irritation of this centre is conducive to vascular contraction, lowering of the temperature of the respective extremities and increase of blood pressure. The thermically active vaso-motor fibres which emerge from these centres extend downward through the internal capsule.

The irritation, respectively destruction, of the *corona radiata fibres* which emerge from the above-named centres and which converge downward, causes, according to the size of the affected portion in the medullary layer, more or less extensive irritation or paralysis of the corresponding peripheral innervation district. It will not be necessary for us here to enlarge upon these facts, inasmuch as we refer to the previously described anatomical relations.

Basal Cerebral Ganglia.—But we shall briefly mention the significance of the *basal ganglia of the cerebrum*. The *corpus striatum* serves almost exclusively as a passageway for a very considerable mass of corona radiata fibres which originate in the cortex of the cerebrum and for the greatest portion perforate the corpus striata, in the form of the internal capsule. So soon as the latter is also affected in lesions of the corpus striatum, extensive motor and sensory disturbances of function must be the consequences. The lesion of the lenticular nucleus and of the caudate nucleus itself does not produce any disturbances of function which at present can be positively determined, although there cannot be any doubt, that, from the caudate nucleus as well as from the putamen (similar as from the cortex of the brain), corona radiata fasciculi emerge which permeate the internal capsule and enter into the thalamus, furthermore into the corpus subthalamicum and into the substantia nigra. A lesion of the tegmental fibres (see Fig. 52, 3), which extend to the cortex of the cerebrum and permeate the lenticular nucleus from below up, may possibly cause an alteration of the muscular sense in the contralateral extremities, which has been observed after injuries of the corpora striata. Disturbances of deglutition, which are observed in affections of the corpora striata, especially of the putamen, also render it probable that the last-named structures contain the centre of deglutition.

Our knowledge regarding the function of the *optic thalami* is a little better; it has recently been more elucidated, especially by the extensive experimental investigations of Bechterew. According to the views of the latter author, the optic thalami are centres of movement, by means of which so-called "*reflex movements of expression*" are executed, i. e., movements which, for the expression of sensations and mental emotions, are made by animals even after removal of the hemispheres of the cerebrum. They are more or less completely absent if the optic thalami are destroyed isolatedly, i. e., movements of expression, which are called forth by psychical impulses or effects, and also those which are brought about reflexly by irritation of sensory nerves, will then no longer be produced in any manner whatever. Besides, *choreic movements* sometimes occurred in Bechterew's experiments on dogs after injury of the optic thalami. Whereas the posterior layers of the optic thalami, especially the pulvinar, contain one of the so-called primary *opticus centres* (see above, p. 627), the anterior portion of the thalamus contains, according to Bechterew, a *centre for the movements of the urinary bladder*, which, through a special bundle of fibres, is in connection with the cortex of the cerebrum, and the

irritation of which is said constantly to cause contraction of the bladder. The optic thalami may also contain, according to Bechterew, special *centres for the contraction and relaxation of the small and large intestines.*

CLINICO-DIAGNOSTICAL PRELIMINARY REMARKS

Topico-Diagnostical Points of View of Affections of the Anterior Brain.—If we place ourselves upon the above-described anatomico-physiological basis, and if we also consider, above all, the so far available material of clinical observation and of autopsies, we thus obtain the view-points which must guide us in the topical diagnosis of affections of the cerebrum.

Affection of the Optic Thalami.—In connection with what has just been stated regarding the functional significance of the optic thalami, we mean first to discuss the symptoms which may be utilized in the diagnosis of *affections of the optic thalami.* The *symptoms of motor paralysis,* which are frequently observed in these diseases, *are not the effect of the affection of the optic thalami* proper, but they are due to the action of the same upon the adjacent internal capsule or the cerebral peduncle. The same is the case with *anæsthesias* which occur in these affections as soon as a pressure upon, or an extension of the lesion to, the lowest portion of the internal capsule takes place. However, this explanation of the occurrence of anæsthesia is not sufficient in a small fraction of the cases; in these we must derive the *contralateral anæsthesia directly* from the action of the malady of the thalamus, which fact, in view of the radiation of numerous sensory fibres, especially of fillet fibres, into the optic thalamus (see Fig. 50), cannot meet with any anatomical objections. But these fibres are evidently rarely affected, because in quite a number of diseases of the optic thalami there was no sensory disturbance whatever. If, therefore, unilateral motor paralyses can never, and hemianæsthesia only very rarely, be referred to thalamic foci, and as, in the latter case, the unilateral sensory paralysis is, of course, not at all characteristic of an affection of the optic thalami, yet there exist certain symptoms, the presence of which points directly to such a disorder and renders the diagnosis possible. These are: (1) Peculiar motor-irritation symptoms, which manifest themselves either as *tremor* or as *athetosis* or *hemichorea* and *ataxia*; they have been repeatedly observed in affections of the optic thalami. As choreic movements also occurred sometimes in the test animals upon experimental lesion of the optic thalami, we are, in my opinion, still more entitled to assume a direct connection of such anomalies of movement with affections of the thalamus and also to utilize their presence in the diagnosis of the given case. Of greater importance is (2) the *occurrence of disturbances of vision in the form of contralateral homonymous hemianopia.* This condition may be expected if the lesion affects the posterior third of an optic thalamus and especially the posterior interior portion of the thalamus, i. e., the region of the primary optic centre which is situated in the optic thalamus. But the most important symptom, because pointing directly to an affection of the optic thalami, is (3) *the abolition of the movements of expression on the opposite side of the face upon psychical*

emotions. If the voluntary innervation of the facial nerve is retained in such cases, this condition is in favour of an isolated affection of the thalamus if the latter seems probable in other respects also. It is true, the opposite condition is of much more frequent occurrence: Abolition of the voluntary innervation of the region of the facial nerve, but undisturbed movement of the muscles innervated by the facial nerve upon emotions. Such patients, therefore, can laugh only involuntarily, not, when ordered to do so; this condition is found in the most varying cerebral affections with interruption of the usual tract of the facial nerve innervated by the will, and in such cases we may *exclude* an affection of the facial fibres which govern the involuntary (affective) movements of the face and which are located in the optic thalamus and in the tract of the tegmentum of the cerebral peduncle (see p. 473). *But if, on the other hand, the voluntary and the involuntary laughter are both abolished at the same time,* it is a question of an interruption of conduction of both tracts of the facial nerve by *one* focus or in such a manner that the focus has injured one facial-nerve tract directly, the other indirectly. In future cases of affections of the thalamus we shall have to observe also, in view of the most recent discoveries of Bechterew, the condition of the bladder and intestinal movements.

Foci in the Lenticular Nucleus and in the Caudate Nucleus.—While, accordingly, the diagnosis of affections of the optic thalami is possible under favourable conditions, *we are without any diagnostic points of support in recognising foci which are strictly limited to the component structures of the corpus striatum, the lenticular nucleus and caudate nucleus.* It is true, contralateral hemiplegias have been observed in cases of this character; but they were, if they existed at all (thus especially in processes with acute onset), always of a transitory, indirect nature, so that the diagnostic postulate of Nothnagel: "If after an apoplectic insult permanent hemiplegia remains, the latter cannot be restricted to the lenticular or caudate nucleus alone" under all circumstances remains in force. Contralateral athetosis existed in a case of Landouzy of an exclusive affection of the lenticular nucleus; but as this observation is, according to my knowledge, without any other analogy in the literature, it is, for the time being, of not much use for diagnostic conclusions; we shall later on refer to the significance of athetosis, hemichorea, etc., in the diagnosis of affections of the capsule. In cases of genuine affection of the nuclei in the corpus striatum we must pay particular attention, for reasons previously stated, to eventual alterations of the muscular perception and to disturbances of deglutition.

Foci of the Internal Capsule—Motor Disturbances.—Of incomparably greater importance is the implication of the *internal capsule,* an affection which occurs quite often, which represents the most frequent focal disease of the brain in general and which is usually easily diagnosticable. The most striking and frequent symptom of capsular foci is a contralateral hemiplegia, i. e., the unilateral paralysis of the arm and leg and partly also of the trunk, of the facial and hypoglossal nerves. Regarding the various paralyses it should be stated in detail that *paralysis of the hypoglossal nerve* is limited to a deviation of the tongue towards the side of

the paralysis; permanent slight dysarthria is rarely connected with it. Of the *facial nerve* only the lower branches, i. e., those which supply the mouth and the cheeks, the upper branches of the facial nerve, for the orbicularis palpebrarum and frontalis muscles, remain functionally intact.[1] *Arm and leg are always paralyzed together,* because the fibres of the upper and lower extremities are crowded into so narrow a space in the capsule that an isolated affection of the fibres of one extremity (a monoplegia) will scarcely ever take place. Of course, the occasional occurrence of a monoplegia due to a capsular focus is conceivable, after it has been demonstrated that the fibres of the lower facial nerve, which are adjacent to the fibres of the extremity in the capsule, can be affected isolatedly by a capsular focus, and, likewise, the latter alone without involvement of the fibres of the facial nerve. However, these are all rare occurrences which should be considered only under very exceptional circumstances. A permanent paralysis of the extremities is quite commonly followed, after weeks and months, by a *contracture* of the paralyzed members; a complication of hemiplegia with *hemichorea* or *hemiathetosis* is of much rarer occurrence. As previously stated, these anomalies of movement are found in foci of the optic thalami; but they are also met with in affections of the internal capsule without simultaneous involvement of the optic thalami, but always only when the posterior part of the internal capsule had been affected. If, therefore, we find (post- or pre-hemiplegic) chorea besides hemiplegia, we may think of an affection of the posterior portion of the internal capsule if other symptoms of an affection of this part, especially cerebral hemianæsthesia, to which we shall refer presently, are simultaneously present. Whereas in hemiplegias which occur in consequence of a focus in the internal capsule paralysis of the hypoglossal and facial nerves is very commonly found (the latter almost without exception), the other cerebral motor nerves always remain free from paralysis in this affection; it seems that their cerebral tracts have nothing to do with the fibre tracts in the internal capsule. But it should be understood that the above-named motor disturbances are found only when the focus is located in the anterior half of the posterior shank of the internal capsule.

Sensory Disturbances in Affections of the Capsule.—*Disturbances of sensibility* are often observed in capsular affections. In such cases it is the question, primarily, of a *hemianæsthesia.* The latter may be transitory (as transmitted focal action) or permanent, in which case it forms an important factor for the localization of the focus in a certain part of the capsule, i. e., for the assumption that the posterior third of the posterior shank of the capsule is affected. If we examine more minutely into the character of these persisting hemianæsthesias, we find that the sphere of sensibility of the entire skin has become affected, i. e., face, trunk and extremities and, besides the skin, the mucous membranes are anæsthetic, and,

[1] The dissociation of the fibres of the facial nerve above the facialis root (see p. 600) into a *superior* and an *inferior* facialis tract is an undoubted fact. But the course of the former in the corona radiata is not determined as yet, neither is the cortical centre of the superior facialis nerve ascertained.

furthermore, that the various qualities of perception, viz., pressure, perception of temperature and also muscular perception, are uniformly destroyed. The condition of the *reflex irritability* varies: The *skin reflexes* (especially the cremasteric reflex) are, as a rule, at least diminished or entirely *lost* on the affected side, the *patellar reflex*, on the other hand, is *increased* in all older, but also in quite recent cases—owing to its "reflex inhibitory fibres," the course of which runs with the pyramidal tract. The *higher organs of sense* are also, at least in the majority of cases, implicated in the sensory disturbance, because their central fibres, although separated from the fibres of cutaneous sensibility, yet in close proximity of the latter, extend through the lowest portion of the posterior shank of the capsule: Taste, smell, hearing may be reduced or abolished on the paralyzed side, and homonymous hemiopia (of course, with negative ophthalmoscopical finding) has been observed repeatedly. If the focus affects solely the most posterior parts of the posterior shank of the capsule, it may occur that the motor hemiplegia, which was originally caused by remote action upward besides the hemianæsthesia, disappears and only hemianæsthesia remains permanently.

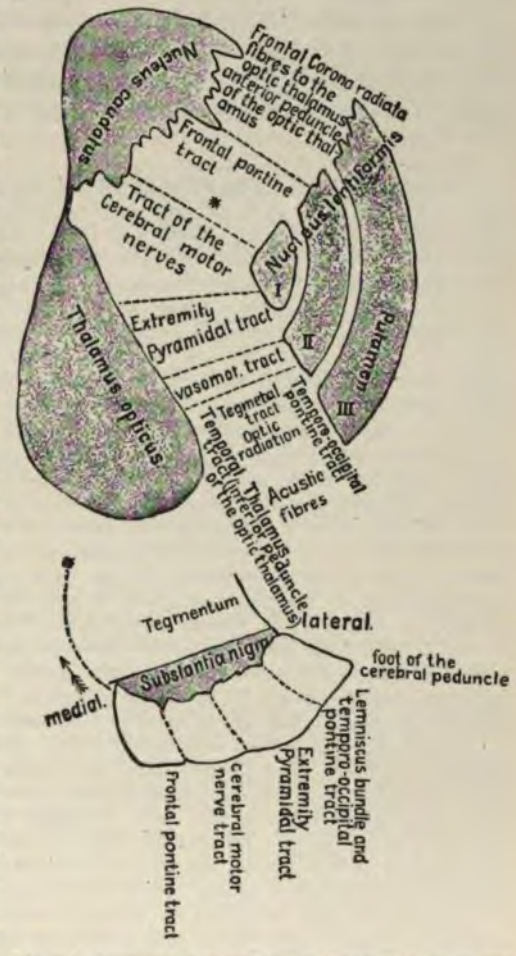


FIG. 56.—POSITION OF THE VARIOUS FIBRE TRACTS IN THE FOOT OF THE CEREBRAL PEDUNCLE AND IN THE INTERNAL CAPSULE (HORIZONTAL SECTION).

Vaso-Motor Disturbances occur in affections of the capsule the same as in foci in the cerebral peduncles and in the pons. They consist, at least in recent cases, in an increase of temperature of 1½° F. on the affected side compared with the healthy side. The skin of the paralyzed members may then be reddened and, especially under the influence of cold, become more rapidly cyanotic, it may also be œdematous and perspire more than that of the healthy side. The above symptoms become, in rare cases, associated with such as are dependent upon a *paralysis of fibres of the cer-*

vical sympathetic nerve: Narrowing of the interpalpebral fissure, receding of the bulb into the orbit and narrowing of the pupils. *Acute (malignant) bedsore* is also, although rarely, observed in affections of the internal capsule. Vaso-motor disturbances are met with in association with hemiplegia, more rarely with hemianæsthesia; in some cases they do not occur at all. The position of the vaso-motor fibres in the internal capsule is not yet quite ascertained (the region of the vaso-motor fibres in Fig. 56 is, in keeping with the usual assumption, drawn in the posterior shank of the internal capsule between the territories of the motor and sensory fibres).

Position of the Various Fibre Tracts in the Internal Capsule.—The above diagram Fig. 56 will aid us in obtaining a clear conception of the position of the various fibre tracts in the internal capsule and their relations to one another. If we start with the fact that the foot of the cerebral peduncle, entering between thalamus and corpus striatum, becomes the internal capsule, we shall have to find again in the latter at first those fibre tracts which constitute the foot of the cerebral peduncle. They are actually found here in the same order as there; only we must assume that the fibre tracts of the pes pedunculi rotate in such a manner that the fibres which are situated most medially in the foot of the cerebral peduncle, turn anteriorly, whereas the lateral ones become the most posterior in the internal capsule. The latter itself is composed of two shanks which meet in the "knee."

The *anterior shank* contains: The frontal tracts to the thalamus (anterior stalk of the optic thalamus) and the frontal pontine tracts.

The *posterior shank* contains: At the knee the central fibres of the lower *facial nerve* and the *fibres of the hypoglossal nerve*; then comes the (extremity) *pyramidal tract* and posteriorly adjacent to the same probably the vaso-motor (and thermic) fibre tracts. Farther posteriorly follows then, approximately corresponding to the posterior third of the posterior shank, a convolution of *sensory fibres* ("carrefour sensitif"), namely: The tegmental radiation, the radiation of vision, fibres of the olfactory nerve and the fibres of the acoustic nerve which extend to the temporal brain. Through the most posterior part of the capsule radiate, furthermore, the temporal corona radiata fibres of the thalamus which form the lower stalk of the optic thalamus (see Fig. 52, 4) and the temporo-occipital pontine tracts.

It goes without saying, considering such a great abundance in the internal capsule of important fibre tracts which are functionally most different, and all of which appear to be situated together within the internal capsule and for the greatest part upon the posterior shank, that, according to the situation of the focus, very varying clinical pictures will result. If, nevertheless, such is not the case, and, on the contrary, affections of the corpus striatum show a certain uniformity of the symptom-complex, it is caused simply by the fact that, owing to the crowding of the fibre tracts, even small foci are bound to injure the various fibre systems *simultaneously*, always, however, in such a manner that in the given case, according to position and size of the focus in the internal capsule, *one* tract appears to be more affected than the other or, in very small foci, one single tract alone may be involved. The diagram of Fig. 56 may be of some help in the topical diagnosis of such cases.

Pathological foci which are situated in the *cortex of the brain* are decidedly rarer than the foci in the corpus striatum. However, since physicians, stimulated principally by the physiological discoveries regarding the function of the various portions of the cortex, have recently devoted an increased amount of interest to affections of this portion of the brain, the number of published cortical foci has become greater and their diagnosis in relatively many cases feasible.

Foci of the Occipital Cortex.—*Cortical foci in the occipital convolutions* do not cause motor paralyses nor convulsions, but *disturbances of vision* have often been observed in foci of the occipital cortex, which is quite conceivable according to the distinct physiological relations of the occipital brain to the process of vision. To conclude from clinical experiences, it appears probable that foci of the occipital cortex in the first occipital convolution and in the cuneus connected with the latter, cause contralateral homonymous *hemiopia* (bilateral lesion of these cortical areas naturally causes total blindness), without any ophthalmological changes being demonstrable at the same time, provided that they are not produced incidentally by certan causes as, for instance, choked disk by a tumour.

If the focus is located in other portions of the occipital brain, especially in the anterior portions of the occipital brain, situated upon the outer surface of the brain (especially in the second occipital convolution), which may be considered as optic-association centres, *psychical blindness* manifests itself (see p. 642), i. e., those processes of consciousness appear to be disturbed which render the *recognition* of subjects possible. *The optic pictures of memory are entirely or partly lost*; it appears, therefore, although *the objects are fully seen, difficult or impossible to explain them correctly with the aid of the sense of vision.*

Foci of the Parietal Cortex.—Foci of the *parietal convolutions* likewise do not in themselves cause motor paralyses; but the parietal brain may be considered the area of reception of numerous sensory impressions, for tactile perceptions as well as for stimulations of muscular perceptions; a connection of the cerebellum through the thalamus with the parietal brain also appears at least probable. Thus it becomes conceivable that hemianæsthesia and disturbances of muscular perception with ataxia depending upon the same, were observed in foci of the parietal brain.

Landouzy and Wernicke have recently maintained that the *inferior parietal convolution* is the centre for conjugate movements of the eyes, the lesion of which is followed by conjugate deviations to the *affected* side as a symptom of paralysis. Wernicke diagnosticated at the time, by reason of this symptom, in a case of softening of the inferior parietal convolution, the seat of the focus correctly, and I succeeded recently in making the same diagnosis in a case of cerebral hæmorrhage in the corpus striatum with extension of the focus to the inferior parietal convolution, in which, besides the hemiplegia, a *constantly persisting* conjugate deviation of the eyes towards the focus and a very pronounced contralateral paralysis of the *superior* branches of the facial nerve existed. It is still questionable whether, to account for the movement of the upper eyelid, a motor area of the cortex, the lesion of which would cause contralateral ptosis, may be considered to be present in the superior parietal lobe.

Foci of the Central Convolutions.—Foci in the *central convolutions* and in the paracentral lobe are the most frequent local affections of the cortex. It may be considered certain at present that *motor* disturbances which originate exclusively in the cerebral cortex, depend upon a lesion of the central convolutions and of the paracentral lobule, and it may be possible that paralyses which are brought about solely by affections of those parts of the cortex, are *permanent* ones, with secondary contractures and secondary degenerations. The paralysis may occur in the form of a com-

mon hemiplegia (contralateral paralysis of both extremities and of the facial nerve). But often it presents itself (and thus the diagnosis of the paralysis as being a cortical paralysis becomes considerably more probable, in fact almost certain) as *monoplegia*, i. e., as isolated paralysis of the facial or hypoglossal nerves, or as facialis-arm paralysis, leg paralysis, or even as an extremity paralysis restricted to isolated muscles. A comparison of the different forms of monoplegia with the respective lesions as found post mortem led to the general result that *the cortical area for the facial (lower branches of the face) and hypoglossal nerves, and probably that of the motor branch of the fifth nerve, is situated in the lower third of the central convolutions (especially of the anterior), that for the upper extremity in the middle third of these convolutions, that for the lower extremity, finally, in the upper third of the anterior, and in the two upper thirds of the posterior, central convolutions and in the paracentral lobe* (see Figs. 53 and 54). It is probable that, besides motor paralysis, *vaso-motor* disturbances are also found more frequently than has been observed until now. At any rate, especial attention should be paid to them, as the area of the "thermic centre" undoubtedly is located within the region of the cortical-motor area. Furthermore, contralateral *disturbances of sensibility* (especially disturbances of muscular perception) are eventually explainable as accompanying manifestations in focal affections of the central convolutions and of the paracentral lobule, because the principal loop terminates in the posterior central convolution (but eventually also in the most anterior adjoining portion of the superior parietal convolution).

If *monoplegias* are observed in a case, the probability is in itself great that an affection of the cortex be present. Indeed, even if the facial nerve is not affected in the picture of a common hemiplegia, we may think of the fact that the paralysis is brought about by a cortical affection, and this is still more the case in an isolated paralysis of the facial nerve of a central character. It is true, both occurrences take place also in foci in the internal capsule and in the pons; in fact, even in foci in the pedunculus it may happen that the facial nerve remains unaffected, although here the nerve tracts for the extremities and for the facial nerve are crowded into the smallest area in the centre of the foot (see Fig. 51), but all these occurrences happen only very rarely. If, moreover, monoplegia of an arm or of a leg takes place, or if one extremity is slightly, the other markedly, paralyzed, the probability of a cortical affection being present becomes considerably greater; in such a case foci in the centrum ovale should really only be considered, besides cortical foci, as we shall see later on. Then the diagnosis of focal affection of the cortex becomes certain if, besides paralyses, still other symptoms are present which in particular point to an involvement of the cortex. These are, primarily, *epileptoid twitchings* in the paralyzed parts. They either precede the paralysis, or they follow later (after weeks and months). The last-named condition is especially conclusive of the existence of cortical foci. These spasms are mostly clonic, more rarely tonic; sometimes, and this is
cially typical, a hemiplegia with an apoplectiform onset is lat

by general epileptiform, spasmodic paroxysms, which originate in the paralyzed muscles; these facts correspond well with the experimental result of artificial irritation of the unilateral motor area (see p. 641). *Consciousness is generally not disturbed in such cases. The intelligence was intact* in cases of isolated affection of the central convolutions, as was distinctly stated in some instances. Another symptom favouring the diagnosis of disease of the cortical motor area is the *complication with cortical* aphasia, inasmuch as the cortical points of a lesion which are to be considered in the origin of this condition, are located in closest proximity to the central convolutions (see below).

Foci in the Temporal Cortex.—*Pathological foci in the cortex of the temporal lobes* cause neither motor nor sensory disturbances. It is certain that the temporal brain is the central station for the perceptions of hearing in general. Destruction of both temporal lobes, respectively of their medullary layer, by tumours causes central deafness, as is proven by a case of Wernicke; but unilateral focal affection in the temporal lobe is not followed by unilateral deafness, so that it appears that the radiation of the fibres of the acoustic nerve does not take place strictly with decussation. It is without doubt, furthermore, that *the (first) superior temporal convolution of the left half of the brain forms the central station for word sounds.* The destruction of this cerebral convolution, i. e., the destruction of the word-sound area, causes a form of aphasia which is designated as "sensory cortical aphasia" or "word deafness."

Foci in the frontal cortex generally do not cause disturbances either of sensibility or of motility. Recently the *pes,* i. e., the posterior portion of the frontal convolution, which is adjacent to the inferior precentral sulcus and to the anterior central convolution, is considered to *belong to the motor area of the cortex.* The remaining frontal brain represents to the greater part, as we have seen, the anterior association centre, an affection of which causes disturbances in the mental utilization of special sense impressions, in thinking. It has been ascertained beyond doubt, furthermore, that the *third left frontal convolution* (adjacent to the fossa of Sylvius) *is in connection with the formation of speech.* It is the immortal merit of Broca to have recognised this fact by reason of clinical observations; his discovery gave rise to the study of disturbances of speech in general, especially of *aphasia.* We have repeatedly mentioned this diagnostically highly important manifestation which so frequently occurs in diseases of the brain, without referring to it in detail. The latter I intend to do in the following chapter and I shall endeavour to treat the subject as briefly and as concisely as possible. But I have convinced myself that a more detailed analysis of the same cannot possibly be avoided in order to understand this complicated subject sufficiently.

DISTURBANCES OF SPEECH—APHASIA, ALEXIA, AGRAPHIA

Aphasia, as is well known, means injury to the process of speech formation which takes place in the brain. Disturbances of speech which are due to injury to the mechanism of articulation, are distinguished from aphasia as *anarthria.* Anarthria is usually the consequence of a paralysis or weakening of the peripheral appa-

ratus of speech, either of the muscular or of the peripheral nervous part (counted from the bulbar nuclei on). But it may also be produced by partial lesion of the articulation cells in the cortex, i. e. of the pyramidal cells in the central convolution, from which emerge the tracts to the cells of the pons-oblongata (*cerebral, cortical anarthria*); such cerebro-cortical anarthrias were observed in recoveries from aphasias and recently also in cases of softening in the region of the central convolution.

Since Broca's discovery it has been attempted continuously, aided by clinical experiences regarding aphasia and its various forms, to divide the process of speech and its disturbances into its individual composing parts and to assign certain areas in the brain to the various functions concerned, as an anatomical basis, and we must say that the question of aphasia at present, at least in several directions, has come to a distinct termination.

The diagnosis of a special form of aphasia in a given case is actually possible only after we have thoroughly grasped the different phases of the process of speech as it takes place in the normal brain. The tracts the passage of which must be supposed in *speaking*, are in themselves complicated; but the analysis becomes still more involved if we, as will be necessary to understand the different forms of aphasia, also take into consideration the processes of *reading* and *writing*.

There is really an enormous difference between the speech of an animal which is expressed by a gesture and an inarticulate cry, partly also that of a new-born child, on the one hand, and the artificially articulated, thoughtful speech of an adult, cultured human being. The latter is the finished product of long-lasting labour of the human brain which is gifted with speech and which possesses the ability to retain the impressions of sense perceptions, to associate the newly obtained stimulation with previously gained impressions and to shape them into a conception, into an idea. The written and spoken word, according to the total development of mankind, under normal circumstances absolutely governs the process of thinking. Gradually the child learns simultaneously how to speak and how to think; the ability of speech attains a certain termination, a relative perfection comparatively early in life, whereas the ability of thought, after it, also, at a certain period of life, has reached an average level of development, is capable of further improvement and refinement as long as life lasts, unless general processes of degeneration take place during senility. The analysis of the various processes of speech and of the formation of conceptions and their mutual relations causes least difficulties if we follow the complicated act from its origin on in a *child* which learns how to speak and how to think. We can imagine the process in such a manner that the child, through its various organs of sense, obtains different impressions of an object in its surroundings and retains them as an image of memory probably in those convolutions (border zones) which are adjacent to the area of reception of sense impressions (the special sense centres). The images of memory gradually become firmly associated among themselves so that the image of hearing regularly stimulates the respective image of vision, etc., and from the sum total of the individual images and conceptions of memory then develops—probably in the central portions of the association areas—the conceptional recognition of the subject in question (see Fig. 62). To this complex of images, and the conception belonging to it, a certain word corresponds, which the child learns from its relatives, educators, etc., as pertaining to those. The same as other impressions of hearing, so probably does the word it hears produce a certain image of sound memory which the child associates with the conceptions and images of memory obtained by the various means of sense perceptions. After this association has become firmly established by practice, the image of word-sound memory probably at once calls forth the imagination of the object in question, and *vice versa*. This process renders the understanding of words possible, and the circle of this process gradually widens, principally also because the stimulus to the formation of conceptions and, indirectly, to sense perceptions from external sources is greatly enhanced by speech. It follows from the above that speech, although not absolutely necessary to logical thinking, yet materially improves the ambiguity and perfection of the latter. *As a matter of fact, the vast majority of human beings think almost exclusively by mental pictures of speech*, i. e., with the images of memory of spoken words, inasmuch as these arise either in

connection with special sense perceptions or without any impulse from external sources, are translated into ideas and give form to abstract thought.

If we trace the different phases of speech (see diagram Fig. 57), we find that the word as it is heard is carried on the tract of the acoustic nerve to the *first temporal convolution* (Fig. 57, *A*), which contains the sense centre for spoken words; the word sounds which are received here, are carried farther, as we may assume, into the lateral border zones of the first temporal convolution and into the second temporal convolution and are retained here as images of word-sound memory. If the word as heard is not to be a mere sound but is to be meaningly understood, it will become necessary to excite from here the conception of the object that corresponds to the word heard. The conception itself is formed by firmly connected associations of individual images of memory, which were deposited at various locations of the cerebral cortex as the product of stimulations of nerves of special sense which are brought about by certain qualities of the object in question: of the acoustic nerve, if it is a sounding object, of the optic, olfactory, tactile nerves and so forth. Thus, probably, a sum total of partial images, which associated among themselves and conjointly complete the conception of the object, is probably formed in the central portions of the various association areas.

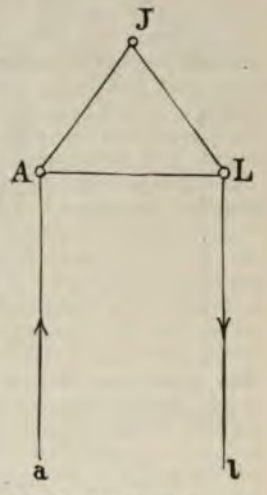


FIG. 57.—DIAGRAM TO EXPLAIN THE PROCESS OF SPEECH.

A, area of word-sound memory; *a A*, tract of the acoustic nerve; *L*, area of the image of speech movement; *L l*, innervation tract of the movement of speech; *J*, sum total of the various areas of conception condensed.

If, then, a conception is to be expressed in words, an association of the areas of conception of the cerebral cortex with cortical parts becomes necessary which, situated in the motor zone, are able to actuate the muscles which are employed in speaking (of the larynx, tongue, pharynx and lips). This is accomplished in the third frontal convolution (Broca's convolution) by translation of the conceptions and word images into the corresponding *images of speech movement*, which, through thousandfold practice, have gradually become absolutely firmly associated with the former, so that the word which was heard or which sounds mentally, brings out the corresponding picture of speech movement. The centre which contains the images of speech movements in the left convolution of Broca is connected with the areas of conception as well as with the areas of word-sound memory, so that, as I assume, *A J L A* (see Fig. 57) forms a tightly closed circle, through the passage of which the relation of the various composing parts of conceivable speech to one another gains a firm hold, a process which may be designated as *control of speech*. Therefore, an interruption of the circle of speech control is bound to be followed by an uncertainty in the conception of the word, respectively by a confusion of words ("*paraphasia*"). From the area of the image of speech movement (L), finally, arises the stimulation of the articulation cells in the region of the central convolutions and from here the innervation of the organs of speech by corona radiata fibres which are in connection with bulbar nuclei of the facial, hypoglossus and pneumogastric nerves (*L l*).

Classification of Aphasia Forms as Sensory and Motor Aphasias.—If we retain the above diagram, it is at once obvious that, if either the above-named collective points or the conduction tracts which connect them with each other, are destroyed by pathological processes, various forms of aphasia must occur which present different symptoms. We are guided best in this chaos if we, in accordance with Wernicke, differentiate two *main groups: Motor aphasias*, which are known for a long time and which are the most frequent, and *sensory aphasias*, the existence of which was first discovered by Wernicke, and the analysis of which has materially elucidated the entire doctrine of aphasia. Each of these groups is again subdivided, and these subforms, according to Wernicke's precedence, have been designated as cortical,

subcortical and transcortical aphasias, according to whether the cortical centres proper (*A* or *L*) or the parts of the tract which are situated on either side of these centres (*a A*, *L l*; *A J J L*) have been destroyed. If the conduction tract is destroyed between the area of word-sound memory and the area of the image of speech movement (*A L*), this condition is designated as "*conduction aphasia*," a name which is not very well selected, which, however, is in general use. It would be more correct, by all means, in the given case to designate this form of aphasia as an aphasia which is brought about especially by interruption of the association tracts between the area of wound sound and that of the image of speech movement.

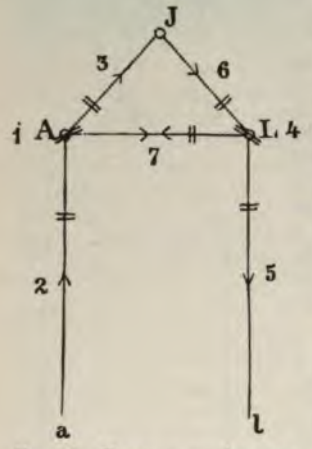


FIG. 58.—DIAGRAM OF APHASIA.

A, area of word-sound memory; *a A*, tract of the acoustic nerve; *J*, area of conception; *L*, area of innervation of movement of speech; *L l*, area of the image of the movement of speech; *A J L A*, circle of control of speech. To repeat words, the tract *a A L l* is used; for voluntary speech, *J L l*; to control speech, *A J L A*; to understand words, *a A J*; = interrupted conduction; *1–3*, sensory forms of aphasia (1, cortical; 2, subcortical; 3, transcortical); *4–6*, motor forms of aphasia (4, cortical; 5, subcortical; 6, transcortical); *7*, aphasia of conduction.

The diagnosis of these different forms of aphasia can be made with sufficient certainty, and in some of the cases it is even possible diagnostically to localize the pathological focus which is the original cause of the individual form of aphasia upon certain districts of the brain.

But it is necessary, before entering upon naming the diagnostic characteristics of the various forms of aphasia, to designate the tracts over which the different acts of the process of speech take place.

The (unintelligent) repetition of words takes place on the tract *a A L l*, *voluntary speech upon the tract J L l*, *control of speech* in the association circle *A J L A*, *the understanding of the spoken word* requires conductivity of the tract *a A J*. With these premises it is easy to examine how the symptomatology of the different forms of aphasia is to be formed and how the differentiation of the same from each other is possible (see Fig. 58).

I. SENSORY APHASIAS

Character of the Various Forms of Aphasia.—1. *Cortical sensory aphasia* (*A* defect) is characterized in that the patient is not able either to understand or to repeat spoken words; but he is able to speak everything spontaneously, without sufficient control of speech, however; he mistakes the words; paraphasia, therefore, is present.

2. *Subcortical sensory aphasia* (*a A* defect): The ability to understand and to repeat spoken words is lost; spontaneous speech is entirely undisturbed, i. e., in contrast to the first form, without paraphasia, because the circle of speech control is undisturbed (genuine "word deafness").

3. *Transcortical sensory aphasia* (*A J* defect): The ability to understand spoken words is lost, but that to repeat words is undisturbed, also spontaneous speech, though eventually with paraphasia.

II. MOTOR APHASIAS

4. *Cortical motor aphasia* (*L* defect): The ability to understand spoken words is intact, but the repetition and spontaneous speech are impossible. The interruption of the control circle manifests itself in that the patients, asked to express by signs the number of syllables of such words as designate objects shown to them (the names of which they are, of course, unable to pronounce), i. e., upon "causing the words to sound mentally," make mistakes ("mental paraphasia").

5. *Subcortical motor aphasia* (*L l* defect): The symptom-complex is the same as in the preceding form, but is distinguished from the same in that the patients are

at all times fully able correctly to name the number of syllables of words designating objects shown to them which they are not able to pronounce.

6. *Transcortical motor aphasia* (*J L* defect): The ability to understand is retained, also the ability to repeat, but spontaneous speech is impossible; mental paraphasia.

III. "CONDUCTION APHASIA" (*A L* DEFECT)

7. The ability to understand is intact in this form and spontaneous speech is also retained, the latter, however, takes place with exquisite paraphasia; the meaningless repetition is also disturbed or impossible.

It is quite obvious from the above explanations that the differentiation of the two original forms of aphasia, (cortical) sensory and motor, and also of conduction aphasia does not present any difficulties. After Wernicke, to whom we are in general indebted for the most important furtherance of the doctrine of aphasia, has taught us how to separate sensory aphasia from motor aphasia and has thus made the most significant step to elucidate the symptomatology and character of aphasia, this differentiation has gradually been acknowledged by physicians the world over: But the question is more difficult to answer whether we are entitled or even obliged also to consider in the diagnosis of aphasia the subforms which were recently established by Wernicke, namely, subcortical and "transcortical" aphasias of a sensory and motor character. There can be no doubt that the conception of "transcortical" aphasia can be maintained only in a limited manner (see below, pp. 658, 659), and that the differentiation of subcortical and cortical sensory and motor aphasia is rather artificial. However, the conclusive or, if I may say so, the logical carrying out of the theoretico-clinical standpoint requires the existence of those subforms in the classification of aphasias. They appear to be more justified if we, to characterize them, also include the disturbances of written language in the range of our diagnostic considerations. The relations of language to reading and writing are so intimate, besides, i. e., are so firmly based upon the manner in which these qualities are acquired that it would not do in cases of aphasia not to investigate whether such patients are afflicted with disturbances in reading or writing. It is true, this will suggest very complicated theoretical questions, and the decision as to what signifies a disturbance in reading and writing is generally still more difficult than the decision regarding the special form of aphasia. It will be necessary in this case, also, before we enter upon the application of disturbances of reading and writing, first to familiarize ourselves with the complicated process of learning and performing the acts of reading and writing under *normal* conditions. The following explanation is intended briefly to answer this purpose.

Analysis of Reading.—We learn how to *read* (as is well known, later than how to talk) in that we conceive optical images of letters or written characters and associate them with the corresponding images of sound and of speech movements which are necessary to pronounce them (on reading aloud). We perform this process always in *spelling*, in that we cause one letter to follow and to associate with the next one in short intervals, which, it is true, in the course of a longer practice in reading, becomes almost inconceivably slight, and also in that, with a still more extensive practice in reading, *groups* of letters are recognised respectively guessed jointly. In such a manner the image of writing, the image of sound and the image of speech movement are obtained step by step, and that in such a manner that they are continually controlled in their formation, i. e., controlling associations continually take place between the individual areas of images. For correct reading, therefore, the tracts (Fig. 59) *O A* and *A L* are of the greatest importance, and the confusion upon the attempt to read, if disturbances of conduction exist in these tracts, is so great that alexia results therefrom. The matter read is understood if the image of writing as acquired in reading, coinciding with a word-sound image that was previously obtained by speech, can be associated with the corresponding conception, if, therefore, the tract *O A J* has remained intact. Consequently, the tract used in loud, comprehensive reading is: *o O A J L l*; in uncomprehended mechanical reading the tract *o O A L l* is used. It may be stated, incidentally, that the

reading of numerals is distinguished from reading of the usual written characters in that the process is not that of spelling. The numerals have simply the meaning, rather, of other optical images of objects and can, therefore, eventually be read without difficulty in otherwise pronounced alexia.

Relations between Alexia and Aphasia.—It follows from the observation of the course of the tract of reading that most of the previously mentioned forms of aphasia also present the symptom of *alexia*, namely, as a glance at the diagram shows, the cortical sensory and cortical motor aphasias, and conduction aphasia; in subcortical motor aphasia reading aloud is at least impossible, reading, whether aloud or otherwise, is done without understanding in transcortical sensory aphasia, whereas alexia is absent in transcortical motor and subcortical sensory aphasias.

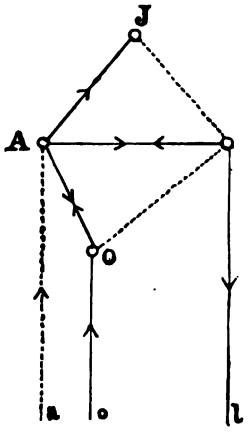


FIG. 59.—DIAGRAM OF READING.

a A, tract of the acoustic nerve; *A*, area of word-sound memory; *J*, area of conception; *L*, tract of the image of speech movement; *L l*, innervation tract of the movement of speech; *o O*, tract of the optic nerve; *O*, area of memory for the optic images of writing.

Analysis of Writing.—More intricate, but of still greater importance for the diagnosis and analysis of the special form of aphasia, are the relations between the ability to write and the latter. To decide the question whether disturbances of writing are present besides those of speech, a very minute knowledge of the tracts in the brain will be necessary, which the process of writing requires under normal conditions; there can be no doubt that it is a very complicated one.

The act of writing is performed in such a manner that we draw a copy of the optical image of memory of the letters of the word (and that, except unusual cases, with the right hand). It is necessary, therefore, that an association exists between the optical images of memory (*O*) and the cells of the central sections of the central convolutions representing movements of the arm (*S*), from which the centrifugally extending nerve tracts emerge to the peripheral nerves of the arm (*S s*). The movements of writing, the same as all co-ordinated movements, are controlled by centripetal kinesthetic stimulations.

The same as in reading, so we proceed by *spelling* when writing (which is a matter of course after what we have explained in the analysis of reading), with a continuous controlling association between the various areas of images of memory, and that not only between the image of writing (O) but simultaneously also between the individual areas of memory of the speech tract, as is represented in the relatively most simple manner by the above diagram (Fig. 60) which was first designed by Wernicke.

The inclusion of the speech tracts is omitted only if we *copy from an original without understanding*. For this purpose the tract *o O S s* is sufficient. *If we write from dictation*, it is a question whether this is done *with* or *without* comprehension of the dictation. In the latter case the tract *a A (L) O S s* is taken with every letter. Until we are well versed in writing and even then, while the word is written and after this has been accomplished, the latter is compared with the word as heard as to its correctness, i. e., the tract *o O* is called into service, in short, the entire reading tract is gone over supplementarily. If we write from dictation *with* an understanding of the latter, besides the above-named tracts the tract *A J* is also used, consequently: *a A J A (L) O S s*. *Spontaneous comprehended writing*, finally, takes place in such a manner that, of course, the entire circle *A J L A* is called into action, whereas, *a A* and *L l* are omitted.

If we now ask in which forms of aphasia the act of writing must be disturbed and in what manner, it will logically follow, according to our views regarding the process of writing, that the mechanical copying from originals is retained in all forms of aphasia; that the unintelligent writing from dictation does not suffer in transcortical aphasias nor in subcortical motor aphasia, whereas the ability of writing from dictation is lost in the other forms of aphasia. The voluntary intelli-

gent writing, finally, is fully intact only in both subcortical forms of aphasia, whereas it is impossible or takes place with at least the commission of mistakes in all the other forms (paragraphia).

Now we may attempt to supplement the table that was outlined previously, containing the symptoms of aphasia, with addition of the disturbances of reading and writing that are present, which, at the first glance, appears to render the diagnosis of the various forms of aphasia more complicated, but which actually renders their more accurate differentiation possible; I call attention only to the fact how easy it is now distinctly to separate the forms 4 and 5, which are very difficult to differentiate diagnostically without reference to the written language. The following table (Fig. 61) renders the results of our explanations in a comprehensive manner, in which + means the retention, — the loss or disturbance of the faculty in question (loss of control of speech means paraphasia).

The forms of aphasia which have been described so far have all been observed clinically, although, it is true, they were not pure, as a rule. After the presence of aphasia has been determined, it remains to be decided in every instance whether the *motor* or the *sensory* form is present, which is not difficult according to the rule given. Not until this has been accomplished, it may eventually be attempted to ascertain the special subdivision of the original form in question. However, often we do not succeed in classifying the given case of aphasia according to the above table, either because *mixed* forms are present (the occurrence of which, as will be explained later on, is easily conceivable according to the anatomical conditions), or because the disturbance of the formation of speech takes place in other manners than those described.

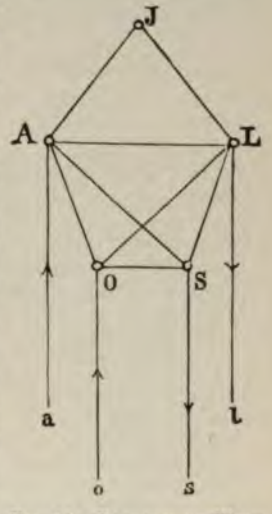


FIG. 60.—DIAGRAM OF WRITING.

A, area of word-sound memory; *J*, area of conception; *L*, area of the image of speech movement; *O*, area of memory for the optic images of writing; *S*, cortical area for the innervation of the movement of writing; *Ss*, subcortical tract for the movement of writing.

Partial and Total "Transcortical" Forms of Aphasia, Optical Aphasia, Amnesic Aphasia.—It should not be forgotten that intelligent speech presupposes the association of the image of word-sound memory with other images of memory which are caused by the various sense perceptions. Thus we see a variety of aphasia in a broader sense occur when those portions of the posterior area of association are injured which border upon the left acoustic sphere. The understanding of the verbal conception is no longer or only incompletely possible in such cases, in that, according to the seat of the lesion, in the parietal brain tactile, in the occipital brain optical, images can no longer be associated with the corresponding images of sound, and retained in their total connection ("tactile" and "optical" aphasias). The connection between optical images of memory and the process of word formation is particularly close; thus it becomes conceivable that, upon a lesion of the cerebral optical centres and conduction tracts (the respective cases were usually accompanied with defects of the field of vision, especially with hemianopsia), the naming of objects which were examined optically only becomes deficient, or that at least the control of word formation is rendered difficult from the visual sphere and the association centre of vision. Such patients are sometimes able to help themselves, i. e., they are able correctly to name objects shown to them if they employ other senses than vision, for instance the tactile sense, when trying to find the word applied to the object in question. Such cases of *optic aphasia* (Freund) have been observed on several occasions; similar to optical aphasia, logically a *gustatory* and a *tactile aphasia* must also exist, i. e., a partial aphasia must manifest itself for certain words, for the formation of which gustatory or tactile perceptions are employed if pronounced disturbances of taste or touch are present. The above-described aphasias come within the range of transcortical aphasias and, according

which, on their part again, receive fibres inferiorly from the fillet, especially, also, from the trigeminus, the pneumogastric and the glosso-pharyngeal nerves. Thus it is conceivable that images of speech movements are called forth in the third frontal convolution which cause the corresponding articulation cells and speech-tract fibres to become stimulated.

The contention, which at first sounded very remarkable, that so complicated an act of the cerebral functions as the formation of speech should be limited to a single cerebral convolution of *one* half of the brain, proved itself to be irrefutably correct by means of a large number of clinical observations and autopsy reports. But it was found in the course of time that there are cases of aphasia which do not answer to a lesion of Broca's convolution. Wernicke especially has found as anatomical substratum of the second original form of aphasia discovered by him, *of sensory cortical aphasia, the posterior portion of the (most superior) first temporal convolution,* while he reserved Broca's convolution for the other, more frequent original form, motor cortical aphasia. The statements of Wernicke have since been fully confirmed by numerous cases. If it is a question of clinically genuine pictures of both fundamental forms of aphasia, *the diagnostician may, without hesitation, assume the third left frontal convolution as being affected in a case of a motor cortical aphasia, and the first left temporal convolution in case of a sensory cortical aphasia,* and we are also entitled, physiologically, to assign to these two convolutions the cortical areas for the memory images of the speech movements, on the one hand, and for those of word sounds, on the other (Fig. 62, *L* and *1*).

Besides these two areas there is another, to which fact attention was called by Naunyn, the third one which may be designated as important for the occurrence of aphasia, namely, the portions of convolutions which are continuous with the angular gyrus, of the second parietal convolution (antero-inferiorly) and of the second temporal convolution (postero-inferiorly), which have been found destroyed in associative alexia (without hemiopia). It may be assumed that this region contains the memory area for optical written characters (see Fig. 62, *O*), and also the association area between the latter and word images, and that lesions of this region are followed not only by alexia, but also produce aphasia in persons who are used to employ optical images in speaking and thinking.

Affections of the insula also cause disturbances of speech, as is proven by autopsy findings. They are probably the anatomical substratum for so-called "conduction aphasias"; for the insula may well be considered as a centre in which associations take place between word images and images of speech movements.

Subcortical Speech Tracts.—We know of but few reliable facts as yet regarding the course of the subcortical motor speech tracts (*L l*) which come forth from those areas of the cortex. There can be no doubt only as to their peripheral terminal points. But it has not been determined as yet what course in detail is taken by the fibres of the tract *L l* between the convolution of Broca and the bulbar nuclei. In my opinion, at first there should be postulated a connection of the speech area in the third frontal convolution with the cortical places of origin of the hypoglossal and facial nerves in the anterior central convolution. But, as glossofacial aphasia is sometimes observed and at other times not found in cortical monoplegia, possibly

Cortical Areas which are employed in the Production of Speech.—Dax, as early as in the thirties of the last century, determined the highly interesting fact that *disturbances of speech coincide with right-sided paralyses*, i. e., *with left-sided affection of the brain.* Apparent deviations from this most important rule, i. e., cases in which aphasia was associated with *right-sided* affection of the brain, are usually explicable in that, in the affected individuals, it is a question of *left-handed* persons, i. e., of human

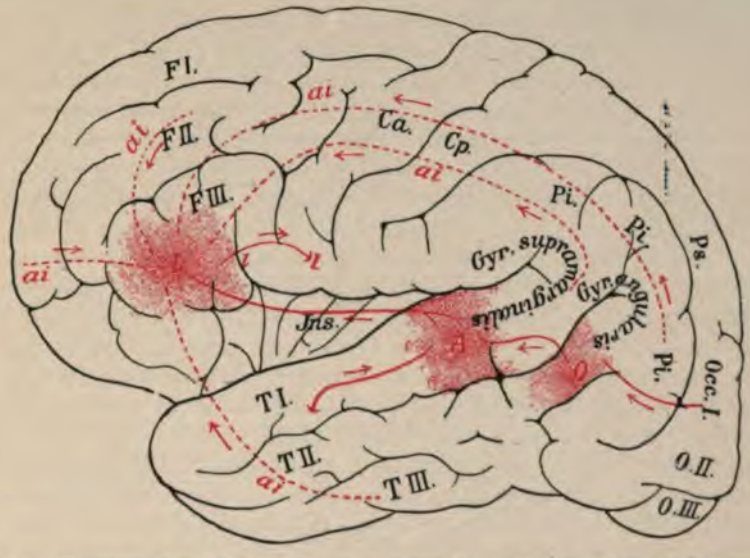


FIG. 62.—DIAGRAM OF THE SPEECH TRACT WITH THE VARIOUS CENTRES OF THE CEREBRAL CORTEX, WHICH ARE CONCERNED IN SPEECH.

L, area of the image of speech movement; *A*, area of word-sound memory; *O*, area of memory for the optic images of writing; *Occ. I, O. II, O. III*, first to third occipital convolutions; *F I, F II*, first and second frontal convolutions, *F III*, third frontal (Broca's) convolution; *T I, T II, T III*, first to third temporal convolutions; *Ca*, anterior central gyrus; *Cp*, posterior central gyrus; *Ps*, superior parietal gyrus; *Pi*, inferior parietal gyrus; *Ins*, island of Reil; *ai*, association tracts between *L* and the central portions of the association areas; *L l*, motor-speech tract.

beings who (contrary to the condition in the vast majority of people) from youth on have used the right hemisphere unilaterally in functions of the brain or who, later, may have been compelled to use the same after having acquired, in the course of their lives, a severe cerebral affection which precluded the full activity of the left hemisphere.

Twenty-five years later, 1861, Broca *localized the ability of speech with absolute certainty in the third left frontal convolution, and he maintained that an affection of the latter causes the occurrence of aphasia.* This opinion was fully confirmed by clinical and anatomical findings which were made subsequently. The third frontal convolution is the junction of the conception areas and of the area of word-sound memory, on the one hand, and, on the other, of sensory stimulations from the mucous membrane of the mouth, pharynx and larynx, in that a portion of the corona radiata fibres of the third frontal convolution is connected with subcortical centres, with the thalamus and with the lenticular nucleus,

which, on their part again, receive fibres inferiorly from the fillet, especially, also, from the trigeminus, the pneumogastric and the glosso-pharyngeal nerves. Thus it is conceivable that images of speech movements are called forth in the third frontal convolution which cause the corresponding articulation cells and speech-tract fibres to become stimulated.

The contention, which at first sounded very remarkable, that so complicated an act of the cerebral functions as the formation of speech should be limited to a single cerebral convolution of *one* half of the brain, proved itself to be irrefutably correct by means of a large number of clinical observations and autopsy reports. But it was found in the course of time that there are cases of aphasia which do not answer to a lesion of Broca's convolution. Wernicke especially has found as anatomical substratum of the second original form of aphasia discovered by him, *of sensory cortical aphasia, the posterior portion of the (most superior) first temporal convolution*, while he reserved Broca's convolution for the other, more frequent original form, motor cortical aphasia. The statements of Wernicke have since been fully confirmed by numerous cases. If it is a question of clinically genuine pictures of both fundamental forms of aphasia, *the diagnostician may, without hesitation, assume the third left frontal convolution as being affected in a case of a motor cortical aphasia, and the first left temporal convolution in case of a sensory cortical aphasia,* and we are also entitled, physiologically, to assign to these two convolutions the cortical areas for the memory images of the speech movements, on the one hand, and for those of word sounds, on the other (Fig. 62, *L* and *A*).

Besides these two areas there is another, to which fact attention was called by Naunyn, the third one which may be designated as important for the occurrence of aphasia, namely, the portions of convolutions which are continuous with the angular gyrus, of the second parietal convolution (antero-inferiorly) and of the second temporal convolution (postero-inferiorly), which have been found destroyed in associative alexia (without hemiopia). It may be assumed that this region contains the memory area for optical written characters (see Fig. 62, *O*), and also the association area between the latter and word images, and that lesions of this region are followed not only by alexia, but also produce aphasia in persons who are used to employ optical images in speaking and thinking.

Affections of the insula also cause disturbances of speech, as is proven by autopsy findings. They are probably the anatomical substratum for so-called "conduction aphasias"; for the insula may well be considered as a centre in which associations take place between word images and images of speech movements.

Subcortical Speech Tracts.—We know of but few reliable facts as yet regarding the course of the subcortical motor speech tracts (*L l*) which come forth from those areas of the cortex. There can be no doubt only as to their peripheral terminal points. But it has not been determined as yet what course in detail is taken by the fibres of the tract *L l* between the convolution of Broca and the bulbar nuclei. In my opinion, at first there should be postulated a connection of the speech area in the third frontal convolution with the cortical places of origin of the hypoglossal and facial nerves in the anterior central convolution. But, as glossofacial aphasia is sometimes observed and at other times not found in cortical monoplegia, possibly

the following presumption is in order: In a tract like the speech tract, which is so frequently used and which so prominently dominates the entire intellectual life of man, it seems to be quite probable that certain ganglion-cell groups in the cortical area of the facial and hypoglossal nerves are used exclusively for this function and are associated with the convolution of Broca, whereas other groups of ganglion cells are independently taken up by other functions of those nerves. To those types of ganglion cells would then correspond also certain corona radiata fibres, and in this modified sense it would be permissible then to speak of a distinct "motor-speech tract" which does not coincide with the usual tract of the hypoglossal and facial nerves. The correctness of this view is also supported by the clinical fact that a pronounced *anarthria* occurred upon destruction of the central convolution with simultaneous exemption of the third frontal convolution. The other possibility that the motor-speech tract originates in ganglion cells *of its own* in the base of the third frontal convolution is less probable, accordingly. Results of autopsies and clinical experiences, furthermore, point to the fact that a centre of articulation exists not only in the left, but also in the *right* hemisphere, and that association fibres probably extend through the corpora striata from the left frontal convolution, respectively central convolution, to the same place of the right hemisphere, the right-sided articulation centre (Kattwinkel). Thus it would become conceivable that disturbances of speech were frequently observed in affections of the right hemisphere alone, i. e., in left-sided hemiplegia, and that this was the case especially when the focus was located in the corpus striatum; in such cases it would, therefore, be a matter of course that subcortical motor aphasia is not found in unilateral affections of the cerebral peduncles.

Ætiological Factors of Aphasia.—If we ask, finally, *regarding the character of cerebral changes which lead to aphasia*, we shall find that the most manifold processes which destroy the above-described speech centres or conduction tracts in the brain or which impede their function, may give rise to aphasia. Thus we observe *aphasia in consequence of hæmorrhages, abscesses, tumours, skull fractures, inflammations of the substance of the brain, of the meninges or of the bones of the skull*, etc., so soon as those processes exert an injurious action directly or indirectly upon the speech tract. But *emboli and thromboses of the artery of the Sylvian fossa*, above all, lead to aphasia, which is quite conceivable if we consider that this artery, besides the corpus striatum and the central convolutions, also supplies the convolution of Broca, the insula and the first temporal convolution with separate branches. If it is a question, therefore, of an embolus of the main trunk (the usual case), the result will be, not a distinct subform of aphasia, but a "mixed" form of this condition, associated, of course, with right-sided hemiplegia. Only upon obstruction by emboli or thrombi of some branches of the artery of the Sylvian fossa and if a sufficient collateral circulation does not develop in the cortex, can we observe, according to one or the other vascular branch being affected, isolated softening of the respective portion of the brain and thus more or less pure, special forms of aphasia. *Transitory aphasias* are not infrequent, the aphasia either being due to transitory circulatory or *functional* disturbances of the brain (as in hysteria, epilepsy, infectious diseases), or it forms a symptom of short duration, a remote action of focal affections of the brain (of apoplexies, etc.), or, finally—such cases were also observed—it disappears, after having lasted for some time, with vicarious interference of the right hemisphere. All these ætiological factors must be considered in the diagnosis of aphasia.

After this digression we may return to what remains to be discussed regarding the topical diagnosis of diseases of the anterior brain.

Foci in the Centrum Ovale.—*Focal affections in the centrum ovale* are not very frequent, generally, and quite varying as to their manifestations, according to the seat of the focus, i. e., according to whether it occupies more the posterior, anterior, or the various central portions of the white medullary substance or whether it is situated more towards the cortex or

the basal ganglia. Except the association fibres of the cortex and of the trabecular radiation, the fibre masses of the medullary layer converge in all directions from the cortex towards the thalamus and the internal capsule. It is scarcely necessary to mention that, consequently, the fibres of the corona radiata extend in a more crowded manner inferiorly, and thus it becomes obvious that a comparatively small focus, which is situated in the centrum ovale or in the neighbourhood of the capsule, comes into simultaneous contact with more fibres than if it were situated near the cortex; manifestations caused by the same will, therefore, cause in one case more the picture of an *affection* of the *capsule*, in other cases again that of a *cortical implication*. *But in the latter case the epileptoid convulsions, which are so characteristic of affections of the cortex, are, almost without exception, absent;* they are observed only when the area of the medullary substance is situated immediately under the cortex and irritates the latter also. On the other hand, in foci of the centrum ovale which are situated towards the cortex, we often find *monoplegias* and *aphasia* as soon as the corona radiata fibres of those cortical portions are affected which represent the central areas for the motor-innervation tract or for the speech tract, i. e., therefore, if the focus occupies the medullary area which corresponds to the central convolutions or to the cortical region of speech. *In this case the subcortical form of aphasia occurs, an important symptom for the diagnosis of foci in the medullary substance. Disturbances of sensibility* have been rarely observed in foci in the centrum ovale; they are most liable to be noted in lesions of the white medullary substance under the parietal lobes and the central convolutions. But *in any variety of a more pronounced hemianæsthesia* we should, as a rule, not think of a focus in the centrum ovale but of one in the posterior portion of the internal capsule. We are still more justified in doing so if the hemianæsthesia is associated with disturbances of vision and hearing on the same side. For, while the fibres of cutaneous sensibility and of the higher organs of sense in the "carrefour sensitif" of the internal capsule are crowded into a narrow space and are especially liable to be injured, they radiate very divergently when passing into the centrum ovale; then it may be possible that an affection of the white substance of the occipital lobe may cause hemianopsia, but no longer, unless it extends far beyond the occipital lobe, at the same time hemianæsthesia. The occurrence in the clinical picture, finally, of *psychical disturbances* in cases in which there may be a question of focal disease, is generally in favour of a focus in the cortex with its association centres. *However, psychical alterations may, nevertheless, be possible also in foci in the centrum ovale if they are situated towards the cortex and if they have destroyed association fibres.*

The diagnosis of foci in the centrum ovale is, accordingly, *a very uncertain one; we are entitled to make it at least with a certain degree of probability if the clinical picture of the cerebral affection in question differs in certain (above-discussed) respects from the usual picture of a cortical affection, on the one hand, and of a capsular affection, on the other.* A differential diagnosis is less possible, the more the location of the focus in the centrum ovale approaches the region of the basal ganglia. It may

be emphasized, finally, that foci in the centrum ovale may sometimes occur without any symptoms and may be met with at the autopsy as accidental findings.

Focal Diseases remaining Latent in Other Parts of the Brain.—According to the clinical material in our possession it will be impossible to outline a symptom-complex which corresponds to affections of other portions of the brain than those mentioned. Therefore, foci in the *cornu ammonis, corpus callosum, claustrum, external capsule, etc.*, are, for the present, not subject to diagnosis. It is true, hæmorrhages into the external capsule occur comparatively often, but they usually continue from here to the corpus striatum, respectively to the internal capsule; if they remain restricted to the external capsule, the symptoms connected with the same are to be explained essentially as remote actions upon the neighbourhood, the corpus striatum and the insula.

Profuse hæmorrhages into the ventricles do not cause any characteristic local symptoms either. Usually they are accompanied with coma, primary contracture or general resolution of the limbs and rapidly occurring fatal termination. It may be possible, sometimes, to utilize these symptoms also, which, however, are by no means specific, in the diagnosis. In cases in which a hæmorrhage into the substance of the brain or an abscess of the brain has been diagnosticated, it is permissible to assume the possibility of a supplementary perforation into the ventricles if a supervening severe insult is accompanied with coma and general resolution or convulsions and rapidly leads to a fatal termination. But even in this condition of the symptom-picture there cannot be any question of anything else but a probable diagnosis.

DIAGNOSIS OF THE VARIOUS FOCAL AFFECTIONS OF THE ANTERIOR BRAIN

CEREBRAL HÆMORRHAGE—SANGUINEOUS APOPLEXY—HÆMORRHAGIA CEREBRI

CEREBRAL hæmorrhages, except those which occur in connection with traumatism and the hæmorrhagic diathesis, are principally caused, according to recent experiences, by *luetic and atheromatous changes of the wall of the cerebral arteries,* owing to which they lose their elasticity, and rupture.

Symptom of the Insult.—The immediate effect of such a hæmorrhage is the so-called *apoplectic insult* (paralytic stroke, apoplexy); the affected person *loses consciousness more or less rapidly,* falls, becomes comatose, and the *entire voluntary movements* and *conscious sensations are lost,* in severe insults all *reflex movements* also; urine and fæces are passed involuntarily, in other cases there exists retention of the urine. The *urine* may, if examined after the attack, contain albumin and sugar, and be voided in larger quantities; however, these urinary changes persist only for a short while, usually not longer than a day (see below). The *face is congested,* hot to the touch, the carotid arteries pulsate markedly; *respiration* is sometimes retarded and, owing to the paralysis of the soft palate, snoring, in other cases it may be of the Cheyne-Stokes type. The cheeks expand during expiration (relaxation of the buccinator muscle); a tracheal râle arises when the saliva is swallowed and enters the trachea. The *pulse* is full, tense, retarded, the *temperature of the body* is abnormally low, later normal; in cases with an unfavourable termination a preagonal increase of temperature occurs. The head and the eyes are frequently forcibly turned to one side, to which fact Prevost was the first to call attention, they are in "*conjugate deviation,*" and that towards the non-paralyzed side, so that the patient in a manner "looks towards the lesion in the brain." The *pupils* show a varying condition, they are sometimes dilated or narrowed, at other times unequal, sometimes reacting to light, at other times not. In some cases during the coma, in place of the usual immovability and relaxation of the extremities, tonic rigidity or even convulsions occur, the latter especially when an extensive hæmorrhage or even a perforation of the extravasation of blood into the ventricles has occurred and the effects of the compression thus becomes more marked.

Explanation of the Insult and its Consequences.—The insult generally occurs in the affected persons during the best of health; sometimes it is preceded by *premonitory signs*, viz., headache, vertigo, a feeling of weakness, tinnitus aurium, muscæ volitantes, etc., or even indications of paralyses, aphasia, etc., and the manifestations of the insult sometimes reach their fullest extent at once, at others they develop very gradually in the course of hours and days in a very much retarded measure.

Regarding the *cause of the insult in general, it is the consequence of a sudden increase of intracranial pressure and of the deficient blood supply to the brain caused thereby, of the adiæmorrhysis* (R. Geigel), which, in this case, is brought about by compression of the smallest arteries, veins and capillaries.

It is at once conceivable that, with this sudden change of the circulatory conditions in the brain, the entire functions of the same will become deficient and, with it, voluntary movements and sensibility *in toto*, consciousness, is lost. It will be considerably more difficult to explain the *condition of the reflexes*; we might expect that, with the absence of function of the brain, i. e., after the interruption of the influence of the will and of the reflex inhibitory tracts upon one side, the reflexes should take place more easily and more regularly. But this is not the case; on the contrary, during the time of deep coma the reflexes are entirely abolished, possibly because in so severe a disturbance of the cerebral functions as is caused by an apoplectic insult, *any* activity of the nerves, and with it the conduction of reflexes, is lost. So soon as, with the disappearance of the effect of the insult, the reflexes are liberated, the removal of the reflex inhibition which originates in the brain, becomes evident—the tendon reflexes of the healthy side are moderately, those of the paralyzed side *considerably, increased*; on the other hand, the *cutaneous reflexes* are *diminished* both during the time of the stroke and afterward. Especially the *cremasteric*, respectively the *abdominal muscle reflexes* are absent on the paralyzed side, which fact may sometimes be utilized, in unconscious patients, to determine for the time being whether and upon which side the hemiplegia is located. The cause of this remarkable difference in the condition of the tendon and cutaneous reflexes may possibly be due to the fact that the arcs of the cutaneous reflexes, in contrast to the tendon reflex arcs, are situated more superiorly and are directly or indirectly injured in their continuity by the cerebral disease in question.

The *slowing of the respiration* during the insult finds its experimental analogy in the slowing of the respiration upon artificial pressure upon the cerebrum or upon electrical irritation of its surface; the occurrence of *Cheyne-Stokes respiration* can be easily explained by the extreme exhaustion of the respiratory centres by adiæmorrhysis. Recent observations have shown that the centre for *conjugate deviation of the eyes* is situated in the inferior parietal lobule; its lesion causes the well-known deviation of the eyes towards the side of the focus. It is restricted solely to the time of the insult in the greatest majority of cases, and it disappears with the decrease of the severe symptoms in a few days. The symptom very rarely reaches a certain independence, so that the conjugate deviation of the head and eyes persists for weeks and months with a relatively small degree of the general manifestations. Except the last-named, exceptional symptoms, which point to a local lesion of the parietal brain, the manifestation should be considered an "indirect" symptom of the cerebral lesion. For this reason alone it is transitory; moreover, it is transitory even when it should be due to a focus in the inferior parietal lobule. This is to be explained, according to Wernicke, in that the conjugate deviation of the eyes is not under the limited influence of one hemisphere, but also under the influence of the other, so that, therefore, the centre of the healthy hemisphere usually, unless especial obstacles (disturbance of consciousness, etc.) are present, after a short while interferes vicariously and causes the unilateral symptom of paralysis to disappear. The lowering of the *temperature of the body* (probably due to an irritation of the thermic centre) is likewise an indirect symptom, depending upon the increase of intracranial pressure, the same as the tense condition and the retardation of the *pulse* (the latter to be referred to an irritation of the cardio-pneumogastric centre, the former to an irritation of the vaso-motor centre). Of further, usually very transitory, remote actions upon the medulla oblongata there occur *albuminuria*,

polyuria **and** ***melituria.*** **All these symptoms point to a more marked degree and a more considerable extension of the pressure effect, so that they should be considered partly as prognostically suspicious manifestations.**

If a fatal termination does not occur in the apoplectic coma within a few hours or days, now, after a decrease of the dangerous general symptoms, those special manifestations become prominent which must be considered as the proper consequences of the extravasation of blood, *focal symptoms of the cerebral hæmorrhage.*

Focal Symptoms of the Cerebral Hæmorrhage—Transitory, "Indirect" Focal Symptoms.—Among these *focal symptoms* proper we must distinguish between *transitory* ("indirect") and *permanent* ("direct") symptoms. The former do not depend upon an actual destruction of the substance of the brain with its fibres and ganglion cells, but upon the effect which the hæmorrhage exerts upon its neighbourhood. They refer, therefore, to functional disturbances of a portion of the hemisphere which was affected by the hæmorrhage; it is rare that, during this later stage, any of the symptoms manifest themselves which were due to the pressure which originally acted upon the entire brain. A strict differentiation between transitory and permanent focal symptoms is possible only by observation of the further course of the affection and of the later loss of function (*Ausfallssymptome*). *But it is possible, even at an earlier period, at least to draw a probable conclusion which symptoms are most likely to remain permanently and which are most apt to recede.*

The picture of focal symptoms in most cases presents itself typically, in the form of a *hemiplegia,* which, however, even after the most severe insult, may recede entirely. This is the case if the hemiplegia is only the expression of the effect of the hæmorrhage upon the adjacent main conduction tracts of motility and sensibility. If, on the other hand, the latter themselves are affected by the hæmorrhage, the hemiplegia will be a permanent one. The reason why hemiplegia occurs with a certain uniformity in by far the majority of cases of cerebral hæmorrhage is to be looked for in the fact that the hæmorrhage takes place quite conspicuously, often into the region of the cerebral ganglia (namely certainly in two thirds to three fourths of the cases if those are counted in which the extravasation is not restricted to the ganglia only, but also destroys the surrounding medullary layer), and, furthermore, in that the nerve-fibre tracts which pass through the ganglia of the cerebrum and which functionate exclusively unilaterally, react with pronounced paralysis upon any injury, even if it takes place from the neighbourhood.

It is impossible exactly to determine in advance from what period of time on the focal symptoms may be regarded as permanent, direct ones, as the transitory focal symptoms recede, sometimes more rapidly, at other times less so, in the individual case. *But it can be considered as certain that what has not receded of focal symptoms after several (three to six) months, will remain a permanent manifestation of absence of function,* although I must emphasize particularly, according to my experience, that slight improvements in the intensity of the paralyses may occur even after a very long time.

It is impossible to outline a clinical picture which answers *all* cases and corresponds to the time of indirect focal symptoms. Nevertheless, certain typical symptoms are almost always predominant, primarily, as has been previously stated, by *hemiplegia.* The latter is *contralateral* and extends over the greatest portion of the muscles of the affected half of the body. Taking special part in the paralysis are the *arm* and the *leg,* which, upon passive elevation, fall back as flaccid masses. The patellar tendon reflex of the paralyzed leg is increased, the cremasteric reflex of the paralyzed side is lessened or abolished; furthermore, the reflex which was recently described by Babinski (bending of the toes, especially of the big toe, upon irritation of the sole of the foot), is said to be altered in hemiplegias in such a manner that an extension of the toes takes place instead of the normal bending. Besides paralysis of the extremities it is particularly the unilateral paralysis of the facial nerve, and that of its lower branches, which becomes prominent; the mouth is placed obliquely, it is turned down on the affected side; the respective nasolabial fold is obliterated, and the soft palate of the affected side sometimes stands lower, etc., whereas the region of the upper facial nerve apparently remains unaffected by the paralysis. However, upon a close examination we find almost always indications of paralysis also in the movements of the frontalis, corrugator and orbicularis palpebrarum muscles.

The *affective reflex movements of expression* are not interfered with, as a rule, in spite of paralysis of the voluntary movement of the facial muscles. It is rare that a synchronous loss of the voluntary and of the affective movements is observed, and still more rarely we note a loss of the affective reflex movements of expression. The latter occurrence may be looked for only when the hæmorrhagic focus does not touch the *facialis pyramidal tract,* but only the optic thalamus, or if it injures that tract which extends to the tegmental radiation of the cerebral peduncle and of the pons and which causes mimic movements (see pp. 472, 473 and 644).

Besides the facial nerve, usually a portion of the fibres of the hypoglossal nerve is paralyzed also: The tongue deviates when protruded, and that in such a manner that, owing to the effect of the one non-paralyzed genioglossus muscle, the point of the tongue deviates towards the affected side. The *articulation* is rarely markedly disturbed, in spite of the fact that the facial and hypoglossal nerves are involved in the paralysis; very pronounced dysarthria which becomes prominent after the insult, always points to an unusual seat of the hæmorrhage, especially in the pons-oblongata. The *musculature of the trunk,* also, is quite usually affected by the contralateral hemiplegia: The shoulder, owing to paralysis of the trapezius muscle, slightly droops on the affected side, and the paralyzed half of the thorax lags in its movements upon very deep inspiration. If pneumonia develops in the course of the affection, it usually localizes in the lung of the paralyzed side.

Sensibility is also disturbed in most cases in the form of a *hemianæsthesia* which uniformly affects the perceptions of touch, temperature and pain. It is usually but little pronounced and disappears after a few weeks, whereas the simultaneously present, indirect motor hemiplegia eventually persists for months. Hemianæsthesia rarely presents a marked degree; then we must expect that it will turn

out to be a direct focal symptom. In isolated cases it may also occur that, besides the unilateral disturbance of cutaneous perception, the *muscular sensibility* is also altered; the patients have lost the conception as to the position and attitude of their limbs. However, such disturbances of muscular sensibility are in themselves not a sign of the cortical character of the focal affection, they may be nothing but an indirect symptom, caused by remote action on the part of the hæmorrhage upon the central convolutions or upon the centripetal tracts which transmit the muscular sense. *Unilateral disturbances of vision* (hemiopia) occur as an indirect or as a direct focal symptom, caused by transitory or permanent lesion of the cortical radiation of vision. There is rarely an opportunity to determine transitory disturbances of smell, taste or hearing, which follow the apoplectic insult.

Vaso-motor and trophic disturbances are also observed in the stage of indirect focal symptoms. The most interesting, most severe, but rare, affection of this kind is the (*malignant*) bedsore of the buttock of the affected side, which was first closely observed by Charcot. Very rare is, furthermore, the inflammatory swelling of the joints of the affected side, accompanied with pains, which was also demonstrated by Charcot. The temperature of the paralyzed members is first increased, the palpebral fissure and the pupil are narrowed, the bulb sunken.

One of the relatively frequent indirect focal symptoms is *aphasia*, both the motor and the sensory forms; of course, it is much oftener found in left-sided hæmorrhage and may then become a permanent focal symptom; in right-sided hæmorrhage sometimes a very transitory aphasia is observed, which is present only in the first days after the attack and which is to be explained as a sign of diminished pressure action of the apoplectic focus upon the left hemisphere.

Permanent "Direct" Focal Symptoms.—A great portion (in fact, in rare cases all) of the above-named sequelæ of cerebral hæmorrhage disappears in the further course of the affection, i. e., after months; another part remains and then forms the *permanent "direct" focal symptoms.* Whereas from the transitory focal symptoms it can be concluded with certainty only which hemisphere is the seat of the hæmorrhage, and while the observation of the severity of the insult as well as the weighing of the relative intensity of the different indirect focal symptoms allows at most of a probable conclusion as to the lesion of a certain area of the affected hemisphere, *the certainty of the topical diagnosis only commences with the stage of permanent focal symptoms.* The diagnosis is to be made in the given case according to previously (pp. 643–651) discussed view-points which are determining of the localization of the focus in the cerebrum. We do not intend again to enter into details here; only a few important facts will be mentioned as a guide and to supplement the diagnostic rules previously given.

Exempted Areas in the Paralyzed Region.—It has been explained in the description of indirect focal symptoms that the hemiplegic paralysis does not extend uniformly over all innervation areas of the affected (paralyzed) half of the body; and this applies still more to direct focal symptoms, in which *this inequality of the paralysis* becomes markedly prominent. *Thus the arm appears more paralyzed than the leg, and, in the arm again, the movements of the hand are more impaired than those of the arm in its entirety.* Furthermore, the facial nerve is completely paralyzed only in its lower branches, the hypoglossal nerve only partially nonfunctionating; the paralysis of the musculatures of the neck and trunk, also, is always decidedly less marked than the paralyses of the extremities, etc.

This, at first glance remarkable, more and less pronounced participation or exemption of various innervation areas in the paralysis can be explained either by directly demonstrable, more marked degeneration of the fibres intended for various portions of the body, or by a hypothesis which was first established by Broadbent. According to the latter, the exclusive innervation from *one* hemisphere applies only to those groups of muscles which can be designated as acting most arbitrarily, the movements of which are finely graduated and which usually take place isolatedly unilaterally, not synchronously combined with those of the other side. Accordingly, in unilateral interruption of conduction they will be especially affected by the paralysis, whereas others, as, for instance, the muscles of the neck and trunk of one side, cannot only (because usually moved on both sides simultaneously) be innervated from one (the opposite) hemisphere, but also, although less markedly, from the other. *Thus it happens that such muscles, which act symmetrically on both halves of the body, are paralyzed only very temporarily, never permanently, by unilateral foci*, simply because the intact hemisphere interferes vicariously. This also applies, besides to the *muscles of the neck and trunk* already mentioned, especially to the greatest part *of the muscles which are innervated from the facial and hypoglossal* nerves, and to the *muscles of the eyes which rotate them laterally*, the paralysis of which from *one* hemisphere manifests itself in the, *always transitory*, character of the "conjugate deviation." If later, after such paralyses have become equalized by the interference of the activity of the intact hemisphere, in the latter a focus appears which is located symmetrically with the former focus, then a complete *bilateral* paralysis will take place. It is obvious that such cases, which are highly interesting theoretically, are rare; but there can be no doubt as to their occurrence; I have recently had an opportunity clinically to observe a demonstrative case of this kind.

Broadbent's hypothesis also harmonizes with the fact that, in recent hemiplegias in those motor regions which are subject to the influence of both hemispheres, disturbances of movement manifest themselves not only upon the one side opposite to the focus, but also upon the same side. According to what we have explained, they will be more prominent in the *leg* than in the arm, thus, for instance, in a hæmorrhage in the left hemisphere, not only the right arm and the right leg will be paralyzed, but the left arm and, above all, the left leg will also show a decrease of strength.

Associated Movements.—Some of the *associated movements* which sometimes occur in hemiplegias, should, in my opinion, be explained by the innervation of various muscles from both hemispheres. Associated movements are involuntary movements which are brought about by transmission of the irritation of motor tracts upon other motor tracts. Thus we see, for instance, that a patient who is afflicted with hemiplegia, upon being asked to move the paralyzed hand, moves the latter a little or not at all, but, without intending to do so, he moves the unaffected hand—or that, *vice versa*, upon voluntary movement of muscles o. the healthy side, muscles of the affected side also become active. Furthermore, it may happen that, instead of the intended movement of muscles of the extremities, other muscles, for instance those of the face, contract, or the extensors upon an intended flexor movement. It is also possible that upon reflex movements other motions are brought about, i. e., reflex movements on tracts which are situated outside of the usually employed reflex arcs. If we look for an explanation of this remarkable manifestation, we may be allowed to assume, besides innervation of the muscles from both hemispheres, that, in patients with hemiplegia, inhibitions in the central nervous system, especially in the crus cerebri and spinal cord, are abolished, and that the occurrence of involuntary movements on the affected and on the healthy sides is thus facilitated.

Hemiplegia a Direct Focal Symptom.—Owing to the frequency of their occurrence, we wish further to emphasize particularly that motor hemiplegia is a very complete one in *hæmorrhages into the internal capsule*, that, according to the hæmorrhage affecting the upper or lower portion

of the posterior shank of the capsule, motor hemiplegia occurs isolatedly or combined with unilateral vaso-motor and sensory disturbances. A persisting hemiplegia in the meaning of a direct focal symptom always allows us to infer the interruption of a pyramidal tract. But this destruction of continuity may occur not only in the internal capsule, but also in the pons, in the pedunculus, in the centrum semiovale, between the internal capsule and the central convolutions, or, finally, in such a manner that the paracentral lobule or the central convolutions are very extensively affected by the hæmorrhage; a permanent hemiplegia may be the result in all these cases. In order to determine in which portion of the pyramidal tract the seat of the hæmorrhage should be looked for, it is best to proceed according to the following view-points:

In the far greater majority of cases the hæmorrhage affects the corpus striatum, the internal capsule and the adjacent portions of the centrum semiovale. It is of practical importance, therefore, first to ask whether positive reasons in the given case are *against* the assumption of the seat of the hæmorrhage in these portions of the cerebrum.

Topical Diagnosis of Motor Hemiplegia.—First of all we must observe an eventful synchronous paralysis of the oculo-motor nerve. *If the paralysis of the oculo-motor nerve, in comparison to the paralysis of the extremities and of the facial nerve, is an alternating one, this fact would decidedly point to a peduncular focus,* as in foci which are situated higher up, a unilateral paralysis of the oculo-motor nerve (especially of the levator palpebræ superioris) scarcely ever occurs as a direct focal symptom, at most only when the focus which causes the hemiplegia extends as far as the region of the parietal lobule; but such a paralysis of the oculo-motor nerve would, at all events, be contralateral, i. e., on the same side as the paralysis of the extremities.

If we find, besides paralysis of the extremities of one side, paralyses of several cerebral nerves (from the fifth nerve on) of the opposite side, this combination points to a focus in the pons-oblongata, more so if dysarthria and disturbances of deglutition and respiration are present at the same time.

If hæmorrhages in the pedunculus and pons cannot be diagnosticated, owing to the absence of the above-named special symptoms—I wish to state particularly that rare cases also occur in which alternating paralysis of the oculo-motor nerve is absent in peduncular foci, and alternating paralyses of the facial, fifth and abducens nerves are absent in pontine foci, thus rendering the differential diagnosis impossible—we must decide now whether the motor hemiplegia as a permanent focal symptom is of such a character that its dependence upon a *cortical* hæmorrhage becomes probable. Monoplegias, which are characteristic of the latter, are not to be considered here, as we are to discuss only under what conditions an (ordinary) *completely* contralateral hemiplegia is not to be referred to a hæmorrhage in the corpus striatum and in the adjoining portions of the centrum semiovale, but to an affection of the cortex or to a lesion of that portion of the medullary substance which is situated nearest to the cortex. We presume in our differentio-diagnostical consideration, therefore, that

it is a question of a cortical hæmorrhage which has *very extensively* attacked the central convolutions, the paracentral lobule or the corona-radiata fibres in their first beginnings, which emerge from those portions of the cortex. Pathognomonic of such cortical foci is that, *after the course of several weeks or months, paroxysmal twitchings occur in the paralyzed extremities and in the face,* or even epileptiform spasmodic attacks, usually with loss of consciousness. The presence of *aphasia*, if it is a permanent focal symptom, also points decidedly to an affection of the cortex, because permanent aphasia in consequence of an affection of subcortical portions of the brain is a decidedly rare occurrence and in such cases must always present the picture of subcortical aphasia. Disturbances of cutaneous sensibility and of muscular perception should only indirectly be considered in the diagnosis.

If we thus, by exclusion, arrive at the conclusion that those less frequent points of origin of hemiplegia cannot be diagnosticated, we may, with a degree of probability which is almost a certainty, diagnosticate a hæmorrhage into the corpus striatum or into the adjoining portion of the medullary substance (which contains the closely crowded motor tracts). As previously stated, a sharp differentiation between foci of these two parts of the brain is impossible.

Monoplegia as a Direct Focal Symptom.—The case is different if *monoplegia* is present as a focal symptom. Paralysis of a single extremity occurs only in hæmorrhages in areas in which the fibres for the arm and the leg are farther apart, consequently in the centrum ovale towards the motor region of the cerebral cortex or in the latter itself. The same rule also applies to a monoplegic paralysis of the facial and hypoglossal nerves, although isolated paralyses of the facial nerve have been observed also in extremely rare cases in capsular and pontine foci. If, therefore, monoplegia generally points to cortical hæmorrhage, yet the diagnosis does not become certain until the paralysis is, later, supervened by the previously mentioned epileptoid spasms, and if aphasia is present besides paralysis (see p. 660). *Ceteris paribus,* the absence of the latter manifestation points rather to a hæmorrhage in the centrum ovale in contrast to hæmorrhage in the cortex of the brain.

Conduct of the Paralyzed Muscles.—*The paralyzed muscles and nerves remain in such a condition that they can be irritated by the electric (the faradaic as well as galvanic) current*; the electric irritability is even increased in some cases, but a reaction of degeneration can never be determined. The volume of the paralyzed muscles remains intact for a long while; only after the lapse of some time does a, usually insignificant, atrophy (of inactivity) occur, which, however, never presents the character of a degenerative atrophy. It is a very common occurrence in the later course of cerebral hæmorrhage that *contractures* develop *in the paralyzed muscles,* especially in those of the arm. They are, mostly, so-called "passive contractures," in that the movements of the joints, which have become fixed owing to the muscular inactivity, gradually are more restricted in the course of the paralysis, thus causing a permanent shortening of the muscles. The increase of the tendon reflexes also adds to

fully known, sometimes present the clinical picture of apo- so because hemiplegic symptoms also occasionally become in meningitis. The demonstration of irritative symptoms prin- cides in favour of meningitis in such cases: The hyperæsthesia of and muscles, the violent headache and, above all, the *contractures musculatures of the trunk and neck,* although it must not be for- that painful rigidity of the back of the neck occurs also in hæm- ages, especially in those situated in the posterior cranial fossa. *Con- sions* are observed in both these cerebral affections; but, whereas in emorrhage they are a rather infrequent symptom during the time of the stroke and are more of a transitory nature, in meningitis they form a persistent, predominating symptom. In the latter affection they are mostly bilateral, in hæmorrhage unilateral, as a rule. The converse is the case with *hemiplegia*; it is the fundamental symptom in the clinical picture of hæmorrhage and is absent in exceptional cases only, whereas it is generally rarer in the course of meningitis and appears only when *one* hemisphere is accidentally very markedly affected by the meningitic exudate. Nevertheless, a confusion of both diseases during this stage is possible if we are dealing with a comatose patient and do not know the course of the affection in the given case. In such instances we must pay especial attention to certain symptoms the determination of which secures the diagnosis of *meningitis.* They are, *besides the hyperæsthesia previously mentioned which extends over the entire body, and general convulsions and contractures, a gradually occurring paralysis of the nerves of the muscles of the eyes, persistent fever and the result of the ophthalmoscopical examination, especially the demonstration of an optic neuritis.*

The differentiation between *pachymeningitis cerebralis hæmorrhagica* and certain forms of cerebral hæmorrhage is more difficult. If the latter affects the cortex or the centrum ovale, it is quite obvious that the clinical picture of these intracerebral hæmorrhages must be similar to that of dural hæmorrhages which act in an irritating and paralyzing manner upon the cortex of the brain. As a matter of fact, pachymeningitis makes its appearance under the picture of the apoplectic insult with coma, slowing of the pulse, unilateral convulsions and contractures or hemiplegias, when the dural hæmorrhage is unilateral, as occurs in about half the cases. It is usually absolutely impossible, under these conditions, to differentiate between the two diseases. It is more probable that pachymeningitis is present if, besides the above-named symptoms, narrowing of the pupils, choked disk and rise of temperature are found, if the symptoms of irritation and paralysis are less sharply pronounced, and if longer-lasting prodromes were present in the concrete case. The diagnosis of *hæmatoma* of the dura mater is especially favoured by preceding mental diseases, particularly paralytic dementia, or by chronic alcoholism, as it is well known that these morbid conditions play the most important part in the ætiology of pachymeningitis hæmorrhagica.

Symptomatic Apoplexies.—The course of some affections of the brain is accompanied with attacks which bear a great resemblance to the clinical picture of hæmorrhagic apoplexy, and which, therefore, may give rise to

the tonic tension of the paralyzed muscles and to a restriction of articular motility. In the leg usually an extension contracture is observed, particularly often a contracture of the musculature of the calf (in that the paralyzed foot sinks plantarward owing to the weight), in the arm we find flexion contracture. The fingers are then in a flexor position, the forearm in pronation, the upper arm in adduction contracture.

So-Called "Post-Hemiplegic" Chorea.—After hemiplegia has persisted for some time, in a few rare cases an involuntarily motility of the paralyzed side, preferably of the arm, occurs in the form of *choreic or athetotic movements.* They occur in some cases of hemiplegia, or hemiplegia with hemianæsthesia, during the time in which the paralyzed parts commence to become movable; we speak, therefore, of a *"post-hemiplegic" chorea or athetosis.* Experience teaches us that hemichorea or hemiathetosis occurs principally in foci which are situated in the optic thalamus or in the posterior portions of the internal capsule adjacent to the same. But their value as topico-diagnostic symptoms is a very limited one, because hemichorea has been, although less frequently, observed also in cerebral foci that were located elsewhere.

Hemianæsthesia, which is often observed as a transitory focal symptom, is much rarer as a permanent focal symptom. It persists in all cases in which the tract of the sensory fibres, the course of which is well known, is affected by the hæmorrhage, especially, therefore, if the latter is located in the posterior third of the posterior shank of the internal capsule or in the medullary substance, in so far as the latter adjoins the central convolutions, or, finally, in the above-named portions of the cortex. There can be no question, however, of an isolated affection of the latter, if, besides unilateral cutaneous anæsthesia, paralyses of the nerves of the special senses are present at the same time, as their cortical areas are, relatively, very remote from the central tactile sphere. On the other hand, according to clinical and physiological experiences we may assume an adjoining position of the entire sensory (also of the special sense nerve) tracts within the internal capsule (and the adjacent corona-radiata region), and, therefore, hemianæsthesia (of the skin) with synchronous paralysis of the entire nerves of the special senses points directly to a focal affection in the internal capsule. But we wish to emphasize particularly that, in foci in the internal capsule, unilateral cutaneous anæsthesia may also occur *without* paralysis of the nerves of the special senses, namely when the central fibres of the optic and acoustic nerves, which, it is true, are situated in closest proximity to the tracts for cutaneous sensibility, are, accidentally, not affected (see Fig. 56).

Differential Diagnosis.—It is not necessary to enter more closely upon a localization of cerebral hæmorrhages; the details of this part of the diagnosis have been exhaustively discussed in a previous chapter. But a more detailed discussion is necessary of the differential diagnosis between cerebral hæmorrhage and other diseases which are accompanied with coma, which present paralyses, etc., which, in short, show a certain similarity in certain stages of their course with the picture of cerebral hæmorrhage.

Meningitis.—Patients who suffer from *meningitis,* if the course of the

disease is not fully known, sometimes present the clinical picture of apoplexy, more so because hemiplegic symptoms also occasionally become prominent in meningitis. The demonstration of irritative symptoms principally decides in favour of meningitis in such cases: The hyperæsthesia of the skin and muscles, the violent headache and, above all, the *contractures of the musculatures of the trunk and neck,* although it must not be forgotten that painful rigidity of the back of the neck occurs also in hæmorrhages, especially in those situated in the posterior cranial fossa. *Convulsions* are observed in both these cerebral affections; but, whereas in hæmorrhage they are a rather infrequent symptom during the time of the stroke and are more of a transitory nature, in meningitis they form a persistent, predominating symptom. In the latter affection they are mostly bilateral, in hæmorrhage unilateral, as a rule. The converse is the case with *hemiplegia*; it is the fundamental symptom in the clinical picture of hæmorrhage and is absent in exceptional cases only, whereas it is generally rarer in the course of meningitis and appears only when *one* hemisphere is accidentally very markedly affected by the meningitic exudate. Nevertheless, a confusion of both diseases during this stage is possible if we are dealing with a comatose patient and do not know the course of the affection in the given case. In such instances we must pay especial attention to certain symptoms the determination of which secures the diagnosis of *meningitis.* They are, *besides the hyperæsthesia previously mentioned which extends over the entire body, and general convulsions and contractures, a gradually occurring paralysis of the nerves of the muscles of the eyes, persistent fever and the result of the ophthalmoscopical examination, especially the demonstration of an optic neuritis.*

The differentiation between *pachymeningitis cerebralis hæmorrhagica* and certain forms of cerebral hæmorrhage is more difficult. If the latter affects the cortex or the centrum ovale, it is quite obvious that the clinical picture of these intracerebral hæmorrhages must be similar to that of dural hæmorrhages which act in an irritating and paralyzing manner upon the cortex of the brain. As a matter of fact, pachymeningitis makes its appearance under the picture of the apoplectic insult with coma, slowing of the pulse, unilateral convulsions and contractures or hemiplegias, when the dural hæmorrhage is unilateral, as occurs in about half the cases. It is usually absolutely impossible, under these conditions, to differentiate between the two diseases. It is more probable that pachymeningitis is present if, besides the above-named symptoms, narrowing of the pupils, choked disk and rise of temperature are found, if the symptoms of irritation and paralysis are less sharply pronounced, and if longer-lasting prodromes were present in the concrete case. The diagnosis of *hæmatoma* of the dura mater is especially favoured by preceding mental diseases, particularly paralytic dementia, or by chronic alcoholism, as it is well known that these morbid conditions play the most important part in the ætiology of pachymeningitis hæmorrhagica.

Symptomatic Apoplexies.—The course of some affections of the brain is accompanied with attacks which bear a great resemblance to the clinical picture of hæmorrhagic apoplexy, and which, therefore, may give rise to

the tonic tension of the paralyzed muscles and to a restriction of articular motility. In the leg usually an extension contracture is observed, particularly often a contracture of the musculature of the calf (in that the paralyzed foot sinks plantarward owing to the weight), in the arm we find flexion contracture. The fingers are then in a flexor position, the forearm in pronation, the upper arm in adduction contracture.

So-Called "Post-Hemiplegic" Chorea.—After hemiplegia has persisted for some time, in a few rare cases an involuntarily motility of the paralyzed side, preferably of the arm, occurs in the form of *choreic or athetotic movements.* They occur in some cases of hemiplegia, or hemiplegia with hemianæsthesia, during the time in which the paralyzed parts commence to become movable; we speak, therefore, of a *"post-hemiplegic" chorea or athetosis.* Experience teaches us that hemichorea or hemiathetosis occurs principally in foci which are situated in the optic thalamus or in the posterior portions of the internal capsule adjacent to the same. But their value as topico-diagnostic symptoms is a very limited one, because hemichorea has been, although less frequently, observed also in cerebral foci that were located elsewhere.

Hemianæsthesia, which is often observed as a transitory focal symptom, is much rarer as a permanent focal symptom. It persists in all cases in which the tract of the sensory fibres, the course of which is well known, is affected by the hæmorrhage, especially, therefore, if the latter is located in the posterior third of the posterior shank of the internal capsule or in the medullary substance, in so far as the latter adjoins the central convolutions, or, finally, in the above-named portions of the cortex. There can be no question, however, of an isolated affection of the latter, if, besides unilateral cutaneous anæsthesia, paralyses of the nerves of the special senses are present at the same time, as their cortical areas are, relatively, very remote from the central tactile sphere. On the other hand, according to clinical and physiological experiences we may assume an adjoining position of the entire sensory (also of the special sense nerve) tracts within the internal capsule (and the adjacent corona-radiata region), and, therefore, hemianæsthesia (of the skin) with synchronous paralysis of the entire nerves of the special senses points directly to a focal affection in the internal capsule. But we wish to emphasize particularly that, in foci in the internal capsule, unilateral cutaneous anæsthesia may also occur *without* paralysis of the nerves of the special senses, namely when the central fibres of the optic and acoustic nerves, which, it is true, are situated in closest proximity to the tracts for cutaneous sensibility, are, accidentally, not affected (see Fig. 56).

Differential Diagnosis.—It is not necessary to enter more closely upon a localization of cerebral hæmorrhages; the details of this part of the diagnosis have been exhaustively discussed in a previous chapter. But a more detailed discussion is necessary of the differential diagnosis between cerebral hæmorrhage and other diseases which are accompanied with coma, which present paralyses, etc., which, in short, show a certain similarity in certain stages of their course with the picture of cerebral hæmorrhage.

Meningitis.—Patients who suffer from *meningitis,* if the course of the

disease is not fully known, sometimes present the clinical picture of apoplexy, more so because hemiplegic symptoms also occasionally become prominent in meningitis. The demonstration of irritative symptoms principally decides in favour of meningitis in such cases: The hyperæsthesia of the skin and muscles, the violent headache and, above all, the *contractures of the musculatures of the trunk and neck,* although it must not be forgotten that painful rigidity of the back of the neck occurs also in hæmorrhages, especially in those situated in the posterior cranial fossa. *Convulsions* are observed in both these cerebral affections; but, whereas in hæmorrhage they are a rather infrequent symptom during the time of the stroke and are more of a transitory nature, in meningitis they form a persistent, predominating symptom. In the latter affection they are mostly bilateral, in hæmorrhage unilateral, as a rule. The converse is the case with *hemiplegia*; it is the fundamental symptom in the clinical picture of hæmorrhage and is absent in exceptional cases only, whereas it is generally rarer in the course of meningitis and appears only when *one* hemisphere is accidentally very markedly affected by the meningitic exudate. Nevertheless, a confusion of both diseases during this stage is possible if we are dealing with a comatose patient and do not know the course of the affection in the given case. In such instances we must pay especial attention to certain symptoms the determination of which secures the diagnosis of *meningitis.* They are, *besides the hyperæsthesia previously mentioned which extends over the entire body, and general convulsions and contractures, a gradually occurring paralysis of the nerves of the muscles of the eyes, persistent fever and the result of the ophthalmoscopical examination, especially the demonstration of an optic neuritis.*

The differentiation between *pachymeningitis cerebralis hæmorrhagica* and certain forms of cerebral hæmorrhage is more difficult. If the latter affects the cortex or the centrum ovale, it is quite obvious that the clinical picture of these intracerebral hæmorrhages must be similar to that of dural hæmorrhages which act in an irritating and paralyzing manner upon the cortex of the brain. As a matter of fact, pachymeningitis makes its appearance under the picture of the apoplectic insult with coma, slowing of the pulse, unilateral convulsions and contractures or hemiplegias, when the dural hæmorrhage is unilateral, as occurs in about half the cases. It is usually absolutely impossible, under these conditions, to differentiate between the two diseases. It is more probable that pachymeningitis is present if, besides the above-named symptoms, narrowing of the pupils, choked disk and rise of temperature are found, if the symptoms of irritation and paralysis are less sharply pronounced, and if longer-lasting prodromes were present in the concrete case. The diagnosis of *hæmatoma* of the dura mater is especially favoured by preceding mental diseases, particularly paralytic dementia, or by chronic alcoholism, as it is well known that these morbid conditions play the most important part in the ætiology of pachymeningitis hæmorrhagica.

Symptomatic Apoplexies.—The course of some affections of the brain is accompanied with attacks which bear a great resemblance to the clinical picture of hæmorrhagic apoplexy, and which, therefore, may give rise to

mistakes. These are apoplectiform attacks which occur in the course of "*sclerose en plaques*," of *progressive paralysis* and of *cerebral tumours* or *abscesses*. The differentiation of such symptomatic apoplexies from those caused by cerebral hæmorrhage offers more difficulties from a theoretical than from a practical standpoint, because the development and the clinical picture of these affections are entirely different from cerebral hæmorrhage. It is only of importance for the diagnostician always to bear in mind that apoplectiform attacks are not unusual in the course of these diseases, that they are followed by transitory hemiplegias and may alternate with epileptiform attacks. Here, also, the ophthalmoscopical examination will, as a rule, give applicable points of support in doubtful cases, in that, for instance, gray atrophy of the optic nerve points to sclerosis, pronounced choked disk to brain tumour, etc. It may occur, also, that non-symptomatic, genuine *epilepsy* must be considered in the differential diagnosis, as the epileptic attack sometimes sets in with simple unconsciousness or with scarcely perceptible convulsions, and, on the other hand, cerebral hæmorrhage may be accompanied, in rare cases, with epileptic convulsions. The differential diagnosis, in such instances, must consider principally the consecutive manifestations and the course of the disease. During the coma proper, the colour of the face of the patients may in advance give some direction as to the diagnosis; the face in epileptics generally appears pale at the onset of the attack, in an apoplectic insult highly flushed.

Uræmia.—The decision of the question whether coma is due to a cerebral hæmorrhage or to a cerebral infection of a *uræmic* or even *septic* nature, may eventually be more difficult than the differential diagnosis between cerebral hæmorrhage and the pathological conditions previously named. As stated on a former occasion, it is always hazardous to diagnosticate the onset of a cerebral hæmorrhage during the comatose stage, and it is certainly correct to leave the differential diagnosis in suspense at this period, even when it is known that the patient is suffering from contracted kidney, an affection which predisposes equally to uræmia and to cerebral hæmorrhage. To forego a positive diagnosis is still more advisable if nothing can be learned regarding the history of the case. For the mere demonstration of albumin in the urine and a profuse secretion of urine does not prove anything in regard to the presence of contracted kidney or of uræmic coma, because it is a demonstrated fact that, in the severe cases of cerebral hæmorrhage, the excretion of watery, albuminous urine constitutes one of the symptoms of the stroke. However, the occurrence of convulsions and vomiting at the very onset of the coma probably points rather to uræmia, whereas a more marked relaxation of *one* extremity and of *one* half of the face (although transitory hemiplegias have been observed in rare cases of uræmia also), as well as the absence of the cremasteric reflex on one side and the conjugate deviation of head and eyes indicate the presence of cerebral hæmorrhage. But the feasibility of a positive decision will soon be apparent, in that the focal symptoms become distinctly prominent within a few hours or days.

Sepsis.—The differential diagnosis between cerebral hæmorrhage and certain cases of *acute septic infection*, with special localization of the

toxine in the brain, sometimes appears absolutely impossible at the first glance. However, the stage of the coma, which sets in suddenly in such cases, only lasts a short while; and this is soon followed, unless they have pre-existed, by symptoms of a different localization of the septic process: The signs of acute endocarditis, of metastatic affection of the skin, articular affections, etc.

Hysteric Hemiplegia.—Finally, we will also mention *hysteric hemiplegias,* because their differentiation from apoplectic hemiplegias frequently presents difficulties. Hysteric *paralyses* are usually not accompanied with paralysis of the facial nerve, nor with hemianopsia, severe coma, etc. But exceptions to this rule occur everywhere. The decision becomes easier if we succeed in determining the psychical character of the paralyses, i. e., to demonstrate them to be "paralyses of the will," or if the paralyses are subject to a remarkably rapid change in regard to intensity and localization, as is usually the case in hysteric paralyses. Furthermore, the *sensibility* shows disturbances only exceptionally in apoplectic hemiplegias, whereas it is severely affected in those of hysteric origin. Besides, hemianæsthesia of the hysteric is usually accompanied with unilateral disturbance of hearing, smell and vision, and, finally, all kinds of other hysteric symptoms may coexist with hemiplegia (for particulars see the chapter on Hysteria).

After having recognised the insult as probably or certainly being of an apoplectic nature, with due consideration of the above-named affections which play a part in the differential diagnosis, it always remains to decide whether the insult and the focal symptoms following the same are the consequences of cerebral hæmorrhage or whether they are not more likely to be due to **softening of the brain,** to the obstruction of a cerebral vessel. The differentio-diagnostical factors which are determining in the decision of this question, will be fully entered into in the discussion of the diagnosis of embolus of an artery of the brain, which will be taken up presently; we only wish to state now that, in spite of the most painstaking consideration of all the facts which play a part in the same, the diagnosis is about as often wrong as correct in the given case, so that it is advisable generally to be satisfied with probable diagnoses in such instances. The diagnosis of *meningeal hæmorrhages,* finally, will be specially discussed later on.

Unusual Pictures of Apoplexy.—There are certain deviations, exceptional cases, from the above-described, usual picture of cerebral hæmorrhage, which must be familiar to the physician to avoid errors in the diagnosis. We shall, therefore, briefly mention them. *Primarily, there are cases of cerebral hæmorrhage in which no insult exists,* in which, therefore, without the presence of the latter, hemiplegia or another focal symptom more or less suddenly takes place. This *rapid* occurrence of a focal symptom (which then, as is conceivable, is generally very unlikely to disappear), in such cases. although no stroke is present, points to the existence of cerebral hæmorrhage or of softening of the brain, which likewise occasionally is brought about without insult. In other cases the course of the *insult is so slow* that, for many hours, only deliria, drowsiness, etc.,

in short, so-called *"prodromes"* exist until coma is complete. Whereas in these cases the "prodromes" (headache, vertigo, tinnitus aurium, melancholia, difficulties of speech, oppression, etc.) pass into severe coma, they disappear in other cases without leaving any deleterious consequences. Then it is obviously a question of small hæmorrhages which, in the latter case, remain small, but, in the former, gradually assume dangerous dimensions. In other cases, again, the prodromes do not occur suddenly but come and go slowly, persist for months and are then, probably, the expression of disturbances of circulation due to atheromatous degeneration of the arteries of the brain which, later, occasionally leads to cerebral hæmorrhage or encephalomalacia.

EMBOLISM AND THROMBOSIS OF THE CEREBRAL ARTERIES: SOFTENING OF THE BRAIN; ENCEPHALOMALACIA.

Encephalomalacia is characterized by necrosis of the cerebral tissue which occurs in foci and the origin of which should be looked for in deficient arterial blood supply.

The shutting off of the blood from circumscribed portions of the brain itself can be brought about in various manners: Usually by emboli which originate either in the heart (in endocarditis, *valvular defects* of the left heart, thrombosis due to deficient action of the heart, especially in the left auricle), or in the larger arteries (atheromatosis and aneurysms); or by thrombi which form in the cerebral arteries proper in consequence of *chronic arteriosclerosis* or *syphilitic endarteritis*. It is obvious that the last-named processes combine eventually with embolism, in that small masses of fibrin become detached from the thrombi and, carried into smaller cerebral vessels, may obstruct the latter. As the arteries of the crus cerebri, especially the branches of artery of the Sylvian fossa, are terminal arteries, a collateral compensation of the circulatory stagnation created by embolism or thrombosis in this arterial region (which comprises the corpus striatum, internal and external capsules, insula, central convolutions, second and third frontal and the parietal convolutions and a part of the temporal brain) is impossible. Thus, in accordance with the usual process of a hæmorrhagic infarct, necrosis of the affected portion of the brain is brought about. Only in the regions of the centrum ovale and of the cortex is the vascular arrangement such that a collateral compensation of the circulation is actually brought about, at least in some of the cases. At any rate, to insure a sufficient collateral circulation, the arterial anæmia of the embolic region should not last longer than a few days; otherwise necrosis of tissue will invariably occur in the form of softening of the brain. The arteries of the Sylvian fossa are most frequently affected by embolism, the left oftener than the right one, as we know from experience; emboli in the optic thalami, cerebral peduncles, cerebellum and pons, in short in the region of the vertebral artery, are of much rarer occurrence. This great predilection of the arteries of the Sylvian fossa for embolism is the reason why embolism of the brain presents a certain uniformity in the clinical picture.

The manifestations which are caused by an obstruction of a cerebral vessel, are connected partly with the act of the obstruction itself (first stage), partly with the process of softening (second stage) and the absence of function caused thereby. In chronic softening of the brain only the latter is considered.

Stage of the Stroke.—Without prodromes, or after headache, vertigo, paræsthesias, sometimes also weakness of the extremity, have preceded,[1] an *apoplectic attack* with its marked symptoms occurs. It is characterized by an *insult*, the condition of which corresponds to the insult in cerebral hæmorrhage.

Cause of the Insult.—The *cause of the insult* has been elucidated by experiments which were made by Geigel, Jr., in the laboratory of the Würzburg clinic.

Accordingly, at the moment of onset of the embolism, not only a circulatory disturbance of the embolized vessel occurs, but of the entire brain. The arterial pressure in the embolized vessel ceases with the embolism; but then the vascular tension in this vessel reaches its full action; the artery endeavours to contract, and this tugging action, which corresponds to the vascular tension of the embolized vessel, is communicated to the walls of those arteries which are not embolized, trying to expand them. The latter, however, is compensated by the intrinsic tension, acting inwardly, of the non-embolized vessels, so that the tension in the latter may be equal to naught for a short moment of time. Thus the blood will force the tensionless wall of the non-embolized vessels outward, but will no longer, in this case, flow towards the veins, and stasis in the capillaries will be the consequence. But even if the latter is not complete, at all events, during the time of the following effect of the embolism, *the blood, which was utilized to expand the non-embolized arteries, is lost to the capillaries and, thus, also to the blood supply of the cortex, in such a manner accounting for the apoplectic insult in embolism.*

The insult is more or less pronounced according to the extent of the occluded region. *It may even be entirely absent* if a small vessel is affected or if it is a question of a simple contraction, not of a complete occlusion of the vessel. It is quite obvious, according to the above-described physical action of the embolism upon the circulation of the blood of the brain in general and of the cortex in particular, that *conditions of epileptic convulsions* are observed *comparatively often* in embolic insult, and it is likewise conceivable that a *slowly occurring* thrombosis of the cerebral arteries is not accompanied with insult, but produces an insult only when the contraction of the vascular district, which had developed until then, by detachment of a larger fibrinous coagulum, gives rise to a sudden complete occlusion of an adjoining vessel.

The *duration* of the insult is generally *shorter* in embolism than in cerebral hæmorrhage. If, exceptionally, it lasts very long, i. e., many days, even then (contrary to the condition of the insult in cerebral hæmorrhage) the prognosis is not absolutely unfavourable, in that, even under such circumstances, a restitution of the circulatory conditions is still possible. According to experience, the embolic insult shows some further peculiarities which, at least sometimes, may direct the differential diagnosis between embolic and hæmorrhagic insult, without, however, being sufficient to claim determining significance differentio-diagnostically. Contrary to the insult in cerebral hæmorrhage, *the initial decrease of the temperature of the body, which is observed in the latter, is absent*; on the

[1] Prodromes are found almost only in those cases of softening of the brain which depend upon atheromatosis of the cerebral vessels, and they are either due to the deficient circulation of the brain which is connected with this condition, or they depend directly upon smallest vascular obstructions and areas of softening.

contrary, often, soon after the insult, a *rapid rise* of the temperature occurs, and this fact does not signify an unfavourable prognosis, as in cerebral hæmorrhage. However, it will still require considerably more confirmation whether this condition of the body temperature is characteristic; neither is the cause of it quite clear as yet. Furthermore, the marked congestion of the face, which is observed in hæmorrhagic insult, is not present at all or less pronounced than in the former, the slowing of the pulse is also absent, as is the specific remote action in form of disturbances of urinary secretion and of respiration. The period of the insult is then followed by the **stage of focal symptoms.** They are, the same as in cerebral hæmorrhage, either transitory or permanent. The *transitory focal symptoms,* which manifest themselves as hemiplegia, hemianæsthesia, etc., may disappear very rapidly when the development of necrosis is prevented by collateral compensation of the circulation, as it may occur in the region of the cerebral cortex. No injurious consequence of the threatening danger can, under such circumstances, be determined within a few days after the attack. In other cases, in which a complete circulatory compensation does not occur, but, on the other hand, a more or less extensive area of softening develops, some of the focal symptoms will, it is true, also disappear gradually, but, according to the location and extension of the area of softening, *permanent (direct) focal symptoms* will continue to persist, i. e., losses of function, which represent the clinical picture of permanent functional disturbances depending upon the necrosis of certain portions of the brain. Their specific description is unnecessary; they are, of course, the same as in cerebral hæmorrhage, and, the same as in the latter, so also in encephalomalacia, location and extension of the individual area of softening should be determined, according to the previously described view-points, from the special character and conduct of those direct focal symptoms. The period of time which must have gone by to characterize the focal symptoms as direct, permanent ones, varies in length in each individual case; a certain term cannot be named in these instances. If focal symptoms occur *without any insult whatever,* we must, from the beginning, expect them to be direct, irreparable ones.

As usually the arteries of the Sylvian fossa and their branches are affected by the emboli, the symptom-picture of the resulting focal symptoms is, as a rule, rather uniform (as in cerebral hæmorrhages), namely that of an *ordinary cerebral hemiplegia.* We have stated before that remarkably often the embolus is driven into the left artery of the Sylvian fossa, which is the reason why *aphasia* is one of the most frequent sequelæ of embolism of the cerebral arteries. Besides, aphasia may manifest itself also in right-sided embolism as an indirect, rapidly passing focal symptom.

Differential Diagnosis.—The factors, which were discussed in the differential diagnosis of the apoplectic insult, are to be considered one after the other in the diagnosis of embolism of the cerebral arteries; otherwise it remains questionable whether it is at all possible to differentiate with absolute *certainty* between an embolism or softening of the brain and a cerebral hæmorrhage. *These questions must be answered in the negative;*

but, on the other hand, it is permissible in most cases, in fact necessary on account of the therapy to be resorted to, at least to attempt to make a probable diagnosis of one of the two affections. In the given case the following symptoms point

TO EMBOLISM	TO HÆMORRHAGE
Youthful age; in a more advanced age embolism and hæmorrhage occur nearly equally often.	More advanced age; hæmorrhage scarcely ever occurs in youthful individuals.
Advanced atheroma, affections of the heart, following acute rheumatism, sepsis; chronic valvular defects, adipose degeneration of the heart, weakness of the heart in general, aneurysms.	Atheromatosis, besides hypertrophy of the heart.
Demonstration of emboli in other organs.	Demonstration that the patient was "quite well" up to the time of the attack. Demonstration of casts in the urine, and of other symptoms of chronic nephritis.
During the insult any marked congestion of the face is absent. The pulse is normal, eventually accelerated in cardiac affections, irregular.	*During the insult*: Flushed face, marked pulsation of the carotid arteries, pulse retarded.
Temperature normal, usually rising very soon after the attack, without indicating a bad prognosis.	Temperature of the body *decreasing* during the insult, increased shortly before death.
Duration of the insult usually short; although exceptionally long in extensive embolism, yet circulatory compensation is possible even then.	*Duration of the insult* comparatively longer than in embolism. Upon long (about two days') duration of unconsciousness recovery does not take place.
Remote actions, especially upon the parts of the posterior cranial fossa (disturbances of respiration, etc.), occur rarely. Hemiplegia is usually right-sided with aphasia.	Remote actions are quite common; changes in the urinary secretion, albuminuria, polyuria, etc.
Ophthalmoscopical finding: Eventually the ophthalmoscopical manifestations of a recent or an old embolism of central retinal artery.	The retinal arteries may present the various stages of atheromatous degeneration; consequently retinal hæmorrhages may have occurred, or the ophthalmoscopical picture of a thrombosis of the central vein of the retina may present itself. In those rare cases in which the hæmorrhagic extravasation perforates into the ventricles, a marked narrowing of the pupils becomes manifest.

Ætiological Diagnosis.—If the diagnosis has been made of probable softening of the brain, there still remains, besides determination of the seat and extension of the morbid area, which takes place according to the rules previously given, the more precise verification of the *ætiological diagnosis.* Primarily the *heart* must be examined for valvular murmurs (mitral and aortic defects), for signs of weakness and adipose degeneration of the musculature; aneurysm must be investigated, and, furthermore, the condition of the *vessels* must be examined for the presence and degree

of atheromatosis. A scrupulous investigation for recovery from *syphilis* must never be neglected, particularly on account of the therapeutic measures eventually to be taken. It is characteristic of syphilitic as well as of senile thrombotic softening of the brain that the prodromes are almost always very marked in these cases, and that an aggravation of the condition takes place in paroxysms, so that intercurrent improvements alternate with attacks of oppression or actual insults, and, finally, that the intelligence is reduced to complete dementia. If *youthful* individuals are attacked by apoplexy, we must always think of *syphilitic softening of the brain,* as they seldom suffer from cerebral hæmorrhages, and as atheroma is not to be considered ætiologically, provided other plausible causes of encephalomalacia (heart defects, etc.) can be excluded. This rule has always stood me in good stead both in diagnosis and in therapy.

Chronic Progressive Softening of the Brain.—The course of senile and of syphilitic softening of the brain is generally decidedly chronic and is characterized by a varying condition, i. e., by improvements and intercurrent, paroxysmal aggravations. But, besides, there occur still other cases of senile softening, which differ from the usual picture, *in that they develop decidedly chronically from the very beginning and cause focal symptoms* (localized manifestations of irritation, later *hemiplegias*, furthermore hemianæsthesias, paræsthesias, sensation of weight in the paralyzed limbs, etc.) *without insult and general symptoms.* The course of these cases is slowly *progressive,* extends over months, even several years, until the focal symptoms have reached a certain stage of development and now remain stationary. At the autopsy these cases present more or less extensive areas of softening, mostly in the medullary substance. The diagnosis of these cerebral softenings, the course of which is slow, without insult, is never easy to make, best by way of exclusion, in which case abscess of the brain and tumour of the brain are to be chiefly considered diagnostically. Development and course of the latter conditions are similar, yet differ in several respects; the ætiology, to some extent, but, above all, the predominance of severe general symptoms, which are almost entirely absent in chronic softening of the brain, decide in favour of abscess or tumour, to the discussion of which the following chapter is devoted.

NEOPLASMS OF THE BRAIN, CEREBRAL TUMOUR, TUMOUR CEREBRI

The diagnosis of tumour of the brain can be made, with great certainty, in most cases. It is based, principally, upon observation of the effects of the growth of the tumour—the *compression.* The latter manifests itself in a diffuse manner and in local symptoms, most markedly in those areas at which the brain is contiguous to firm, more or less unyielding, adjacent structures (falx, tentorium, wall of the skull).

Effects of Pressure.—This *diffuse pressure effect* is indubitably shown, on exposing the brain, in obliteration of the sulci, in a chronic inflammatory thickening of the dura and also in traces of pressure at the cerebral nerves which may be forced against the bony base, displaced and flattened. The veins with relaxed walls are also affected by the compression; this may manifest itself externally in a stasis of the frontal veins (which communicate with the ophthalmic vein). Furthermore, the increased pressure and the compression of the veins within the cranium give rise to exudation of serum into the ventricles, thus causing a further increase of the contents of the skull. Hydrops ventriculi is especially liable to develop if, besides venous stasis, the deflux of the ventricular fluid is

obstructed by direct local pressure of the tumour upon the canalicular system of the cerebral ventricles. *Thus we observe that hydrocephalus develops especially markedly and early in tumours of the corpora quadrigemina, of the pineal gland and of the cerebellum, in short, of the portions of the brain which are situated in the posterior cranial fossa.*

The general effect of pressure is present even in the softest tumours, although they are not really possessed of an actual compression effect. As this pressure effect manifests itself in pronounced clinical symptoms, it must undoubtedly be designated, even in a diagnostic respect, as the *most important effect of the tumour.* The general symptoms depending upon the same considerably precede the local effects of the tumour, which will be discussed later on (the focal symptoms).

Headache is the most constant general symptom which is the earliest to appear. It is very violent, as a rule, mostly bilateral, more rarely unilateral, but usually *not sharply localized.* The tumours of the posterior cranial fossa form an exception in that the headache in such a case, although general, yet is concentrated upon the neck and back of the head. It is particularly suspicious if individuals, who, until then—for decades—never suffered from headache, are attacked by a persistent inexplicable cephalalgia, and if a percussion of the skull at a circumscribed area, and always here only, is painful. The cause of the general headache is ascribed to an irritation of the meninges, especially of the dura mater which abounds with nerves; that headache which is brought about by *local* irritation of certain pain-producing portions of the brain (especially of the pons), will not be referred to at present. *Headache is entirely absent only in the rarest cases,* namely when the mechanical insult is very gradually brought about by tumour which grows very slowly and uniformly, causing the sensory nerves to be inirritable without having previously passed through a period of irritation.

Convulsions.—*Epileptic convulsions* are a second important general symptom of tumours of the brain. They are, it is true, less frequent than the headache, yet they are surely observed in about one half the cases. They also, like the headache, are due to irritation of the surface of the brain.

The same as the headache, they sometimes signify a *local* irritation, when the tumour is located in a certain area of the surface of the brain, viz., in the motor region of the cortex; but they may also be, and this fact is, for the present, interesting, the expression of a *general* compressive action of the tumour, which becomes manifest at the surface of the brain. Epileptic attacks which are produced in such a manner, make their appearance mostly unilaterally upon the contralateral side, but, upon increased irritation, also bilaterally (as in the physiological experiment). In such cases consciousness is greatly impaired or lost. Whereas, at the onset of the disease, the epileptic attacks occur only very rarely, they become considerably more frequent towards the end.

Choked Disk.—Of much more diagnostic importance than both the above-described general manifestations is a third one, the *choked disk,* which can be easily demonstrated by an ophthalmoscopical examination. It is rarely entirely absent in the course of the development of the tumour,

but it is understood, then, that the ophthalmoscopical examination is not made only once in the course of the disease, but often at shorter intervals. The repetition of the ophthalmoscopical examination is of the greatest importance, because in quite a number of cases any variety of functional disturbance is absent, especially at the beginning, and does not become prominent, as a rule, until the so-called atrophic stage of choked disk is ushered in. Choked disk may sometimes be more and earlier pronounced in one eye than in the other.

Cause of Choked Disk.—The occurrence of choked disk is due to the general increase of pressure in the cranial cavity which becomes most developed in cerebral tumour; in that respect, therefore, choked disk is quite especially characteristic of cerebral tumour, but, of course, it is not absent in other affections of the brain which are associated with increase of pressure, as in abscess of the brain, internal hydrocephalus, and others. Experimental investigations have shown that an increase or a decrease of the cerebro-spinal fluid does not exert any influence, but only the increased intracranial pressure which is transmitted along the optic nerve sheaths; the latter, as is well known, are direct continuations of the enveloping membranes of the brain, and, likewise, the subdural space, the subarachnoid spaces of the brain are in immediate connection with the same spaces of the optic nerves.

Other General Symptoms.—Besides the above-named three main effects of the general compressive action of brain tumours, there are still some other general symptoms which sometimes attain diagnostic significance. Of these, *slowing of the pulse* is the most important. It is the expression of an irritation of the pneumogastric nerve centre, which is caused either directly by tumours of the posterior cranial fossa, or indirectly by general intracranial increase of pressure. Furthermore, *vertigo* and *vomiting* are frequent accompaniments of tumours of the brain. The vomiting can easily be recognised as being of cerebral origin, as it occurs independently of changes of the gastric digestion; this can be easily demonstrated by an exploratory sounding of the stomach. *Vertigo* is of subordinate importance as a general symptom; it is of greater significance as a focal symptom of tumours of the posterior cranial fossa, in which case it is very intense and more constant. Of greater importance as general symptoms are *disturbances of the mind and of the sensorium*. The patient becomes indifferent to external impressions, drowsy or stuporous; the acuteness of thinking diminishes, the images of memory are not retained, the memory becomes poor; the facial expression of the patient is without energy, stupid. The patients may finally become fully demented, discharge fæces and urine involuntarily, etc. These psychical disturbances are occasionally associated with *coma*; intercurrent *apoplectiform attacks* may take place, caused by a suddenly occurring, more extensive filling of the vessels, or by hæmorrhages within the region of the tumour, or by a rapidly appearing increase of the ventricular dropsy. It may also be mentioned, finally, that most patients with brain tumours are afflicted with obstinate *constipation*, and that general *debility and emaciation* become prominent; however, the latter symptom occurs also in the course of other severe affections of the brain.

As previously stated, the compressive action of a brain tumour may be

restricted to the above-named general symptoms. This is the case when the mass of the brain can give way to a tumour which grows from without inward, or if it is simply infiltrated with the intracerebral neoplasm (infiltrating tumours), so that more of a forcing apart than a progressive destruction and conduction inability of the tissue elements results. In other cases, again, in the course of the development of the tumour there occur, besides the general compressive action, *local effects of the growths of the tumour,* destruction of ganglion cells and nerve fibres, and thus so-called *focal symptoms* become manifest.

Focal Symptoms.—It is true that much depends upon the seat of the tumour in such cases. As we do not fully know yet, up to now at least, the symptoms which are caused by the destruction of the frontal lobe, corpus callosum, gyrus fornicatus, etc., we have no manifestations which could be explained with certainty, of the local effect of a tumour of these regions. It should be noted, furthermore, that the local effect is much more pronounced if the growing tumour is situated at the base of the brain (at which locality especially the peripheral nerves cannot give way) or near the falx, than if it develops at the convexity or near the medullary substance, at which places a forcing apart of the fibres is, at least temporarily, possible. Part of the local effect of the tumours also depends upon the development of blood vessels in the same, upon the irritating, inflammatory action upon the surrounding parts and, finally, also upon hæmorrhage or softenings in the neighbourhood of the tumour.

The *focal symptoms,* the consequences of such a local action of the tumour, are either of a *direct* or of an *indirect* nature. The occurrence of *indirect focal symptoms* depends, above all, upon the extent of the pressure, and as the latter again is the cause of the general symptoms, we may conclude that, *the less the latter, the more may we interpret the focal symptoms as direct ones,* i. e., *as manifestations caused by the seat of the tumour.*

Direct Focal Symptoms.—Furthermore, we may expect, also, *that the topical diagnosis becomes more certain the smaller the tumour.* In fact, the *seat of the tumour* can be determined with desirable exactness only in those cases in which it is not a question of too large a tumour and in which the general symptoms are little pronounced. Whoever does not consider these principles derived from experience, will be very much astonished to find the seat of the tumour, either post mortem or on trepanation, at an entirely different location from that at which he expected apparently with the greatest probability or even with certainty to find it, by reason of (indirect) focal symptoms.

Hemiplegias which occur during the growth of a tumour, should be judged in the same sense; the slower and steadier a hemiplegia develops and the less the general symptoms become prominent, the more certain it is that it should be considered a direct focal symptom, a local action of the tumour upon the motor-fibre tract. *Monoplegias* are of still greater importance in the localization of the tumour, especially when, as is the case in cortical lesions, different monoplegias occur in a gradual sequence, or when *monocontractures* take place owing to the irritative effect of the tumour. The same as monocontractures, so are *irritative hemicontractures* (in which, in contrast to passive contractures, the motility of the

joints does not become greatly impaired) to a certain extent characteristic of the tumour; at all events, they occur much oftener in cerebral tumours than in other focal affections of the brain. The contractures are, naturally, followed by *epileptic attacks.* As previously stated, they are frequently the expression of the general compressive action of tumours of the brain. But if the epileptic convulsions are restricted regularly to a single member or even to a small muscular region, they may be utilized as a direct focal symptom. As certain tumours occur preferably at the surface of the brain, especially syphilitic tumours and cysticerci, we may expect, above all, epileptic attacks, if they are present. The occurrence of *aphasia* may, eventually, also be utilized in the localization of the tumour, namely when it appears isolatedly with slight general symptoms or associated with monoplegia of the upper extremity, whereas aphasia, when combined with complete hemiplegia, with other paralytic symptoms and with very marked general symptoms, is of subordinate value in the topical diagnosis only as an (indirect) focal symptom. Similar view-points apply to the consideration of *disturbances of sensibility,* hemianæsthesia.

Paralyses of Cerebral Nerves.—Particular caution should be exercised in the diagnostic utilization of *paralytic symptoms of the various cerebral nerves,* which so frequently occur in tumours of the brain. They, also, implausible as it appears at the first glance, yet, as experience teaches us, *may be solely the effects of a pronounced general intracranial pressure.* It must not be forgotten that the brain at its base, owing to the outpassing cerebral nerves, is considerably less displaceable than at other places, and that the nerve trunks, therefore, become especially impaired at this locality by the general compression. It may also happen, as I saw not long ago, that a tumour (of the cortex) flattens the outgoing cerebral nerves at the base not only at the side corresponding to the tumour, but also at the opposite side, so that, then, alternating paralyses become prominent in the clinical picture. Thus, in the above-mentioned case, among the focal symptoms were found right-sided paralysis of the oculomotor nerve and left-sided paralysis of the abducent nerve, a combination which exceedingly aggravated the diagnostic localization of the tumour. If, on the other hand, it is a question of cases in which paralyses of cerebral-nerve trunks occur without marked symptoms of general compression or occur *earlier than the latter,* the diagnostic importance of these nerve paralyses is quite considerable. They are, under such circumstances, a symptom of the *local* action of the tumour and point to the presence of *basal brain tumours* which, in general, occur frequently (above all, as syphilitic neoplasms, gummatous periostitides and sarcomata), and which originate either in the periosteum, respectively the dura, or in those portions of the brain which are situated at the base, especially also in the hypophysis. In such cases the oculo-motor, abducens, facial, fifth nerves, most rarely the hypoglossal nerve, are affected. The paralysis of the various nerves shows, of course, the character of peripheral paralyses.

Paralysis of the *nerves of the muscles of the eye* occurs especially often; in the region of the *fifth nerve* generally only a lesion of the sensory fibres can be determined, which manifests itself, at the onset, as an irritation (neuralgia), later as

anæsthesia dolorosa (interruption of conduction in the peripheral, irritation in the central, portion of the nerve by the tumour) or as total anæsthesia, an order of symptoms which, as such, points from the onset to the character of the affection of the fifth nerve being a lesion of the trunk. Paralysis of the *facial nerve* is characterized as a paralysis of the trunk (in contrast to paralysis of its fibres in the brain) by the fact that the nerve appears *totally* paralyzed, i. e., in all its branches, including the superior ones, that the reflexes are *entirely* lost, and that reaction of degeneration sets in. If the trunk of the *hypoglossal nerve* is affected, this also becomes manifest by the fact that all its branches are paralyzed, one half of the tongue is then immovable and emaciates, deglutition and speech are impeded, etc. (see Paralysis of the Hypoglossal Nerve).

Usually it is not a question of isolated paralyses of one of the above cerebral nerves (the oculo-motor nerve, it is true, is an exception, as its paralysis, caused preferably by syphilitico-gummatous processes and then commencing with ptosis, may occur quite isolatedly), but of combinations of paralyses of different nerves, corresponding to their topically closely connected places of exit. It is obvious that an affection of the nerve trunks by a growing tumour of the brain is indicated principally if the adjoining nerves become gradually paralyzed one after the other, and that this condition is also an important indicator in determining the seat of the tumour.

Tumours of the Posterior Cranial Fossa.—Thus, paralyses in the region of the posterior cerebral nerves (V to XII, see Fig. 64) point to tumours in the *posterior cranial fossa.* In such a case it is not necessary, of course, that the nerves are individually injured by the tumour, as tumours in this region always, owing to the limited space in which they develop, and also, probably, owing to the immediate pressure upon the larger vein of Galen, exert a powerful general-pressure effect even upon slight development. This also accounts for the fact that the symptoms of choked disk manifest themselves so early in this case, and often attacks of blindness occur, which fact points to a considerable extension of the chiasmal recess in association with a very pronounced internal hydrocephalus. Of course, the development of tumours in the posterior cranial fossa manifests itself, besides by paralysis of the cerebral nerves, by cerebellar and pontine symptoms, which symptoms were exhaustively discussed in another chapter (see p. 607 and p. 621).

Tumours of the Central Cranial Fossa.—We have repeatedly mentioned *bilateral disturbance of vision* and *bilateral choked disk* as symptoms of cerebral tumour, and we have seen that these manifestations are the effects of the general compression and, in so far, cannot be utilized to determine the seat of the tumour. But unsymmetrical hemianopsia, the temporal as well as the nasal, is a diagnostically applicable focal symptom of *tumours of the central cranial fossa.* According to the cause or the slower or more rapid development, the ophthalmoscopical finding may be negative at the beginning and later show the manifestations of a moderately choked disk or those of chronic neuritis and atrophy of the papilla of the optic nerve; blindness usually occurs later. Besides, the optic nerve in its intracranial course may be found affected only unilaterally in the form of blindness, without original ophthalmoscopical finding, i. e., an alteration of

the optic papilla, atrophy, etc., may be visible from the beginning. These disturbances and alterations refer to tumours which originate in the frontal or temporal lobes, in the hypophysis or in the base of the skull in the region of the chiasm. It is not astonishing, moreover, according to the anatomical arrangement, that the originally unilateral disturbance of vision soon becomes bilateral. Outside of the pressure upon the optic

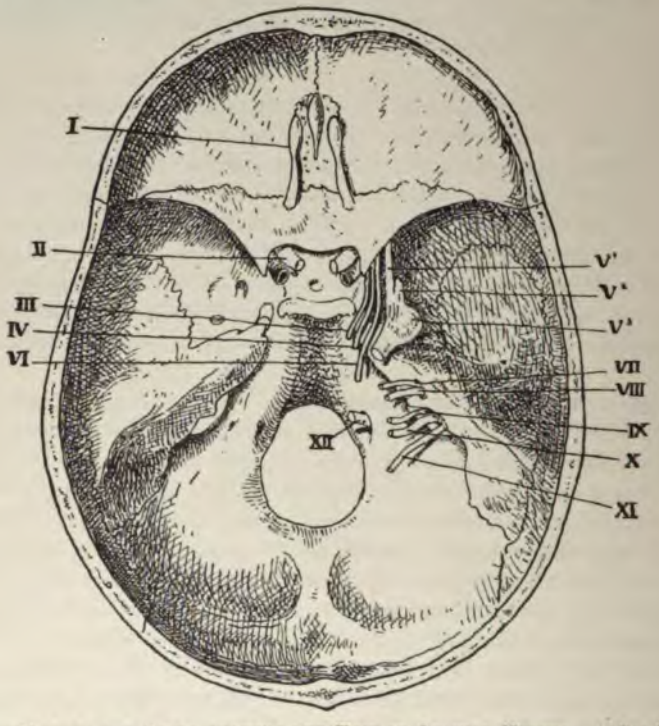


FIG. 63.—BASE OF THE SKULL SHOWING THE EXIT OF CRANIAL NERVES. (After Henle.)
As a guide to the local action of brain tumours at the base, respectively in the various cranial fossæ.

nerve, there occurs, in tumours of the central cranial fossa, a pressure upon the cerebral peduncle of that side upon which the tumour is located. This, then, causes contralateral hemiplegia, which is, eventually, combined with paralysis of the trochlear and oculo-motor nerves of the other side (that of the seat of the tumour). It is quite obvious, from the course of the trunk of the trigeminal nerve and its passage into the central cranial fossa at the anterior border of the tentorium, that the trigeminal nerve is also affected by tumours of the central cranial fossa.

According to the seat of the tumour in one or the other of the *lobes of the brain,* different focal symptoms will become manifest in the given case, depending upon the functional significance of the cortical areas and fibres belonging to the part of the brain affected; for instance, disturbances of hearing and, eventually, of speech in the form of cortical sensory

aphasia in tumours of the temporal brain, hemianopsia or psychical blindness in occipital tumours, etc. It is impossible to enter in detail into this part of the diagnosis of brain tumours, because a simple enumeration of the symptoms which may enable us to make a localizing diagnosis, is entirely without value, and because, on the other hand, the diagnostic points of support which are to be considered in this respect, have been exhaustively discussed in another chapter. I refer, therefore, to the explanations on pp. 622 to 641 and only wish to emphasize here how careful we should be in the exact localization of the seat of a brain tumour until the various focal symptoms can be regarded, with a degree of certainty, as direct focal symptoms, by the simultaneous absence of more pronounced general symptoms and by the manner of their occurrence and course.

Differential Diagnosis.—The order of the diagnosis naturally is such that, first of all, the question must be decided whether a tumour of the brain is present at all. The previously described general symptoms: Headache, choked disk, epileptic attacks, psychical changes, vomiting, etc., and, finally, the steady downward progress of the morbid process, are determining in this instance.

Headaches.—At the onset, so long as headache is the only symptom or predominates, if it is unilateral and associated with vertigo, we may think of a more innocent affection as the cause, viz., *migraine*. But the headaches which characterize the latter condition, rather take place in typical attacks; these are interrupted by periods which are entirely free from headache, during which the psychical faculties of the patient are also entirely intact. During the periods of migrainous headaches absolute rest and the exclusion of sensory and psychical irritations are usually a means of ameliorating the headache, whereas all these measures are almost without avail in the headache of patients who are afflicted with brain tumour. Painfulness of cranial percussion in such cases is also suspicious of the presence of a tumour. The same as migraine, so can others of the many kinds of headache be confused with the headache in brain tumours, thus the "habitual" headache in *neurasthenia, anæmia, hysteria, etc.* Determining for the diagnosis is not so much the character which the "cranial pressure" and headache present in such cases—for the last-named variety of headaches may persist continuously for years, accompanied with loss of energy, mental depression, vertigo and debility, and thus simulate a severe cerebral affection—but the other expressions of those neuroses which are combined with the headache, i. e., synchronous spinal irritation, sexual disturbances, nervous-dyspeptic manifestations, etc., symptoms, which in their entirety present an evidently more harmless clinical picture than that of brain tumour. The steadily progressing aggravation of the symptoms in brain tumour lead, above all, the diagnosis into the right direction, even if, at the onset, doubts exist as to the character of the disease. The diagnosis becomes absolutely certain if the ophthalmoscopical examination shows the presence of an indubitable choked disk. Patients with latent *contracted kidney* and chronic uræmia may also present headache, vertigo and vomiting as the only symptoms and thus create the impression of being affected with tumour of the brain.

Epilepsy.—If epileptic attacks predominate in the pathological picture of brain tumour or if they form, for a certain length of time, which is not a very rare occurrence, the only symptom in patients with tumour of the brain, a confusion of the affection with idiopathic epilepsy is possible. However, the concentration of the convulsions upon one half of the body is a rare occurrence in the latter affection, and, more particularly, the epileptic convulsions restricted to certain muscle groups point to symptomatic epilepsy, depending upon a focal affection on the surface of the brain (Jacksonian). Furthermore, consciousness is usually lost suddenly at the onset of the attack in idiopathic epilepsy; in symptomatic Jacksonian epilepsy, on the other hand, consciousness is retained or lost only during the attack if it extends over both halves of the body. Besides, in idiopathic epilepsy the intervals between the different attacks are usually free from symptoms, unless the attacks become so frequent that the symptoms which remain after the attacks in the latter affection (headache, exhausion, psychical disturbances) persist until another attack takes place. Finally, it is obvious that paralyses, aphasia, etc., are very apt to supervene in Jacksonian epilepsy as symptoms of a further development of the cerebral affection. The fact, also, that the attacks of idiopathic epilepsy are almost always ushered in by a cry, and those of the symptomatic variety rarely, deserves consideration. Of greater importance is the fact that idiopathic epilepsy affects youthful individuals mostly with a hereditary taint, whereas tumours of the brain develop at any period of life. But, above all, an *ophthalmoscopic examination* must be made in every case; the finding of *choked disk* causes the epilepsy to appear as a symptom which depends upon intracranial pressure, a supposition which gains in certainty by the determination of a slow pulse and of cerebral vomiting.

Abscess of the Brain, Meningitis, etc.—As soon as an increased intracranial pressure has been demonstrated as the cause of epileptic attacks or of headache, other affections, which are also accompanied with general increase of pressure in the cranial cavity, must be considered in the differential diagnosis. These are *abscess of the brain* and *meningitis.* Both, among themselves, have a common symptom, viz., fever, which is absent in tumour. It is to be regretted that the temperature in those two affections, especially in abscess of the brain, is not increased regularly. A symptom which is common to all three diseases, choked disk, is by no means as frequent and not as much developed in abscess of the brain and meningitis as in tumour. This applies especially to *abscess of the brain,* in which the symptoms of increased intracranial pressure generally become manifest only transitorily. Particulars as to the diagnosis of abscess of the brain will be found in the following chapter.

Meningitis is more rarely to be considered in the differential diagnosis, because, almost always, the course of this disease is acute, it rarely passes into a chronic stage or becomes of long duration on account of recurrences. As meningitis is an affection of the surface of the brain, here the focal symptoms depend upon an irritation or compression of the cortex or upon a lesion of the cerebral nerves at the base. Focal symptoms, therefore, which must be ascribed to a lesion of the fibre tracts within the brain, in

a doubtful case point directly against meningitis and to tumour of the brain. On the other hand, a circumscribed chronic meningitis at the base, if it leads to a considerable thickening of the meninges, may simulate the symptoms of a brain tumour, and we may eventually no longer be able to distinguish it from the latter.

Chronic softening of the brain, the course of which runs without insult and without general symptoms, is to be considered in the differential diagnosis if the general pressure symptoms are absent in tumour of the brain, as is sometimes, though rarely, the case. So soon as the areas of softening are larger and thus cause more striking symptoms, a confusion is no longer possible, because in tumour, after it has once attained a considerable size, the general symptoms are never entirely absent.

Multiple sclerosis, also, at certain stages of the course of a brain tumour, may be confused with the latter, especially when, in the latter condition, choked disk has passed into atrophy of the optic nerve. The same as in neoplasms of the brain, so also in the course of a sclerosis there occur headache, vertigo, mental disturbances, apoplectiform and epileptiform attacks with subsequent hemiplegias, hemianæsthesias, and, furthermore, paralyses of individual nerves of the brain; however, a differentiation between both diseases is usually easy upon close observation. The entire picture of the disease is different in sclerosis; the disturbance of speech, the intention tremor, which takes place in a relatively slow measure, predominate in sclerosis, whereas headache, oppression, vomiting, slowing of the pulse, convulsions, in short, the pressure symptoms, are prominent in tumour; we also find, as a rule, only a *partial* atrophy of the optic nerve in sclerosis.

More difficult, in fact impossible in some cases, is the differentiation between tumour of the brain and *chronic hydrocephalus,* in which the symptoms which point to the presence of a tumour: Headache, vertigo, disturbance of intelligence, slowing of the pulse, convulsions, vomiting, etc., predominate in the morbid picture, and in which that symptom which is most determining of the diagnosis of tumour, viz., choked disk, is also present. However, the abnormal size and shape of the skull, which is present in hydrocephalus of children, the, often long-lasting, remissions and fluctuations in the intensity of the symptoms and the absence of direct focal symptoms in hydrocephalus may be utilized, in the given case, for the diagnosis of the latter condition against that of cerebral tumour. Finally, we do not mean to conceal that, in rare cases, tumours were found at the autopsy which, never during the life of the affected patients, caused any striking symptoms whatever.

Diagnosis of the Seat of the Tumour.—After the diagnosis of the presence of a tumour of the brain has been made in the above-described manner—and this is possible in most cases—the much more difficult question is to be decided in what part of the cranial cavity, respectively of the brain, should the *seat of the tumour* be looked for. This part of the diagnosis is determined by the statements made in the discussion of focal symptoms, and it is not necessary, therefore, again to refer to them.

Diagnosis of the Character of the Tumour.—But now there remains, as

third part of the diagnostic problem, to determine the *nature of the cerebral tumour in question*. This is not difficult to solve in a great number of cases, unpromising as the undertaking *a priori* appears, and that because a certain point of support regarding the character of the tumour is furnished by the other *morbid manifestations* and by the *ætiology* of the case.

This is so in pronounced *syphilis* and in *tuberculous* individuals. Solitary tubercle or multiple tubercles are found principally in *childhood*. Therefore, in case symptoms of a tumour of the brain occur in children, we should always think first of *tubercle of the brain*, especially if tuberculosis of the lymph glands, bones, etc., can be demonstrated at the same time. Both kinds of neoplasm, gummata and tubercles, have in common, regarding their growth, the intracranial pressure, regarding their seat, the predilection for the surface of the brain. It is an acknowledged fact that the favourite seat of *gummata* is the base of the brain, at which they, commencing at the meninx, particularly often infiltrate the oculomotor nerve gummatously, so that an onset of the cerebral affection with ptosis favours a certain presumption of syphilis of the brain in the diagnosis. If the gummata are situated at the surface, they are very commonly, the same as tubercles, accompanied with epileptic attacks. In this respect they resemble *cysticerci*, which are otherwise distinguished by the fact that a general compressive action is usually absent in these small, mostly only pea-sized cysts, and more pronounced general symptoms take place only when very numerous cysticerci develop simultaneously. Epilepsy, therefore, in this case has the importance of a focal symptom and points decidedly more to cysticerci than to other tumours of the cortex, if general symptoms are not present at the same time. If an ætiological factor is added to this, the demonstration that the individual in question was exposed to infection with tape-worm ova, the probability becomes greater that a development of cysticerci is at the bottom of the cerebral affection, and especially great if cysticerci can be directly demonstrated in the periphery of the body, in the skin or in the eye. *Echinococci*, which develop solitarily at the surface of the brain or in the ventricles, contrary to cysticerci, because considerably larger than the latter, cause very marked general symptoms. But it is better not to diagnosticate them, nor to make a probable diagnosis, even if the development of echinococci in other organs could be demonstrated.

The diagnosis of *sarcomata* and *carcinomata* can be made provisionally, if they occur as metastases in the brain and can be with certainty demonstrated as such at the location of their primary seat; thus, for instance, if a mammary carcinoma or a melanosarcoma of the chorioidea has been determined and symptoms of a brain tumour appear in the further course of the disease. *Glioma*, also, can be diagnosticated with a degree of certainty only if it is complicated with a retinal glioma.

If we presume the presence of one of these three varieties of tumour, consideration should be had in the special diagnosis that *sarcomata* represent rapidly growing tumours, *mostly originate in the bone of the skull, especially in the base in the central cranial fossa*, usually are not very vascular and, in growing, only displace,

not infiltrate, the mass of the brain, whereas *gliomata* represent soft neoplasms which infiltrate the substance of the brain specifically, i. e., pass into them without demarcation. Their seat is usually in the middle of the brain substance; they grow slowly and, in their later stages, become very vascular. Then they act, owing to the varying vascular filling, in a more or less compressing manner upon the adjacent parts, or they may cause, if hæmorrhages occur, suddenly appearing and temporarily increasing hemiplegias. Gliomata, therefore, are "oscillating" regarding the intensity of their symptoms. The development of gliomata (and also that of other cerebral tumours) often follows, as we know from experience, upon a *traumatism* which has acted upon the head of the patient. *Carcinomata*, finally, of the brain, the same as those of other parts, are distinguished from other neoplasms of the brain by their rapid growth (like sarcomata). Other tumours occurring in the brain, such as *myxoma, lipoma, enchondroma, cholesteatoma, psammoma*, are only of a pathologico-anatomical interest; this, however, is not the case with *aneurysms* which develop in the arteries of the base and which sometimes attain the size of an egg; they are occasionally diagnosticable.

Aneurysm of Cerebral Arteries.—The course of the disease in *aneurysms of the cerebral arteries* actually differs slightly from the usual picture; the symptoms point to a basal tumour and present a development which is more by bounds, "stage-like" than is the case with other brain tumours. If the "aneurysm" becomes obliterated, recovery may take place after severe tumour manifestations; in other cases sudden embolism of a cerebral vessel communicating with the aneurysm and, with it, circumscribed softening of the brain, is brought about; in other cases again,—and this the most frequent termination—rupture of the sac occurs. In such a case the blood spreads superficially over the surface of the brain and causes a sudden apoplectic attack from which the patients may recover until another rupture leads to a fatal termination. If patients present, furthermore, besides symptoms of brain tumour which show such a bound-like character in the development of the cerebral affection, aneurysmal dilatations of the carotid artery, the diagnosis of aneurysm of the cerebral arteries gains a certain support thereby. This is still more the case if an ophthalmoscopical examination reveals atheromatous degeneration in the vessels of the retina. Of course, choked disk may also have developed with a relatively large aneurysm of the cerebral arteries.

Although we may be successful, with the aid of the above-mentioned points of support, to obtain a certain direction for the diagnosis of the variety of neoplasm, yet we must bear in mind that it is always a question only of diagnoses which are scarcely more than provisional. At all events, in the majority of cases nothing but trepanation or autopsy will teach us the quality of the diagnosticated tumour of the brain in the given case.

ABSCESS OF THE BRAIN—ENCEPHALITIS SUPPURATIVA

Abscess of the brain, in a clinico-symptomatological respect, is midway between tumour of the brain and softening of the brain. It is a question, in this case, of a circumscribed, suppurative inflammation of the brain, of the formation of an abscess in the neighbourhood of which œdematous softening of the brain substance, in the interior of which pus and *débris* of destroyed nerve tissue are found. In the further course of the disease, due to the *progress* of the suppuration, there occur either *sinus thrombosis* and *meningitis*, or *perforation into the ventricles*, or *encapsulation of the*

Acute Abscesses of the Brain.—The above-described clinical picture corresponds to that of brain abscess with a *chronic* course; the *acute* form presents another picture with a most fulminant course. It is accompanied with fever, sensory disturbances, apathy, somnolence, headache and vomiting; these symptoms become associated with uncertainty of movements, pareses, later, possibly, also transitory unilateral or bilateral convulsions, jactitation and severe deliria and coma during which death occurs. It is very rare that abscesses which are accompanied with such severe acute cerebral symptoms do not lead to a fatal termination but pass into a chronic stage and then end in the previously described manner.

Differential Diagnosis of Acute Abscess of the Brain.—The *diagnosis of these acute, rapidly growing abscesses* can often be made with a good deal of certainty, although the symptoms in many respects are quite similar to those of *purulent meningitis.* However, there are certain symptoms in the clinical picture of meningitis which are characteristic of the latter and *absent* in abscess, thus: Rigidity of the muscles of the neck, trismus, retraction of the abdomen, general hyperæsthesia of the skin and of the musculature and, finally, mostly also the paralytic symptoms on the part of the cerebral nerves, such as pareses of the facial nerve, strabismus, etc. However, we should bear in mind that abscess may, secondarily, lead to purulent meningitis, that both processes may occur in combination, therefore. More difficult is the differentiation between acute abscess of the brain and *meningeal hæmorrhage,* which, the same as acute abscess, takes place after traumatism of the skull and which, owing to the pressure of the exudation of blood upon the surface of the brain, is also accompanied with coma and epileptic attacks. But the symptoms in such a case are present to their fullest extent and more from the onset, after the effect of the traumatism; intermittent fever, which is peculiar to abscess formation, is also absent in meningeal hæmorrhage.

Diagnosis of Chronic Abscesses of the Brain.—The *diagnosis of cerebral abscesses with a subacute or chronic course* does not present any great difficulties. It is based upon the entirety of the general symptoms and focal manifestations mentioned. I wish again to emphasize that the general symptoms, upon the whole, are of a moderate and, above all, of a varying intensity, and that the focal symptoms, because they are usually direct symptoms, allow, as a rule, of a rather certain localization of the seat of the abscess. Special attention may also be called to the fact that the hemiplegias occur paroxysmally, and that paralyses of cerebral nerves are only rarely caused by abscesses of the brain. These factors suffice to show a certain deviation of the course of the abscess from the usual condition of the clinical picture of *cerebral tumours,* but the differentiation is facilitated further by an ophthalmoscopical examination, which, in abscess, rarely, in tumour almost always, reveals the presence of choked disk, by observation of the intermittent fever and, finally, *by consideration of the ætiology.*

Ætiological Diagnosis.—The ætiology plays such an important part in the diagnosis of abscess that, by reason of my diagnostic experience, I do not hesitate to recommend as a rule, to which I have adhered for many

years, *not* to diagnosticate *abscess* of the brain unless *a certain cause for the appearance of the same can be ascertained.* Although it is certain that there are "idiopathic" abscesses of the brain, i. e., abscesses which apparently arose spontaneously, their range, however, has become a very narrowed one, and the few remaining instances, the origin of which is unknown, cannot determine me to deviate from the above rule.

The most frequent cause of abscess of the brain is *traumatism* which affects the brain itself or the skull. Uncomplicated fractures of the latter rarely lead to the development of cerebral abscess; we must assume in such cases, according to my opinion, that the traumatism causes a contusion and incomplete necrosis of the brain substance which favours a retention and development of the pyogenic organisms which, occasionally, are introduced by the blood current. The fact that abscess formation sometimes takes place in the neighbourhood of areas of softening, of hæmorrhages and tumours of the brain, should be accounted for in a similar manner.

A further, frequent cause of abscess of the brain is the *local transmission of a suppurating process* from parts of the body which are in immediate connection with the cranial cavity; thus it may be that *suppurations in the naso-pharyngeal space, in the nasal cavity, in the orbital cavity, phlegmons of the cellular tissue of the neck, parotitis, erysipelas of the head,* but, above all, (tuberculous or syphilitic) *caries of the various bones of the skull,* lead to abscesses of the brain. The most frequent source of cerebral abscess is *caries of the petrous bone, respectively of the mastoid process,* which, associated with otitis media, manifests itself by purulent otorrhœa. Especial attention, therefore, should be paid to the latter symptom in all cases in which suspicion of a developing brain abscess is present; the oversight of otorrhœa in such cases is actually a diagnostic offence! Caries of the petrous bone is quite usually the intermediate stage between certain infectious diseases (enteric fever, variola, scarlatina, tuberculosis, etc.) and abscess of the brain. Abscesses which arise in such a manner are usually located in those parts of the brain which are adjacent to the petrous bone, i. e., in the temporal lobe and in the cerebellar hemispheres. It is a rare occurrence that caries of other bones of the skull, of the frontal bone, ethmoid bone, etc., gives rise to abscess of the brain.

The cause of the formation of cerebral abscesses in other cases is the *introduction of pyogenic organisms from remote organs,* thus, in particular, from the lungs, as in *empyema, pulmonary abscess, putrid bronchitis and bronchiectasis.* A case, illustrating the origin of brain abscess from this cause, has been reported on a former occasion (see p. 102). A careful examination of the lungs should, therefore, never be omitted if the presence of abscess of the brain is suspected. An examination of the heart may, likewise, lead to the discovery of the source of a cerebral abscess, as the introduction of pyogenic organisms into the brain may, in *septic endocarditis,* take place at any time by means of an embolism. *Septicopyæmia* generally gives rise to purulent inflammation of the brain, although less in the form of solitary large than as multiple small abscesses. Cerebral abscesses are observed in a like manner to occur in the course of certain infectious diseases, especially in enteric fever, in which suppuration in the brain, if not directly by the respective pathogenic bacteria, is possibly brought about in such a manner that the chemical products of the infecting organisms prepare the soil upon which pyogenic bacteria entering the body display their action.

If we, in every individual case, consider these various possibilities of the source of brain abscess and scrupulously examine the various organs concerned, we will in the rarest cases only look in vain for the source of the brain abscess, with the finding of which the diagnosis becomes firmly established.

SCLEROTIC ENCEPHALITIS—POLIOENCEPHALITIS—NON-SUPPURATING INFLAMMATION OF THE SUBSTANCE OF THE BRAIN

Disseminated Sclerosis of the Brain.—***Disseminated sclerotic encephalitis*** as a partial phenomenon of so-called multiple insular cerebro-spinal sclerosis (see Multiple Sclerosis) is a comparatively frequent affection of the brain, and the diagnosis of a participation of the brain in the process can be made, in the given case, without difficulty from the presence of certain signs in the morbid picture. Such symptoms, pointing to an involvement of the brain, are: Headache, vertigo, apoplectiform or epileptiform attacks with subsequent transitory hemiplegias, especially also psychical alterations (conditions of depression and exaltation, impaired memory, dementia). The small foci affect both the white and the gray substances of the brain.

Superior Polioencephalitis.—So-called ***superior polioencephalitis***, in which the anatomical affection is restricted to the nuclei of the abducent, trochlear and oculomotor nerves, the clinical symptoms to progressive paralyses in the region of those eye-muscle nerves (*progressive ophthalmoplegia*), likewise is an affection which, in a diagnostical respect, is well characterized, which was treated of in the discussion of bulbar paralysis (see Pseudobulbar Paralysis).

On the other hand, the non-purulent encephalitides, which also occur, represent in an anatomical, and, more so, in a clinical, respect, morbid pictures which are as yet insufficiently defined, and the diagnosis of which, accordingly, scarcely attains the level of a probable diagnosis.

Acute infantile encephalitis is the form which is comparatively best understood. The diagnosis must consider principally the acute, febrile course of the disease, showing exclusively cerebral symptoms, in children during the first years of life. The affection commences with fever, vomiting, convulsions and coma, followed in a few days or weeks by a, mostly, unilateral paralysis of the extremities (rarely with synchronous affection of the region of the facial nerve); it does not occur often that the paralysis manifests itself in the form of a monoplegia or even as a paraplegia. If the paralysis does not recede, we observe the growth of the paralyzed muscles to be retarded; *but the nerves and the atrophic muscles* (in comparison to the musculature on the non paralyzed side) *show a normal electric condition, especially no reaction of degeneration, in contrast to the condition in the spinal form*; the *tendon reflexes* are not diminished, as in the other form, but, on the contrary, increased, and *muscle contractures* develop almost regularly ("*hemiplegia spastica infantilis*"). Hemichorea and hemiathetosis also often develop in the later course of the disease; sometimes epilepsy and mental weakness. According to the age of the child and the seat of the cerebral affection we observe *aphasia*, which, however, as is obvious, usually disappears rapidly. *Sensibility* is almost always intact in the cerebral, the same as in the spinal, form.

It is undoubtedly always correct to consider hemiplegias of infants primarily as the result of a cured acute encephalitis, especially "polioencephalitis." Otherwise infancy as such, of course, does not preclude every other manner of origin of hemiplegia, and we must always consider, also, the possibility of embolism, etc., of a cerebral artery due to a heart defect, before assuming the presence of "infantile cerebral paralysis." The diagnosis of the latter condition is determined especially by the *acute febrile onset of the affection*. It is immaterial how hemiplegia in infancy is brought about, the growth of the paralyzed extremities will surely be retarded. But, according to what has been remarked on a former occasion (p. 669), a participation in the permanent paralysis of those muscles which act symmetrically upon both halves of the body should be expected particularly in childhood, owing to the complete interference of the intact hemisphere. The *occurrence of associated movements* (see p. 669), which is *frequently observed* in infantile hemiplegia, can, in my opinion, be explained in the same manner and, in part, also by the loss of inhibitions in the brain.

Rare Forms of Encephalitis.—But multiple hæmorrhagico-encephalitic foci were also found in the brain in the course of infectious diseases, such as enteric fever,

variola, articular rheumatism and, above all, influenza, but even without preceding, demonstrable infection or intoxication. Furthermore, *diffuse scleroses of entire cerebral lobes*, taking a chronic course, with development of connective tissue and nerve-fibre atrophy (probably due to congenital syphilis and to alcoholism) were determined in a few rare cases. I mention these findings only for completeness' sake; diagnostically, I am frank enough to admit, we are, as yet, not able to make great use of them. For the clinical symptoms ascribed to those anatomical changes of the brain: Headache, hemiplegias or general paralyses, symptoms of motor irritation —tremor, epileptiform attacks—psychical alienation, coma, etc., are symptoms which are too frequently occurring and too vague to base a diagnosis upon them. At most, in cases in which the clinical picture does not agree with the well-known pictures of affection of the brain (abscess, tumour, meningitis, etc.) and differs, in particular, from the picture of the above-mentioned, clinically better understood, encephalitic processes, we may be permitted to assume that the cephalic general manifestations, respectively focal symptoms, and the psychical disturbances are due to a change of the brain, which, as yet, is not diagnosticable, such as diffuse cerebral sclerosis. By reason of the above-described symptoms it is much rather possible to diagnosticate acute hæmorrhagic encephalitis, as its febrile, acute, often turbulent course distinguishes this affection from other diseases of the brain.

Paralytic Dementia.—But another form of cerebral affection, which may be designated as a diffuse interstitial encephalitis with atrophy of the nerve fibres (and nerve cells), viz., *progressive paralysis*, is so well characterized clinically that it is usually easily diagnosticable.

DEMENTIA PARALYTICA, PROGRESSIVE PARALYSIS OF THE INSANE

Although this affection shows numerous symptoms which point to an interruption of conduction of the motor, sensory and other nerve tracts, and although degeneration of the posterior or lateral columns in the spinal cord was recently found almost regularly, besides the cerebral changes, yet the psychical alteration prevails in the pathological picture to such an extent that progressive paralysis is correctly counted among the mental diseases. Therefore, I refrain from a detailed discussion of the same, but I cannot forbear at least to direct attention to some diagnostic criteria concerning this affection, because the knowledge of these points is indispensable for the differential diagnosis between this and other affections of the brain and spinal cord.

Of *somatic disturbances* which appear early in the clinical picture of progressive paralysis, the *tabetic manifestations* are, above all, important: *Reflex pupillary rigidity*, inequality of the pupils, paralyses of the muscles of the eye and atrophy of the optic nerve, loss of tendon reflexes (in some cases, however, especially at the onset of the disease, on the contrary, an increase of these reflexes with spastic paralyses of the legs can be determined), rheumatoid pains and neuralgias, disturbances of sensibility, ataxia and disturbances of the urinary bladder. These tabetic symptoms are frequently accompanied, furthermore, with *tremor* (which, at times, is localized, at other times is disseminated over the entire body and takes place especially upon movements and not uniformly), and with certain *disturbances of speech*, the uncertain speech, so-called *syllable stumbling*. The combination of sounds and syllables into a word becomes deficient so that the rapid, correct pronunciation of difficult words is no longer accomplished successfully, because some syllables or sounds are entirely omitted and mixed up by the patient. *Disturbances of reading and writing* also occur: The handwriting becomes extravagant, unequal, trembling; letters and punctuation marks are omitted, and what has been written finally becomes unintelligible. Although the last-named symptoms suffice to indicate a certain loss of memory, the entire psychical conduct of the patient shows sufficiently that distinct loss of mental capacity which is characteristic of the entire disease.

The *mental disturbance* prevails not only over the somatic disturbances, but in the majority of cases it forms the *initial symptom of the disease*. The characteristic sign of the change of the psychical conduct is the alteration of the character

SCLEROTIC ENCEPHALITIS—POLIOENCEPHALITIS—NON-SUPPURATING INFLAMMATION OF THE SUBSTANCE OF THE BRAIN

Disseminated Sclerosis of the Brain.—***Disseminated sclerotic encephalitis*** as a partial phenomenon of so-called multiple insular cerebro-spinal sclerosis (see Multiple Sclerosis) is a comparatively frequent affection of the brain, and the diagnosis of a participation of the brain in the process can be made, in the given case, without difficulty from the presence of certain signs in the morbid picture. Such symptoms, pointing to an involvement of the brain, are: Headache, vertigo, apoplectiform or epileptiform attacks with subsequent transitory hemiplegias, especially also psychical alterations (conditions of depression and exaltation, impaired memory, dementia). The small foci affect both the white and the gray substances of the brain.

Superior Polioencephalitis.—So-called ***superior polioencephalitis***, in which the anatomical affection is restricted to the nuclei of the abducent, trochlear and oculomotor nerves, the clinical symptoms to progressive paralyses in the region of those eye-muscle nerves (*progressive ophthalmoplegia*), likewise is an affection which, in a diagnostical respect, is well characterized, which was treated of in the discussion of bulbar paralysis (see Pseudobulbar Paralysis).

On the other hand, the non-purulent encephalitides, which also occur, represent in an anatomical, and, more so, in a clinical, respect, morbid pictures which are as yet insufficiently defined, and the diagnosis of which, accordingly, scarcely attains the level of a probable diagnosis.

Acute infantile encephalitis is the form which is comparatively best understood. The diagnosis must consider principally the acute, febrile course of the disease, showing exclusively cerebral symptoms, in children during the first years of life. The affection commences with fever, vomiting, convulsions and coma, followed in a few days or weeks by a, mostly, unilateral paralysis of the extremities (rarely with synchronous affection of the region of the facial nerve); it does not occur often that the paralysis manifests itself in the form of a monoplegia or even as a paraplegia. If the paralysis does not recede, we observe the growth of the paralyzed muscles to be retarded; *but the nerves and the atrophic muscles* (in comparison to the musculature on the non-paralyzed side) *show a normal electric condition, especially no reaction of degeneration, in contrast to the condition in the spinal form*; the *tendon reflexes* are not diminished, as in the other form, but, on the contrary, increased, and *muscle contractures* develop almost regularly ("*hemiplegia spastica infantilis*"). Hemichorea and hemiathetosis also often develop in the later course of the disease; sometimes epilepsy and mental weakness. According to the age of the child and the seat of the cerebral affection we observe *aphasia*, which, however, as is obvious, usually disappears rapidly. *Sensibility* is almost always intact in the cerebral, the same as in the spinal, form.

It is undoubtedly always correct to consider hemiplegias of infants primarily as the result of a cured acute encephalitis, especially "polioencephalitis." Otherwise infancy as such, of course, does not preclude every other manner of origin of hemiplegia, and we must always consider, also, the possibility of embolism, etc., of a cerebral artery due to a heart defect, before assuming the presence of "infantile cerebral paralysis." The diagnosis of the latter condition is determined especially by the *acute febrile onset of the affection*. It is immaterial how hemiplegia in infancy is brought about, the growth of the paralyzed extremities will surely be retarded. But, according to what has been remarked on a former occasion (p. 669), a participation in the permanent paralysis of those muscles which act symmetrically upon both halves of the body should be expected particularly in childhood, owing to the complete interference of the intact hemisphere. The *occurrence of associated movements* (see p. 669), which is *frequently observed* in infantile hemiplegia, can, in my opinion, be explained in the same manner and, in part, also by the loss of inhibitions in the brain.

Rare Forms of Encephalitis.—But multiple hæmorrhagico-encephalitic foci were also found in the brain in the course of infectious diseases, such as enteric fever,

variola, articular rheumatism and, above all, influenza, but even without preceding, demonstrable infection or intoxication. Furthermore, *diffuse scleroses of entire cerebral lobes*, taking a chronic course, with development of connective tissue and nerve-fibre atrophy (probably due to congenital syphilis and to alcoholism) were determined in a few rare cases. I mention these findings only for completeness' sake; diagnostically, I am frank enough to admit, we are, as yet, not able to make great use of them. For the clinical symptoms ascribed to those anatomical changes of the brain: Headache, hemiplegias or general paralyses, symptoms of motor irritation —tremor, epileptiform attacks—psychical alienation, coma, etc., are symptoms which are too frequently occurring and too vague to base a diagnosis upon them. At most, in cases in which the clinical picture does not agree with the well-known pictures of affection of the brain (abscess, tumour, meningitis, etc.) and differs, in particular, from the picture of the above-mentioned, clinically better understood, encephalitic processes, we may be permitted to assume that the cephalic general manifestations, respectively focal symptoms, and the psychical disturbances are due to a change of the brain, which, as yet, is not diagnosticable, such as diffuse cerebral sclerosis. By reason of the above-described symptoms it is much rather possible to diagnosticate acute hæmorrhagic encephalitis, as its febrile, acute, often turbulent course distinguishes this affection from other diseases of the brain.

Paralytic Dementia.—But another form of cerebral affection, which may be designated as a diffuse interstitial encephalitis with atrophy of the nerve fibres (and nerve cells), viz., *progressive paralysis*, is so well characterized clinically that it is usually easily diagnosticable.

DEMENTIA PARALYTICA, PROGRESSIVE PARALYSIS OF THE INSANE

Although this affection shows numerous symptoms which point to an interruption of conduction of the motor, sensory and other nerve tracts, and although degeneration of the posterior or lateral columns in the spinal cord was recently found almost regularly, besides the cerebral changes, yet the psychical alteration prevails in the pathological picture to such an extent that progressive paralysis is correctly counted among the mental diseases. Therefore, I refrain from a detailed discussion of the same, but I cannot forbear at least to direct attention to some diagnostic criteria concerning this affection, because the knowledge of these points is indispensable for the differential diagnosis between this and other affections of the brain and spinal cord.

Of *somatic disturbances* which appear early in the clinical picture of progressive paralysis, the *tabetic manifestations* are, above all, important: *Reflex pupillary rigidity*, inequality of the pupils, paralyses of the muscles of the eye and atrophy of the optic nerve, loss of tendon reflexes (in some cases, however, especially at the onset of the disease, on the contrary, an increase of these reflexes with spastic paralyses of the legs can be determined), rheumatoid pains and neuralgias, disturbances of sensibility, ataxia and disturbances of the urinary bladder. These tabetic symptoms are frequently accompanied, furthermore, with *tremor* (which, at times, is localized, at other times is disseminated over the entire body and takes place especially upon movements and not uniformly), and with certain *disturbances of speech*, the uncertain speech, so-called *syllable stumbling*. The combination of sounds and syllables into a word becomes deficient so that the rapid, correct pronunciation of difficult words is no longer accomplished successfully, because some syllables or sounds are entirely omitted and mixed up by the patient. *Disturbances of reading and writing* also occur: The handwriting becomes extravagant, unequal, trembling; letters and punctuation marks are omitted, and what has been written finally becomes unintelligible. Although the last-named symptoms suffice to indicate a certain loss of memory, the entire psychical conduct of the patient shows sufficiently that distinct loss of mental capacity which is characteristic of the entire disease.

The *mental disturbance* prevails not only over the somatic disturbances, but in the majority of cases it forms the *initial symptom of the disease*. The characteristic sign of the change of the psychical conduct is the alteration of the character

and the lessening of the mental capacity. The memory becomes weak; images of memory of sensory impressions are lost, concentrated thinking, the solving of difficult arithmetical problems become impossible, etc. Ethical conceptions become weak and lose their inhibitory effect upon mental life; the mind shows an unwonted lability—from uncalled-for bursts of lamentation or hilarity to mental indolence. To these symptoms are superadded delusions of various kinds, manifesting themselves in delusions of grandeur or in hypochondriacal ideas, so that, in the latter case, the patients, who endure the quecrest sensations in the stomach, intestine, heart, etc., at the first glance create the impression of simple neurasthenics, especially as, at the onset of the disease, they also complain of apparently quite harmless symptoms, such as dizziness, disturbance of sleep, etc. The picture of the psychical alteration gradually becomes more serious in the further course of the affection: Abulia becomes more prominent, and, as the final stage of the entire psychical depravation, loss of mental irritability *in toto* to complete idiocy make their appearance.

Previous to reaching this final stage, sooner or later in the course of the progressive paralysis, certain paroxysms, which are characteristic of the same, manifest themselves, so-called "*paralytic attacks.*" They consist in attacks of an apoplectiform or epileptiform character, lasting from a few minutes to half an hour and longer, usually leaving focal symptoms of various kinds to persist: Hemiplegias, monoplegias, aphasia, psychical blindness, etc., but always a marked loss of mental capability. These attacks may be confused with *ordinary apoplectic or epileptic attacks*, but only when the patient is not seen until during the attack and when there is no opportunity to study him more closely; otherwise the briefly sketched, characteristic symptom-complex which distinguishes progressive paralysis will soon guide the diagnosis in the right direction. Paralytic dementia has in common with *tumours of the brain* the progressive character of the course of the disease, the gradual loss of memory, the epileptic attacks and the cortical focal symptoms. But the general pressure manifestations which accompany the development of cerebral tumours, are absent in progressive paralysis: The violent headache, choked disk, slowing of the pulse and cerebral vomiting. If, aside from paralytic attacks, a typical disturbance of speech has developed, or if the tabetic symptoms become more prominent in the pathological picture, and if we pay full attention to that characteristic progressive alienation and weakening of the psychical powers, which predominates in the entire course of the affection, it will be impossible to mistake paralytic dementia in spite of the ambiguity of the clinical picture, respectively to confuse it with other affections. Progressive paralysis represents, in an anatomical respect, a degeneration, which commences in foci and, later, becomes diffuse, of the ganglion cells and nerve fibres, with atrophy of the nerve elements. The degenerative process comprises not only the cortex of the brain, but also subcortical cerebral regions (thalamus, central gray substance of the cavities, corpora quadrigemina, cerebellum, etc.) and, surely, also the spinal cord. This accounts unconstrainedly for the occurrence of "somatic" manifestations in the clinical picture of this paralysis, and also, as quite especially the association centres and, preferably, the cortical portions of the frontal brain are implicated, for the predominance of mental disturbances in this affection. As, therefore, in paralytic dementia, as we know to-day, the entire central nervous system is more or less affected by the degenerative process, this disease forms the natural transition to the *diffuse* affections of the brain, the discussion of which will be taken up presently.

DIFFUSE AFFECTIONS OF THE BRAIN

GENERAL DISTURBANCES OF CIRCULATION—HYPERÆMIA AND ANÆMIA OF THE BRAIN—HYPERDIÆMORRHYSIS AND ADIÆMORRHYSIS CEREBRI (GEIGEL).

THE doctrine of anæmia and hyperæmia formerly played a very prominent, often misused, part in the diagnosis of affections of the brain; this applies especially to cerebral hyperæmia. The interesting investigations of R. Geigel regarding the circulatory conditions of the brain have recently initiated a reform in this domain of affections of the brain. I shall, as much as possible for the present, take this into consideration in this chapter.

Analysis of Disturbances of Circulation of the Brain.—The regular performance of the functions of the brain is determined, not only by the quantity of blood which is present in the cerebral vessels, but also, above all, by the temporarily better supply of oxygen to the nerve elements of the brain (and elimination of carbonic-acid gas). This interchange of gases, however, apart from the chemical constitution of the blood in general, depends upon the speed of the current in the capillaries. Geigel calls that flow through the cerebral capillaries, which takes place within the normal limits, "*eudiœmorrhysis*"; the negative deviation from the normal, he designates as *adiœmorrhysis*; the positive deviation, *hyperdiœmorrhysis*. Whether one or the other of these conditions is to be considered present, can be decided with more or less certainty, according to Geigel's deductions.

Geigel considers the *speed of the current in the capillaries of the brain* the essential factor for the better or poorer blood supply to the brain. With an unaltered arterial pressure, *a diminution of vascular tension* must be followed by a *decreased speed of the blood current*, i. e., a relaxation and dilatation of the arterial vessels of the brain (paralysis of the sympathetic nerve) does not cause "cerebral hyperæmia," but, on the contrary, *adiœmorrhysis*. *Vice versa*, an increase of *vascular tension*, especially *spastic contraction of the cerebral arteries* (by irritation of the sympathetic nerve) does not lead to "anæmia," but *to an increased speed of the blood current in the capillaries, viz., hyperdiœmorrhysis*.

It appears at the first glance that the *arterial pressure* and its fluctuations are of the greatest importance for the circulatory conditions of the brain. However, Geigel has shown that the speed of the current in the capillaries depends, to a much greater extent, upon the degree of vascular tension than upon the height of arterial pressure. For, inasmuch as an increase of arterial pressure (especially also by greater heart action) causes a more marked dilatation of the vascular wall, secondarily an increase of the vascular tension, in proportion to the dilatation, occurs upon the extent of which it depends whether the speed of the current actually is greater or not.

The above statements show the importance of the existing degree of tension in the arteries of the brain, in regard to the blood supply to the brain. This vascular tension being mostly dependent upon nerve action, it is obvious, further, that the reaction of the vascular nerves is the most essential factor regarding a change of the circulatory conditions in the brain.

The diagnosis of *anæmia* and *hyperæmia* of the brain is based upon the occurrence of certain symptoms which are undoubtedly due to the changed cerebral function: Vertigo, spasm, disturbances of special senses, etc., and, above all, upon the ætiological factors. Upon the latter alone, as last resort,

depends the *differentiation* of anæmia and hyperæmia of the brain. For the different symptom-complexes proper which are *traditionally* ascribed to these conditions, are about the same in both instances. That such must be the case follows from the fact that conditions in which we believe that we may assume a spasm of the cerebral arteries, are generally classified as anæmias of the brain (whereas, according to the principles just set forth, they actually cause a better flow through the brain), and, *vice versa*, that venous stases in the brain are generally regarded as causes of cerebral hyperæmia, whereas it may be assumed that, here, owing to the diminution of the pressure in the arteries and to the increase of the same in the veins, a decreased velocity of the blood current, i. e., therefore, adiæmorrhysis is brought about, not considering the fact that in this instance, owing to the venosity of the blood, the regular nutrition and function of the nerve elements (the same as in a deficient flow of blood through the brain) suffer!

Adiæmorrhysis—Anæmia of the Brain.—*Clinically there cannot be any doubt of the occurrence of adiæmorrhysis of the brain, of anæmia in a sense thus modified, as a pathological condition, and it may be diagnosticated from the symptoms it causes.* They are: Tinnitus aurium, obscurity of vision to complete amaurosis, dilatation of the pupil, acceleration of the pulse, vertigo, inclination to vomit or vomiting, muscular convulsions or actual epileptic attacks, headache, deliria, insomnia, apathy; in severe cases, especially those with an acute course, somnolence or actual loss of consciousness (fainting, syncope).

The occurrence of these symptoms is *explicable*, to the greatest part at least, by deficient flow and nutrition of the brain. It should be considered, in the first place, that a deficient blood supply and deficient nutrition of the nerve elements, which accompanies it, are associated, primarily, with an *increase* of nerve irritability, i. e., therefore, decreasing nerve energy is accompanied with increased irritability before, with the higher grades of disturbance of nutrition, the irritability is abolished; it is known, furthermore, that certain centres of the central nervous system are stimulated especially by blood, which has a deficiency of oxygen. Considering this physiological fact, it is obvious that irritation symptoms of various kinds must occur in adiæmorrhysis of the brain. The most prominent are dilatation of the pupils (owing to irritation by the blood, which is poor in oxygen, acting upon the centre for the dilator of the pupil), vomiting, headache (owing to irritation of the nerves of the dura), insomnia, muscular convulsions (caused by irritation of the cortex). It is not to be wondered at that, besides the signs of an increased irritability, also those of a relaxation of the nerve reaction become prominent, thus the deficient control of the heart action by the fibres of the pneumogastric nerve, due to the deficient nutrition of the pneumogastric centre (acceleration of the pulse); furthermore, the somnolence and apathy, and, upon suddenly occurring deficient flow, loss of consciousness.

It is certain that the *vaso-motor centre* also is irritated by an increased venous condition of the blood, respectively by general anæmia. Thus the possibility is furnished to compensate the dangerous action of great losses of blood upon the brain. However, this compensation, as experience teaches us, either does not succeed at all, as a rule, in the given case, or insufficiently, so that, in spite of the introduction of a better flow through the brain, the symptoms of adiæmorrhysis manifest themselves in the further course of the disease.

Ætiological Diagnosis.—The complete picture of those manifestations of deficient flow through the brain or a part of the same is observed after *great losses of blood,* upon *accumulation of blood in the vessels of the abdominal cavity,* as after puncture of ascites or ovarian cysts, and, furthermore, in all conditions which are accompanied with deficient blood

formation, thus in *chlorosis, anæmia, leucæmia, cachexia, inanition* (hydrocephaloid), etc. Adiæmorrhysis is also caused by the various *conditions of stasis,* as, for instance, defects of the valves of the heart, weakness of the heart, emphysema, etc. Finally, we see syncope or other signs of adiæmorrhysis occur after sudden fright, great psychical emotions, mental exertions, etc. We are not able to tell how, in the latter instance, the interruption of the regular flow through the brain is brought about, whether by sudden paralysis of the vaso-motor nerves or by irritation of the depressor nerves or by some other agent. If an explanation is asked, there are plenty of means for this purpose.

Hyperdiæmorrhysis, Hyperæmia of the Brain.—Whereas, upon the presence of one of the above-named ætiological factors and of the well-known symptom-complex, the occurrence of adiæmorrhysis of the brain is explicable and diagnosticable, the diagnosis of *hyperdiæmorrhysis,* of *"cerebral hyperæmia"* in the former sense, meets with insurmountable difficulties. The symptoms which, according to the usual assumption, are peculiar to this condition, are practically the same as those occurring in deficient flow through the brain. The range of the *ætiological* factors of cerebral hyperæmias, also, is extremely limited, if we do not count the venous-stasis hyperæmias among the "hyperæmias" but classify them as adiæmorrhyses. Those cases in which pressure upon the abdominal aorta (abdominal tumours, fæcal masses) is said to cause a collateral fluxion to the brain, are probably more constructed than observed, and the cause of "active cerebral hyperæmias" which are due to an excessive action of the heart, especially to hypertrophy of the left ventricle, can be explained in a different manner, if they are observed at all. In such cases an atheromatous condition of the cerebral arteries may be the means, either in that it is the primary factor and that an atheroma of the arterial system in general, which is associated with it, is followed by hypertrophy of the heart, or in that the latter secondarily produce atheroma. In other cases, eventually, a temporary weakness of the heart prevails, occurring in the train of hypertrophy of the heart, in other cases, again, there exists, in place of the "effect upon the brain of hypertrophy of the heart occurring in the course of nephritides" a uræmic intoxication. Likewise, the "collateral arterial fluxion to the brain," which is observed in infectious diseases (diphtheria, erysipelas, etc.), should probably be ascribed not so much to altered circulatory conditions of the brain, but to the action of the respective toxines upon the latter.

I do not mean at all thus to deny that cases of an extreme flow of blood through the brain actually exist; but whether the latter is injurious to the brain and manifests itself in distinct symptoms, is another question which I am not able to decide. *At all events, it is impossible, according to my opinion, to diagnosticate such a hyperdiæmorrhysis.* To be honest, I must confess that, in a practice of 25 years, I never learned of a case in which it was necessary for me to assume, or in which I was able to diagnosticate, a cerebral hyperæmia in the meaning as discussed, i. e., with exclusion of passive stasis hyperæmias. Whoever, from redness of the face, concludes upon cerebral hyperæmia, establishes a supposition of the con-

dition of the circulation within the cranium, which he is not able to prove; such a conclusion would be permissible rather from the result of an ophthalmoscopical examination. But there are no cases known of a specific appearance of the fundus oculi in the meaning of a hyperdiæmorrhysis.

Cerebral Œdema.—What I stated regarding the impossibility of a diagnosis of cerebral hyperæmia, is, unfortunately, also true of another condition of the brain, of *œdema of the brain*, which is often diagnosticated and which can often be demonstrated at autopsies. *We may, according to my opinion, possibly, assume the presence of cerebral œdema, perhaps, also, pronounce its presence probable in a given case, but we must not diagnosticate it.* The symptoms which are named as characteristic of the same: Uncertainty of movements, sometimes also epileptic convulsions, loss of memory, somnolence, are of so vague a nature that no diagnosis can be made from them. If we consider, furthermore, the morbid conditions which lead to cerebral œdema, we again find here, primarily, venous stases due to heart diseases, emphysema, etc., various infectious diseases, and, furthermore, renal diseases, which, the same as to dropsy in other organs, may also give rise to cerebral œdema, cachexias, which cause marantic œdema generally, and especially also œdema of the brain, conditions, in short, all of which may also lead to intoxication or to adiæmorrhysis of the brain. *But, with certainty to differentiate the symptoms of the latter from those of cerebral œdema, I do not believe possible,* less so because œdema of the brain causes the width of the tract of the current to become narrower, thus diminishing the quantity of the blood flowing in the cerebral capillaries.

MENINGITIS—SUPPURATIVE AND TUBERCULOUS MENINGITIS—CONVEXITY MENINGITIS—BASILAR MENINGITIS.

The diagnosis of meningitis is among the frequent tasks of the physician. It is easy in many cases, quite difficult in others; I do not hesitate to confess that the diagnosis, in particular, of meningitis was more difficult to me in some cases than any other diagnoses in the sphere of brain diseases. This is caused in that meningitis manifests itself in very varying clinical pictures, and, further, certain symptoms that are peculiar to meningitis occur also in the course of other diseases, so that the one-sided consideration of the same is very apt to lead the diagnosis into a wrong direction. Thus we are quite liable, under the impression of severe cerebral symptoms, to assume an inflammation of the meninx, whereas the latter will be found smooth and glistening at the autopsy. Several pathological pictures of meningitis have been established, that of epidemic, serous, suppurative, tuberculous, etc., meningitis, in the attempt thus to facilitate the diagnosis. I find that there is little advantage in this method, but that we do best, rather, if we at first decide the question whether an affection, in particular, an inflammation, of the meninx should be considered present at all in the given case, and then only, after this has been accomplished, should endeavours be made to classify form and nature

of the meningitis; the latter part of the task is the easier and less important one.

Let us commence with the discussion of those symptoms, the presence of which empowers us to diagnosticate meningitis. As the latter mostly represents an exquisite *diffuse* affection of the surface of the brain, it is obvious that it manifests itself, primarily, in *general symptoms*. To these are added, owing to the concentration of the meningitis upon certain portions of the cortex of the brain, or owing to a propagation of the process upon the substance of the brain, *focal symptoms* which present a distinct type in that they occur, with a certain regularity, in groups.

General Symptoms.—The *general manifestations*, preceding and accompanying the focal symptoms, are: *Fever, headache, vertigo, vomiting, slowing of the pulse, disturbances of respiration, jactitation, epileptiform convulsions, general hyperæsthesia, deliria, involuntary discharge of fæces and urine,* furthermore, bordering upon focal symptoms, *lockjaw, grinding of the teeth, rigor of the extremities and stiffness of the muscles of the neck.*

Headache is a very constant symptom; the cause is to be looked for in a compressive action upon the dura with its abundance of nerves owing to the meningitic exudate itself, to the inflammatory œdema of the brain substance, and to the accumulation of fluid in the ventricles. The intensity of the headache is quite considerable; so long as the patient is conscious, his principal complaint is that of headache; even after coma has set in, its continuance is indicated in that the patient reaches to his head and that the groaning does not abate. I admit that the headache is less violent in one case than in the other; that, exceptionally, it may even be indicated only, and, besides, that its intensity may be subject to considerable fluctuations in the individual case. Nevertheless, I would advise, if *no* headache is present, to be very careful with the diagnosis of meningitis and to consider again and again whether the prevailing symptom-complex should not be traced to another cause than to meningitis.

Not less important in a diagnostical respect are the *jactitations* and *convulsions*. I doubt whether the former are always the expression of an irritation of the cortex; jactitations are, probably, mainly the effect of the severe affection of the general condition, of the pains, etc. On the other hand, the coming and passing epileptiform convulsions are direct symptoms of irritation of the cortex, the same is the case with the *deliria* which, in some instances, so completely predominate that the meningitis may have the entire appearance of delirium tremens. The patients, especially children, often start from the coma with a piercing cry (*"cri hydrencéphalique"*).

The *pulse* is usually retarded, at least at the onset of the disease, which is remarkable in comparison to the *increased temperature* (which, however, may fluctuate very considerably [100.4° to 105.8° F.]); later, the pulse becomes frequent, irregular, small. The retardation of the pulse is to be attributed to the increased intracranial pressure associated with meningitis; this increase is regularly observed also in other conditions connected with it. I leave it undecided whether the later frequency may, without hesitation, be referred to a paralysis of the pneumogastric nerve. The

respiration loses its regular type early; it is, above all, accelerated, sighing, later intermittent, finally to assume, in some cases, the character of Cheyne-Stokes respiration. *Vomiting* and *vertigo*, especially upon the patient assuming an upright position, are also, to conclude from their occurrence in other affections with an increased intracranial pressure, to be considered as the effects of the latter, as well as the *inability to swallow* and the *contraction of the pupils* which is often very pronounced. The latter may possibly be considered to be a spastic myosis due to an irritation of the cortical origin of the oculo-motor nerves or a compressive remote action upon the cerebral fibres of the oculo-motor nerves.

The *rigidity of the cervical muscles* is especially characteristic; *it is only rarely absent.* The patient, upon pronounced rigidity of the neck, presses the back of the head into the pillows, and he shows signs of violent pain if an attempt is made to force the head to the side, and, particularly, if we try to force it anteriorly towards the breast. The cause of this symptom, which in many cases decidedly confirms the diagnosis, is an irritation of the nerves which supply the muscles of the neck, especially also of the spinal accessory nerve. This irritation is a direct one if the meninx of the posterior cranial fossa and of the superior cervical spinal cord is affected by the inflammation; in other cases it must be ascribed to an irritation of certain cortical areas or to a remote action, posteriorly and inferiorly, of the intracranial pressure. The symptom is much more constant and much more pronounced than in the latter. To be counted in the same category are *trismus* and the *grinding of the teeth*, as well as the *spastic contraction of the abdominal muscles*, which causes the board-like tension or scaphoid retraction of the abdomen; the retraction may, in part, be due rather to a spasmodic contraction of the intestinal coils. *Rigidity of the extremities* and *convulsions* restricted to some portions of the body and muscles are also observed in many cases. Of still greater importance, diagnostically, than these symptoms of motor irritation is *hyperæsthesia of the skin and muscles*, especially of the muscles of the neck and calves, which, according to my experience, can quite usually be determined upon a more minute examination. The *reflexes* in the extremities are usually increased during the initial stages of the disease, later they are decreased to become entirely abolished towards the end of the affection.

Trophic, respectively vaso-motor, disturbances in the skin are occasionally observed. An unusual irritability of the vaso-motor nerves, especially, is manifest, so that even a slight stroking of the skin causes long-lasting redness; of the exanthemata we find *herpes*, rarely roseola and others.

The *fæces*, retained at first, are, later, discharged involuntarily; the same condition prevails with the discharge of the *urine*. The latter is excreted in small quantities and may contain *albumin*, probably due to an irritation of the fibres of the splanchnic nerve; but this albuminuria is, by no means, dependent upon the fever, as it may disappear with a decrease of the meningitic symptoms although the temperature rises. An example may illustrate this:

A soldier, twenty years of age, was admitted to the clinic with the symptoms of cerebro-spinal meningitis to which the patient succumbed. Temperature 101.1° to 103.6° F. for five days, during which time the urine proved to be markedly albuminous; suddenly, *on the sixth day, the albuminuria disappeared with improvement of the cerebral and spinal symptoms with simultaneous rise of the temperature* from 101.1° F. (fifth day) to 103.3° F. (sixth day). Later deliria recurred, with pupillary dilatation; then albumin was again present in the urine.

Sugar was also demonstrated in the urine in some few cases, besides albumin, and some investigators, including myself, noted a remarkable (also relative) increase of the secretion of phosphoric acid.

An extremely rapid wasting and the pronounced *emaciation* of the patients cannot be mistaken in most cases of meningitis, and they should, in my opinion, be ascribed to a cerebral action upon the metabolism, which, it is true, is not clear as yet. This wasting of the body substance developed, in one of my recently observed cases, especially markedly and rapidly—in an actually frightening manner, so that a robust, young man, who, until then, was well nourished, emaciated to a skeleton within a few days.

Focal Symptoms.—To the above-discussed, more general symptoms characterizing meningitis are superadded *focal symptoms* which usually establish the diagnosis, which, until then, is often uncertain, and place it upon a firm basis.

Anatomical Findings.—They are caused either by accumulation of masses of pus and especially of tubercles at certain *areas* of the surface of the brain or around the nerve trunks at the base, or by inflammatory affections of the nerves (hæmorrhages into the nerve sheaths, decay of the nerve medulla, etc.) or changes in the cerebral cortex itself (capillary apoplexies and softenings). The last-named changes are found both in the suppurative, as in the tuberculous form of meningitis, and are brought about by propagation of the inflammation or by tuberculous degeneration along the small vessels entering from the pia into the cortex. The larger vessels, also, especially the arteries of the Sylvian fossa, are very commonly affected by the inflammatory, respectively tuberculous, process, thus facilitating the development of thrombosis and its effects (areas of softening). If we consider these anatomical changes, the focal symptoms observed during life are usually quite explicable; in a certain number of cases, it is true, the anatomical findings are disproportionate to the clinical symptoms, so that a satisfactory explanation of the latter, based upon the result of the autopsy, is, very often, quite impossible.

The Various Focal Symptoms.—The most important of the focal symptoms are the *paralyses of the cerebral nerves*: Pareses of the facial nerve, etc., but especially also paralyses of nerves of the eye muscles, in particular of the oculo-motor nerve (strabismus, pupillary difference, etc.). Less frequent are paralyses of the extremities, either *monoplegias* (of the arm, very rarely of the leg), or exquisite *hemiplegias,* which may be preceded by symptoms of motor irritation in the parts paralyzed later. In some cases an exquisite *aphasia* is observed, namely the motor form, whereas the sensory form, although surely present occasionally, cannot be demonstrated positively with the general disturbance of consciousness. In such cases of pronounced aphasia we may assume a localization of the meningitic process in the cortex of the left frontal brain or, at least, a concen-

tration of the same in the region of the left Sylvian artery, an assumption which, in several cases, was confirmed by the autopsy.

Other symptoms of meningitis which are mentioned, such as icterus, deglutition pneumonia, etc., are not peculiar to meningitis as such and should not be considered diagnostically. But, on the other hand, the *ophthalmoscopical examination* is of great diagnostic importance in meningitis, and it should, therefore, never be neglected in a case of pronounced meningitis, nor in cases in which there is a suspicion of the presence of this affection.

Ophthalmoscopical Findings.—The ophthalmoscopical examination in meningitis, as soon as the acute or subacute inflammation extends to the meninges of the optic nerve, reveals the clinical picture of a so-called *optic neuritis*. The papilla of the optic nerve appears slightly swollen, cloudy and more or less red; its boundaries are indistinct, and the retina around the optic-nerve papilla is sometimes raised to a slightly cloudy wall. The venous vessels show a slight dilatation and are tortuous; often separate striated hæmorrhages are visible in the tissue of the papilla and adjoining retina. If the inflammation recedes, the papilla of the optic nerve shows, at first, a veil-like dimness and a rather reddish-yellow, dull, later a white appearance with distinct limitation (white atrophy). White spots can be found in the retina, and especially in the region of the macula, the same as in albuminuric retinitis; an occasional, simultaneous occurrence of tubercles of the chorioid membrane assures the diagnosis of a meningitis to be tuberculous. If the meningitic affection, from the onset on, shows a more chronic character, the cloudiness and redness of the papilla is comparatively slight; the atrophic discoloration is visible much oftener. The venous vessels are, sometimes, accompanied with white streaks. The functional disturbances, so far as their conduct can be at all determined, appear to present rapidly changing fluctuations; they may also be developed in a varying degree in both eyes. Decrease of the faculty of vision with gradual loss of sight, moderate concentric narrowing of the field of vision, in the later stages colour blindness, will probably be the principal symptoms.

Modifications of the Course.—As was stated in the general description of the individual symptoms which are to be considered in the diagnosis of meningitis, the clinical pictures of the course of the disease vary.

Seat of the Meningitic Changes.—This depends, in the first place, upon the *seat* and *extension* of the inflammation. The clinical pictures vary, especially according to the convexity or the base of the brain being affected; the former is generally more affected in the suppurative, the base more in the tuberculous form, so that the latter has been designated as *basilar meningitis*, the former as *convex meningitis*. However, such a differentiation is justified only in a very limited measure. It is correct that, in cases of tuberculous meningitis, especially in those that run a subacute and chronic course, the meningitic changes, according to the dissemination of the tuberculous inflammation in the wall of the vessels, are usually restricted to the base, and that the gelatinous-serous exudate is found developed principally in the basal portions from the pons to the chiasm and in the Sylvian fossa. But it is quite as true that this condition prevails also, although not as often, in suppurative meningitis, and that, on the other hand, the process very generally spreads from the base of the brain to the convexity in tuberculous meningitis.

Basilar Meningitis.—*To an affection of the base* point, of course, paralyses of the cerebral nerves, and, to a certain extent, hemiplegias, the origin of which is, usually at least, ushered in from the base by occlusion of the vessels. However, there are numerous cases in which paralyses exist, and in which we are unable to connect them with anatomical changes that are demonstrable *post mortem*. But, on the other hand, the general symptoms are, as a rule, by no means less pronounced in basilar meningitis than in convex meningitis; they must be ascribed especially to the internal hydrocephalus ("hydrocephalus acutus") and the increased intracranial pressure, which are so very markedly developed in basilar meningitis.

Convex Meningitis.—To convex meningitis point, besides the general symptoms, well-developed monoplegias and localized epileptic convulsions, which always originate in the same extremity or are restricted to the same, and principally psychical disturbances. However, it follows from the above remarks that *localizing diagnoses* are not admissible in by far the majority of cases of meningitis, and the general practice fully confirms this principle. The greater the number of cases of meningitis which we have an opportunity to observe and to dissect, the more careful will we become in our assumptions as to seat and extension of the meningitic changes.

Combination with Spinal Meningitis.—*Combinations of cerebral meningitis with spinal meningitis* are quite common—they are much more frequent than was supposed formerly. This is true not only of epidemic cerebro-spinal meningitis, but especially of tuberculous meningitis also. In some cases it seemed to me that, in them, it was a question of a migration of the tubercle virus, which progressed from lung and pleura locally to the meninx of the thoracic part of the spinal cord, and from here upward towards the base of the brain. But it is certain that, in most cases, the propagation of the process towards the brain does not occur by contiguity, but is due to general embolic transmission of the tuberculosis by way of the lymph- and blood vessels. The simultaneous affection of the spinal meninges manifests itself particularly by stiffness and painfulness of the vertebral column. As most symptoms of spinal meningitis (see p. 530): Hyperæsthesia, muscular spasms, paralyses of the extremities, etc., are found also in cerebral meningitis, that is not complicated with spinal meningitis, and which explain themselves as the effects of the cerebral irritation, it is obvious that a strict differentio-diagnostical separation of the symptoms is not possible in this respect.

Diagnosis of the Nature of Meningitis.—After the diagnosis of meningitis has been made, in consideration of the above symptom-complex, another question which materially supports and supplements the diagnosis is to be decided, viz., *which cause is at the bottom of the meningitis, respectively, what is its character?* As is well known, we distinguish between *suppurative, tuberculous* and *serous meningitis.*

Serous Meningitis.—The latter form, *serous meningitis,* owing to the efforts of Quincke, has become more acknowledged and noted during the last years. The occurrence of the same, according to my experience also, can, therefore, no longer be doubted; only I believe that this form of meningitis is diagnosticated much too often at present. With a disease, the positive demonstration of which, even post mortem, is difficult, the symptoms of which are ambiguous *intra vitam,* and which almost always terminates in recovery so that a diagnosis of the presence of this affection cannot be controlled by an autopsy, we should, under all circumstances, be very conservative in forming a diagnosis.

The diagnosis of serous meningitis, the clinical symptoms of which are the same as in other forms of meningitis (fever, vomiting, headache, rigidity of the neck, hyperæsthesia, irregularity of the pulse and of the pupillary reactions, deliria, coma, paralysis of individual cerebral nerves caused by cerebral pressure, and, quite usually, stasis neuritis of the optic nerve) has increased in probability by the result of the *lumbar puncture* invented by Quincke. From the condition of the cerebro-spinal fluid thus obtained, certain conclusions can be drawn as to the presence of an inflammatory affection of the meninges and as to its nature. But we should be able, at all events, to determine an increased amount of albumin of the puncture fluid of 1 per cent and above (the normal amount is 0.5 to

1 pro mille) and an increased pressure under which it passes out (normal pressure 40 to 60 mm. water), to be justified in considering the presence of a serous meningitis. But both these conditions are found also in other affections of the brain, thus in brain tumours. It is of greater importance, therefore, in my opinion, if we succeed in demonstrating the presence of sufficient numbers of leucocytes in the puncture fluid, and if the latter does not, at the same time, appear materially cloudy. But, nevertheless, even in undoubted, i. e., in bacterially demonstrated, epidemic cerebro-spinal meningitis a water-white exudate has been obtained by puncture! Ætiological, respectively predisposing, factors of serous meningitis are considered: Traumatism of the head, mental overexertions, otitis media (which, however, much oftener leads to suppurative meningitis), influenza. The same as in other serous exudates, sometimes no bacteria, at other times cocci or tubercle bacilli (see below), have been found in the exuded fluid (obtained by lumbar puncture).

Epidemic Cerebro-Spinal Meningitis.—The occurrence of *epidemic cerebro-spinal* meningitis is securely founded and can, usually, be demonstrated easily. In the first place we should note whether, at the time in which such a case is considered diagnostically, an *epidemic of cerebro-spinal meningitis* is present in the locality. We know to-day, especially by reason of the exact investigations of H. Jäger, that epidemic cerebro-spinal meningitis is caused by a certain kind of coccus, namely by the *diplococcus intracellularis meningitides* discovered by Weichselbaum. This important fact can be used also clinically in that the meningococcus was frequently found in the cerebro-spinal fluid obtained by lumbar puncture; the demonstration of the specific bacteria in the nasal mucus of the patients was also accomplished successfully.

However, we do not mean to conceal the fact that, in some epidemics of cerebro-spinal meningitis, pneumococci (Fränkel) were found, sometimes with, at other times without, the diplococcus intracellularis. This fact will allow of the conclusion that, eventually, a pneumococcic infection may also be the cause of epidemic cerebro-spinal meningitis.

Besides the positive finding of specific bacteria in the lumbar puncture fluid, we must, to some extent, consider in the differential diagnosis, to assume the presence of epidemic cerebro-spinal meningitis in contrast to other forms of meningitis, that, in epidemic meningitis, certain symptoms become manifest which point to a general infection and which do not, or only rarely, occur in other forms of meningitis. This is the case with the exanthems roseola or herpes, the latter of which occurs rather constantly and very extensively on the face, less so on the trunk and extremities, and also with articular inflammations, which are remarkably frequent in some epidemics of cerebro-spinal meningitis, as I am in a position to confirm from my own experience. But I cannot admit that the rigidity of the neck is especially intense in the epidemic form, and, on the other hand, hyperæsthesia and stiffness of the back are entirely absent in the non-epidemic suppurative and tuberculous forms of meningitis. I believe it is absolutely impossible to distinguish, from the mere symptom-picture, the

sporadic or first cases of epidemic cerebro-spinal meningitis from other forms of the affection. As to the remainder, I refer to the special discussion of cerebro-spinal meningitis (see Infectious Diseases).

The same stress must be laid also upon the ætiological standpoint, according to my opinion, in the diagnosis of the common suppurative and tuberculous forms of meningitis.

Tuberculous meningitis is of considerably more frequent occurrence than (non-epidemic) suppurative meningitis. The presence of the former should, therefore, be first thought of in every case of meningitis. It is always due to the entrance of tubercle bacilli into the body. The primary focus, from which originated the transmission of the virus to the meninges, is found in the majority of cases, upon a careful examination of the body. In some of the cases this is not possible *intra vitam*, because it is a question of tuberculosis of the bronchial glands, mesenterial glands, etc., from which the importation took place, or because it may occur, although rarely, that the bacilli enter the body without leaving manifest changes at the point of entrance. Tuberculous meningitis is found most frequently in synchronously existing *pulmonary tuberculosis* of longer or shorter duration or in *tuberculous pleuritis*; the most painstaking examination of the respiratory organs should, therefore, never be omitted. It is also necessary to examine the urine, the testicles, the prostate gland and the ovaries for the eventual presence of *urogenital tuberculosis*. Other tuberculous primary affections: *Tuberculous affections of the bones and joints*, especially *affections of the petrous bone, tuberculous glandular tumours* of the neck, etc., can scarcely be overlooked. Traumatism of the head was, in some cases, the cause of localization of tuberculosis in the meninges, which, after three days, produced recognisable symptoms of commencing tuberculous meningitis. The meninges are some of those tissues in which the bacilli, upon their transmission, are especially liable to be retained, and it seems that the meninges of the infantile organism are particularly predisposed to infection. Meningitis occurring in children is actually almost always tuberculous, even if it is not possible positively to locate the source of infection. The *course of tuberculous meningitis* differs from that of other forms of meningitis in that the onset of tuberculous meningitis is usually preceded by a decidedly longer prodromal stage with loss of appetite, constipation, emaciation, insomnia and general sensation of illness, in that the fever, upon the whole (especially at the acme of the disease) is only moderate (100.4° to 102.2° F.), the course of the symptoms is less turbulent, and in that those manifestations predominate which point to an affection of the base of the brain. But all these symptoms are criteria which are of no value for a precise diagnosis. The absence of peptone, respectively of albumose, in the urine is a much more valuable sign in that their excretion usually indicates that a process of suppuration is taking place in the body. However, we possess absolutely certain differentio-diagnostical signs in the demonstration of chorioid tubercles in the fundus oculi and in the finding of tubercle bacilli in the cerebro-spinal fluid obtained by lumbar puncture. This fluid is clear in the majority of cases, and tubercle bacilli can be demonstrated in the sediment, so that the exami-

nation of the lumbar puncture fluid forms a valuable support of the diagnosis of tuberculous meningitis.

Suppurative Meningitis.—If there is absolutely no reason to assume the presence of a tuberculous meningitis, we must now review those causative factors which lead to *suppurative meningitis*; there are quite a number of them. It occurs by far the oftenest that an inflammation which produces a suppurative meningitis spreads from the immediate neighbourhood of the meninges to the latter. The most frequent sources of such a *suppurative meningitis, brought about by contiguity* (*transmitted by way of the lymph-vessels or usually of the veins*), *are caries of the petrous bone and purulent inflammations in the ear* (*otitis media*). Less frequent causes are: Purulent catarrhs of the frontal sinuses, operations of the nasal cavity, the orbits or in the aural cavity which were not performed with sufficient care, traumatism, especially complicated fractures of the bones of the skull, neglected wounds or deep-seated furuncles and abscesses of the scalp. A purulent sinus thrombosis is, in many cases, the cause of a transmission to the pia mater. The same as from external sources, a transmission of the suppurative inflammation to the meninges may occur also from internal regions, namely from a *cerebral abscess* which extends to the surface of the brain or perforates, either into the subarachnoid space itself or into the ventricles, especially into the third ventricle, from which locality the infection towards the base of the brain is easily brought about.

Metastatic Meningitis.—However, the virus causing suppurative inflammation (staphylococci and, above all, the streptococcus pyogenes) are transmitted to the pia mater not only from adjoining structures, but we know from experience that it is also introduced from remote parts of the body by the blood; suppurative meningitis, in such cases, forms a partial manifestation of *septico-pyæmia,* respectively of *puerperal fever.* It is then usually complicated by endocarditis, articular inflammations, miliary abscesses of the lungs, sæptic nephritis, cutaneous suppurations, etc. Meningitis is also noted to occur in the course of certain infectious diseases, *fever* and *acute articular rheumatism,* diphtheria and other infectious diseases. In these infectious meningitides either the specific bacteria of the respective infectious disease (pneumococci, typhoid bacilli), or the usual pyogenic organisms, especially the streptococcus pyogenes, have been demonstrated to be the cause of the inflammation of the meninx. It is possible in rare cases, as I was taught, recently, by a fatally terminating case, that the development of multiple syphilomata in the meninges and in the cortical portions of the brain may simulate a meningitis; it is necessary, therefore, also to consider the possibility of this source of infection in the differential diagnosis.

Differential Diagnosis of Meningitis.—If the entire symptom-complex and the ætiological basis point to the existence of a meningitis, the latter can be diagnosticated with certainty in many cases. However, every experienced diagnostician will agree with me that wrong diagnoses are possible even then. This is the case especially if, furthermore, the examination of cerebro-spinal fluid eventually obtained by lumbar puncture is

without any characteristic result, if the ophthalmoscopical findings are negative, and if the ætiology of the case does not furnish any points of support. In such cases we are restricted, in the diagnosis, solely to the clinical picture as such, and even this may eventually present indistinct symptoms and not show some of the principal manifestations. We are compelled, under such circumstances, to consider in the differential diagnosis a number of diseases which present a clinical picture that is similar to that of meningitis. These are, principally, delirium tremens, hysteria, uræmia, sepsis and enteric fever, which must be considered here, even without the last-named leading to meningitis.

Delirium Tremens.—As to the differentiation between *delirium tremens* and meningitis, it is easy, of course, if the clinical picture of meningitis is the usual one. If, however, the latter takes the so-called "delirious" form, i. e., if the psychical symptoms become prominent, and insomnia, restlessness, tremor and deliria manifest themselves, the differential diagnosis may become quite difficult. Epileptic convulsions occur in both affections, the same as partial convulsions in the region of the facial nerve and in the extremities. The fever is not determining, either, as it may be absent in meningitis, on the one hand (which is the usual occurrence in delirium tremens), and, on the other hand, elevations of temperature to 104° F. and above occur particularly in fatally ending delirium. The differential diagnosis, in such cases, must consider, primarily, the presence of focal *symptoms*; if they are present, it points decidedly to meningitis. Above all, the prominence in the clinical picture of rigidity of the neck and of intense headache, and a positive result of the ophthalmoscopical picture, may guide the diagnosis towards meningitis. It may be stated, incidentally, that the cheerful type of deliria, which is generally characteristic of alcoholic deliria, is exceptionally found in meningitis also, according to my experience, so that it is not permissible to utilize this cheerful type at once against the diagnosis of meningitis.

Hysteria is much less the cause of a wrong diagnosis, if rigidity of the neck, vomiting, convulsions and apparently severe stupor in hysterics simulate meningitis. However, the similarity of both clinical pictures is always a superficial one and can render the decision doubtful for a short while, at the most. A closer observation will show special traits in the picture which guide the diagnosis in the right direction. The effect upon the convulsions by pressure upon certain parts of the body, the stupor due to pronounced sensory irritations, the, occasionally, cataleptic character of the spasmodic condition, the restriction of hyperæsthesia to circumscribed areas of the skin, the exaggerated stress laid by the patient upon some pathological symptoms, etc., will not permit the expert physician to be in doubt for any length of time that he is not dealing with a severe anatomical change of the brain, but with hysteria. Besides, the ætiological basis, so important in the diagnosis of meningitis, viz., optic neuritis, paralysis of the facial nerve, and of the muscles of the eye in the further course of the disease, etc., is absent.

Uræmia is sometimes to be considered in a differentio-diagnostical respect. It has in common with meningitis: Headache and vertigo, vomit-

ing, deliria, coma, convulsions and the irregular, stertorous respiration, and is the cause of diagnostic errors especially when the convulsions are restricted to a few groups of muscles, if partial contractures occur, if the temperature rises temporarily, or if even transitory unilateral paralyses occur (which may appear in uræmia, at least, although very rarely). The most expert diagnostician may be led astray in such exceptional cases; for the most natural sign, the demonstration of albumin in the urine, is of a limited value only, because albuminuria may also occur, as is well known, in association with meningitis. The most important characteristic, in these cases, is the ophthalmoscopical finding, albuminuric retinitis, which is scarcely ever absent in cases of chronic nephritis that lead to uræmia. But in cases of acute nephritis with uræmia, in which the symptoms of retinitis are absent, the condition of the urine (the profuse admixture of blood and epithelial casts) and the development of cutaneous œdema furnish the determining criteria. The hypertrophy of the heart in chronic nephritis can also be utilized in the differential diagnosis. We should, eventually, also consider the fact that, exceptionally, meningitis may sometimes complicate nephritis.

Enteric Fever.—It has occurred most frequently, furthermore, that severe disturbances of the activity of the brain in the course of *enteric fever* have been confused with meningitis. Prostration, stupor, restlessness, insomnia, jactitation, headache and deliria, slight muscular convulsions and retardation of the pulse may be present in a similar manner in both diseases. To the presence of severe enteric fever (in contrast to meningitis) point, in such cases especially: Regular course of the temperature and regular pulse, roseola (which is only exceptionally observed in meningitis), diarrhœas, diazo-reaction of the urine and the positive result of the Gruber-Widal reaction. Meningitis is favoured by: Development, although slight, of focal symptoms, rigidity of the neck and hyperæsthesia, as well as pronounced epileptic attacks, the intensity of the headache, vomiting, the irregular respiration, herpes, eventually optic neuritis and a positive finding of lumbar puncture. Some of these "meningitic" symptoms, such as hyperæsthesia, rigidity of the neck, herpes and vomiting, occur, exceptionally, also in the course of enteric fever, and, yet, no sign of a pathological nature can be determined post mortem in the central nervous system. Swelling of the spleen, although found in both affections, is much more constant in enteric fever, so that the absence of splenic tumour points at once to meningitis. I wish to call attention also to the fact that meningitis, in the course of enteric fever, is sometimes observed as a complication, as I had occasion again recently to convince myself from a marked example.

Septicopyæmia, finally, in such cases in which severe cerebral symptoms are observed *intra vitam,* does not represent a meningitis (which, it is true, is not particularly rare in sepsis), but a septic intoxication of the entire central nervous system or multiple smallest hæmorrhages of a metastatically septic character in the meninges and in the substance of the brain! The ætiological basis of the last-named affections of the brain being the same as that of septic meningitis, the enormous difficulty of the differential

diagnosis in such cases is quite obvious. As a matter of fact, a diagnostic mistake, i. e., the supposition that meningitis is present when post mortem the meninges prove to be smooth and glistening, can sometimes not be avoided under such circumstances. In cases of the latter kind I found upon clinical observation, the same as in meningitis: Coma, deliria, convulsions, contractures of the arms and hands, pronounced general hyperæsthesia, herpes (of the hands), inequality and, later, contraction of the pupils, divergence of the bulbi, respiratory changes in the form of Cheyne-Stokes's phenomenon, in one case even rigidity of the neck and yet no meningitis! It is obvious that there can no longer be a question of a differential diagnosis in such cases, especially because the lumbar puncture may also prove negative and the ophthalmoscopical examination may, eventually, show retinal hæmorrhages in both affections.

Meningitis in Childhood, Hydrocephaloid.—The diagnosis of meningitis, especially that of tuberculous meningitis, requires particular attention if occurring in *children.* The onset of the pronounced meningitic symptoms is generally preceded in such cases by a prodromal stage lasting for days, even for a week and more, and with apparently innocent symptoms: Loss of appetite, retained fæces, restless sleep, etc., until the headache, the vomiting, the uncalled-for sighing and piercing yelling, which evidence the severity of the disease, the grinding of the teeth, strabismus, paralyses and convulsions, the scaphoid abdomen, hyperæsthesia and coma remove the doubts as to the presence of meningitis. On the other hand, it must not be forgotten that deliria and convulsions occur in children very frequently in every febrile disease and should not at once raise a suspicion of meningitis. Furthermore, in debilitated young children, especially in those which are weakened by gastric and intestinal catarrhs, it may occur that inanition manifests itself by symptoms of cerebral anæmia which somewhat resemble meningitic manifestations. Restless sleep, deliria and rigidity of the neck, later, also, convulsions and coma, take place under such circumstances; the pupils, at the same time, are wide, reacting sluggishly, the pulse is small, irregular, respiration superficial, frequent and irregular, the abdomen soft, the extremities cool, the fontanelles sunken, the bones of the head pushed over one another, the temperature subnormal. Marshall Hall has designated this condition as "hydrocephaloid"; the differentiation of the latter from meningitis is not difficult. The diagnosis is determined, except by the ætiology, principally by apyrexia, sunken fontanelles and the absence of all focal symptoms, i. e., the presence of hydrocephaloid must be assumed and meningitis excluded. An ophthalmoscopical examination may also turn the diagnosis in the right direction in that the presence of optic neuritis points directly to meningitis, whereas we may look for a slight filling of the retinal vessels in hydrocephaloid.

THROMBOSIS AND PHLEBITIS OF THE SINUSES OF THE BRAIN—SINUS THROMBOSIS.

Origin of Sinus Thrombosis.—The same as in other veins, a thrombus formation may be brought about in the venous sinuses of the dura mater by marasmus, respectively weakness of the heart (marantic thrombosis); it is favoured by the anatomical condition of sinuses, in that the latter possess an angular lumen and fixed walls and, besides, are in part permeated by reticular trabeculæ. Marantic thromboses are most frequently found in the superior longitudinal sinus and in the cavernous sinus in de-

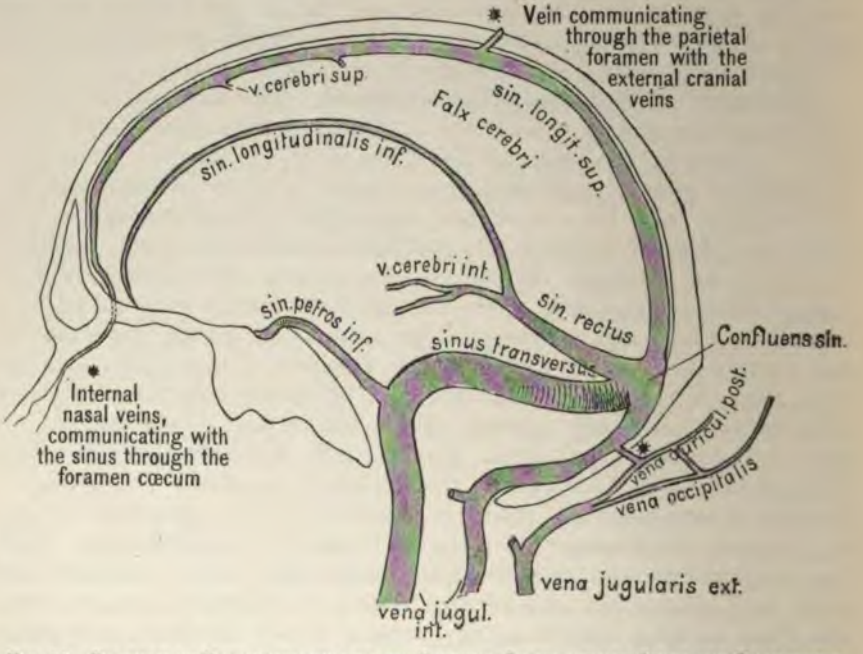


FIG. 64.—DIAGRAM OF COMMUNICATIONS OF THE SUPERIOR LONGITUDINAL SINUS AND TRANSVERSE SINUS WITH EXTERNAL VEINS (*).

bilitated individuals weakened by long-lasting diarrhœas and by severe infectious diseases (enteric fever, variola), in patients suffering from carcinoma, phthisis, etc. *Severe chlorosis*, in particular, may also lead to thrombosis of the cerebral sinus, as was recently determined in several cases by Kockel, etc. A thrombosis may also develop by compression of the sinus from the neighbourhood, thus in brain tumours and fractures of the skull.

More important as a source of sinus thrombosis is *inflammation of the sinus wall* (with subsequent thrombosis), which may develop in all those localities in which inflammatory, especially suppuratively inflammatory, processes take place in the neighbourhood of the dura mater. Thus suppurative sinus thromboses are brought about in brain abscesses, suppurative leptomeningitis (by means of the walls of cerebral veins adjacent to the pia mater), in purulent processes in the bones of the skull, in the nasal cavity, orbits and, above all, in the internal ear. As the veins of the face and neck, as we shall see later on, are partly in communication with the sinuses, it is obvious that erysipelas and abscess of the face, cervical phlegmons, etc., may also give rise to a direct propagation of the suppurative inflammation upon

the sinuses. Finally, sinus thrombosis sometimes forms a link in the chain of symptoms of septicopyæmia or of "puerperal fever."

After the thrombosis has entered the venous sinus, the coagulum often continues into the sinuses which communicate with each other and into the veins which enter into the (thrombosed) sinus from without and within (from the brain); for instance, in thrombosis of the cavernous sinus and inferior petrosal sinus (see Figs.

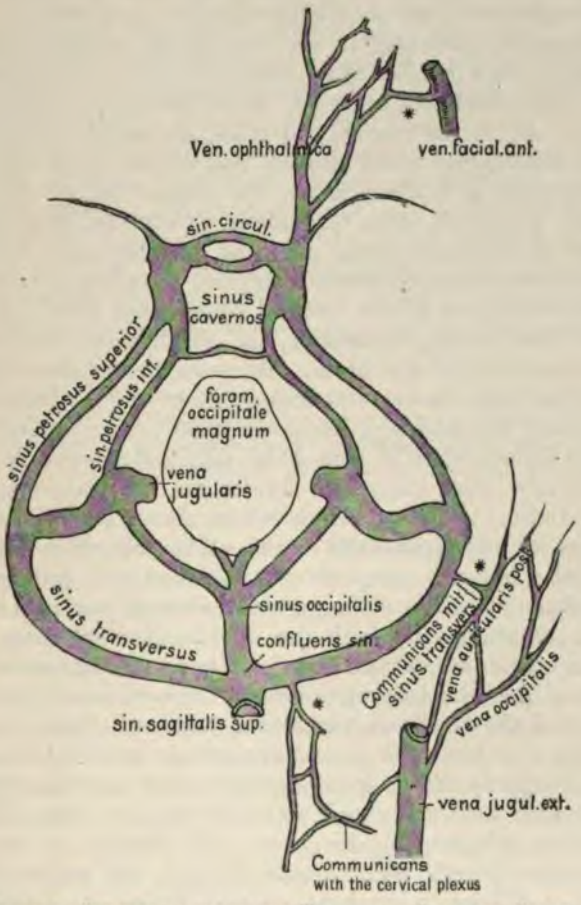


Fig. 65.—Diagram of Communications of the Transverse Sinus and Cavernous Sinus with External Veins (*).

64 and 65) into the internal jugular vein, some of the cerebral veins, of the cervical and occipital veins. But, at all events, the terminal region of the veins discharging into the sinus are overfilled with blood, the tissues—the soft parts as well as the substance of the brain—œdematous. Furthermore, hæmorrhages occur in the meninges and in the brain, in the latter, also, (red) areas of softening of greater or lesser extent; under such circumstances brain abscess may develop in suppurative thrombophlebitis.

Cerebral and Focal Symptoms.—The *symptoms* which are observed when sinus thrombosis is present, vary considerably in the different cases.

It is certain that extensive sinus thromboses are found, and *not one* cerebral symptom ever pointed to this fact during the life of the affected individual. The course of sinus thrombosis, as such, may be quite *latent*, therefore, and the condition may not be diagnosticable. In other cases, in which the engorgement assumes greater proportions towards the inner portions, there occur headache, vomiting, epileptic convulsions, rigidity of the neck, dulness of the sensorium—general symptoms to which are added, furthermore, according to the seat of the capillary hæmorrhages and softenings in the substance of the brain, various focal symptoms (paralyses, etc.). Such cerebral symptoms, which are explicable as the effects of local stasis, are, in the majority of cases, accompanied with the manifestations of meningitis, which usually complicates sinus thrombosis, or with the symptoms of septicopyæmia, the clinical picture of which then predominates. Therefore, sinus thrombosis, surrounded by these affections or by abscess of the brain, could never be diagnosticated, not even surmised, unless certain manifestations take place occasionally which are connected with the location of the cerebral sinuses and with the circulatory disturbances caused by the thrombosis, and which point directly to an obstruction of the sinus of the brain. In order to elucidate the cause of those circulatory disturbances and compressions which, solely, allow of a diagnosis of sinus thrombosis, a discussion upon the anatomical conditions of the various sinuses must be considered absolutely necessary.

The, clinically, most important sinuses are the transverse sinus, the superior longitudinal sinus and the cavernous sinus (see Figs. 64 and 65).

Thrombosis of the Transverse Sinus.—The *transverse sinuses*, commencing at the internal occipital protuberance and extending in the transverse sulcus anteriorly to the jugular foramen, pass into the internal jugular vein, simultaneously with the inferior petrosal sinus. The following vessels, besides the various sinuses entering the torticular Herophilii, discharge blood into the transverse sinus: Cerebral veins (from the posterior portion of the cerebrum, cerebellum and tentorium), external cranial veins, viz. (by means of a communicating branch passing through the mastoid foramen) the occipital and posterior auricular veins (which communicate with each other and with the temporal veins and by their union form the external jugular vein) and, finally, the cervical veins through a venous branch which passes through the posterior condyloid foramen and establishes a communication between the transverse sinus and the cervical reticulum. It follows from these last statements that, if a transverse sinus is occluded by a thrombosis, *venous stasis and* (*painless*) *œdema eventually occurs behind the auricle at the mastoid process and in the cervical region* (Griesinger). *The external jugular vein of the affected side also appeared less filled in some cases than that of the healthy side,* because it discharges its blood (if the thrombus, eventually directly palpable, continues from the transverse sinus into the internal jugular vein) with greater facility into the slightly filled internal jugular vein (Gerhardt).

It may apparently happen, in rare cases, that paralytic symptoms occur also in the region of the vago-accessorius and hypoglossal nerves. Thrombosis of the trans-

verse sinus is most frequently due to *inflammatory processes of the internal ear*, especially to tuberculous caries of the petrous bone.

Thrombosis of the Longitudinal Sinus.—The *superior longitudinal sinus* extends from the foramen cæcum of the frontal bone, communicating through the latter with the *internal nasal veins*, in the upper, attached border of the major falx cerebri along the inner surface of the cranial vault to the internal occipital protuberance. The sinus communicates, aside from its afflux from the brain (superior cerebral veins) and from the falx cerebri, by emissaries (especially by a communicating branch passing the parietal foramen) with the external cranial veins. Therefore, in thrombosis of the superior longitudinal sinus there occurs either *a congestion of the internal nasal veins* (eventually profuse *nose-bleed*) and cyanosis in the region of the anterior facial vein, *or an increased filling of the veins from the temporal region to the vertex*, which is especially distinct in small children. *The large fontanelles* (at the moment of beginning of a thrombosis they are said to sink in) *are large and tense, owing to stasis in the cerebral nerves and to increase of the intracranial pressure.*

The most frequent cause of thrombosis of the longitudinal sinus is the cardiac weakness which occurs in the train of exhausting diseases causing cachexia (carcinoma, phthisis, etc.); i. e., "marantic" thrombosis usually affects the longitudinal sinus. But a "phlebitic" thrombosis of the longitudinal sinus occurs also and may be looked for especially in inflammatory affections of the cranial vault and of the nasal cavity.

Thrombosis of the Cavernous Sinus.—The stasis symptoms are particularly well developed in thrombosis of the *cavernous sinus*. The *cavernous sinuses*, situated near the sella turcica, at both sides of their upper ends receive the *ophthalmic veins*, the upper branch of which communicates with the anterior facial vein at the inner angle of the eye. The effects of thrombosis of the cavernous sinus are, therefore, provided the superior and inferior ophthalmic veins are involved in the thrombosis, as follows: *Œdema of the lid of the eye and of the conjunctiva*, also, eventually, of a greater portion of the face (owing to the communication between the superior ophthalmic vein with the anterior facial vein), *manifestations of stasis in the veins of the retina—congestion and tortuosity of the same, œdema of the retina and papilla, exophthalmus.* The thrombosis may extend, furthermore, upon the central vein of the retina, when those ophthalmoscopical changes will become prominent which are characteristic of a partial or total occlusion of the above vessel. If the thrombosis is of septic origin, the manifestations of a phlegmonous inflammation of the cellular tissue of the orbit may become prominent. As, furthermore, the lateral wall of the sinus is formed by the membrane of the dura mater within which extend the oculo-motor and trochlear nerves and the first branch of the fifth nerve, and, besides, the abducens nerve passes directly through the cavernous sinus, it is obvious that, in thrombosis of the sinus, especially when it is a question of an inflammation of the same, these nerves are very liable to become affected, and paralyses of the muscles of the eye, as well as *neuralgias* in the region of the first branch of the fifth

nerve and *neuroparalytic ophthalmia,* occur in the course of thrombosis of the cavernous sinus.

The sources of thrombosis of the cavernous sinus are usually inflammatory processes in the orbit and in the face, more rarely otitic processes, which cause usually thrombosis of the transverse sinus, sometimes, however, also thrombosis of the superior and inferior petrosal and of the cavernous sinuses.

The above-described effects of sinus thromboses, so far as they refer to a compression of nerves, and to obstruction to the discharge of venous blood from the periphery, render it possible that this disease, which is so difficult to diagnosticate, can be recognised in some cases at least. But as, unfortunately, these effects are by no means constant, the diagnosis of sinus thrombosis rarely attains a desirable degree of certainty. The diagnosis may, eventually, be supported by the occurrence of an *embolism of the pulmonary artery,* the course of which, if sinus thromboses in a condition of puriform dissolution are the sources of such a pulmonary embolism, presents the picture of pulmonary abscess.

MENINGEAL HÆMORRHAGES—HÆMATOMA OF THE DURA MATER.

Cause of Meningeal Hæmorrhages.—Hæmorrhages into the tissue of the meninges or between the meninges (epidural and subdural, subarachnoid and subpial) are, in general, rare occurrences. Relatively often there occurs only the so-called *hæmatoma of the dura mater,* which, likewise, in a minority of cases only is the expression of primary hæmorrhage of the dura mater, but, as a rule, the consequence of chronically inflammatory processes in the fibrous meninx, so that the inflammatory deposits at the inner surface of the dura are infiltrated by vessels which then rupture and cause hæmorrhages (*pachymeningitis interna hæmorrhagica*). The hæmorrhages are situated mostly over the parietal lobe, rarely at the base of the brain, and occur *intermittingly,* form separate layers deposited at various periods and detach the inflammatory membranes from the dura, so that the masses of blood appear sacculated; if the most inferior neo-membrane, facing the cerebrum, ruptures, an extravasation of blood into the subdural space occurs. The *gradual* development of a hæmatoma is of importance in the diagnosis, as well as its *ætiology.* The most important factors in the latter respect are: Chronic affections of the brain, atrophy of the brain, mental affections, especially *dementia paralytica* and *dementia senilis.* The decrease of the volume of the brain, especially the atrophy of the convolutions, in these conditions probably favours primarily the origin of pachymeningitis interna hæmorrhagica. In other cases, as in hæmatomata which occur subsequently to diseases with changes of the blood and blood vessels: Scurvy, leucæmia, pernicious anæmia, renal affections, severe infectious diseases (enteric fever, variola), acute yellow atrophy of the liver, marasmus, etc., we must rather think of hæmorrhages which *originate primarily.* The latter, of course, also applies to free *meningeal hæmorrhages* which are caused generally by *severe traumatism affecting the skull;* such traumatic meningeal hæmorrhages are especially observed in the newborn caused by a difficult, artificial delivery. Furthermore, *rupture of an aneurism* of the cerebral arteries may be followed by extensive accumulations of blood in the meningeal space. The most frequent cause of hæmatoma is, acknowledgedly, *chronic abuse of alcohol,* the harmful effect, etc., of which may possibly manifest itself also in cerebral hyperæmia and atheromatous or fatty degeneration of the vascular walls which, in the course of time, leads either to chronic inflammation or to rupture of the vessels. **The above-named ætiological factors and, very particularly, the eventual presence of mental affections and alcoholism in the history of the patient must always be considered in the diagnosis, because, as we shall see, a consideration of the ætiology facilitates, at least slightly, this uncertain, difficult diagnosis.**

Diagnostic Points of Support.—Meningeal hæmorrhages occur, comparatively often, *without symptoms.* This is the case when the hæmorrhage is scanty or is brought about very gradually; meningeal hæmorrhages may be latent especially in the new-born. In some cases, it is true, more severe cerebral symptoms are present, but they are so little characteristic that no diagnosis is possible in such cases either. In a small number of cases, however, a diagnosis may be made with a certain degree of probability. The principal symptom is *a picture of increased intracranial pressure, taking place under the clinical picture of apoplexy,* which is characterized by vomiting, coma, slowed, irregular pulse, *contracted pupils,* which react with difficulty or not at all, sometimes choked disk, conjugate deviation and, eventually, excessive elevation of temperature, associated with, sometimes, unilateral, at other times, in more extensive meningeal hæmorrhages, with bilateral paralyses. There are present, at the same time, because the hæmorrhage usually acts upon the *cortex,* monocontractures and monopareses, which, later, may pass into hemipareses and hemiplegias, Jacksonian *epilepsy* and also, according to the localization of the hæmorrhage, *aphasia, sensory disturbances,* etc., whereas paralytic symptoms restricted to individual cerebral nerves at the base (corresponding to the most frequent seat of hæmatoma in the region of the vertex) are absent in by far the majority of cases.

Especially the last-named symptoms, pointing to an *affection of the cortex,* are of importance in the *diagnosis of hæmatoma,* but particularly the fact that the severe cerebral symptoms *increase gradually,* but may then become more or less *retrogressive,* to recur in a similar manner in *paroxysms* in renewed hæmorrhages.

If the above-described total of general and special focal symptoms is found in a clinical picture and if this symptom-complex has occurred suddenly, we may primarily think of a meningeal hæmorrhage, especially if a *traumatism* acted upon the skull, or if we have some reason for assuming the presence of an aneurysm or an affection of the vessel walls. If the symptoms of the cerebral affection occur in *attacks,* if improvements and aggravations alternate, we may, with a certain degree of probability, diagnosticate *hæmatoma of the dura mater,* but only when the ætiological factors formerly mentioned, dementia paralytica and alcoholism above all (syphilis is said also to cause formation of hæmatoma), preceded the severe attacks. However, a positive diagnosis cannot be thought of under such conditions.

HYDROCEPHALUS (HYDROPS VENTRICULORUM CEREBRI —HYDROCEPHALUS INTERNUS ET EXTERNUS S. INTERMENINGEALIS)

The accumulation of fluid in the ventricles, as repeatedly stated on former occasions, is a consecutive symptom of various cerebral diseases, especially of tumour and of meningitis. In that case it must be considered diagnostically as complication, in so far as certain cerebral general symptoms in the clinical picture can be ascribed to the same. Chronic internal and external hydrocephalus (intermeningealis, i. e., exudation of fluid principally in the subarachnoid reticular tissue) is found, furthermore, in various conditions leading to atrophy of the brain, thus in old hæmorrhages

and softenings, in senility, etc., and also in conditions of disturbed nutrition and hæmatogenesis: Carcinoma, Bright's disease, leucæmia, alcoholism; but, above all, in rhachitis the development of hydrocephalus occasionally takes place.

Chronic Hydrocephalus.—This symptomatic form of hydrocephalus is opposed by (non-congenital) *idiopathic chronic hydrocephalus*. The latter is rare in adults and older children, occurs as the expression of chronic meningitis and is accompanied with vague cerebral general symptoms: Headache, vertigo, vomiting, psychical disturbances, muscular weakness, tremor, spastic pareses, choked disk, etc. It *cannot be diagnosticated*, it can scarcely be surmised.

Congenital Hydrocephalus.—*Chronic hydrocephalus* occurs much more frequently *congenitally* or it develops in earliest infancy. This form of hydrocephalus (usually only internal) is subject to diagnosis, in fact, can be recognised generally without any difficulty whatever, as the head of the child enlarges enormously with the increasing fluid, owing to the open sutures and fontanelles. The bones of the skull become thin to transparency at the same time, the frontal bones are arched out anteriorly, the parietal and temporal bones laterally. Thus it occurs that the cranial bones project over the external auditory canal and especially over the orbital vault, and that the orbits appear narrowed and shortened. On the surface of the skull we usually see the distended veins shine through as bluish cords and we find the fontanelles and sutures to be open; sometimes we may observe even fluctuations over the fontanelles. The face, in comparison to the enormously distended skull, appears remarkably small, broadening towards the upper end.

The effects of the enlargement of the skull are: Waving of the heavy head, transparency of the cranial bones when turned towards a light, increase of the circumference of the skull to twice its dimensions (normal circumference of the head in the first year 35–45 cm.,[1] growing until puberty to about 50 cm., whereas in hydrocephalus it is 60, even 100 cm. and more). Various symptoms of the cerebral affection, besides these external pathognomonic characteristics, manifest themselves, corresponding to the flattening and thinning of the hemispheres to a few centimetres, primarily, signs of *psychical* weakness which may increase to complete dementia; this is true, at least, in the majority of cases. It is exceptional only that the children learn to speak and show a fair degree of mental development; usually they are, as stated, demented, are not able to speak, and discharge fæces and urine involuntarily. At the same time, pareses of the extremities, besides spastic manifestations and increased tendon reflexes, may be present, and general convulsions and spasm of the glottis may take place. Sensibility, in contrast to motility, is less disturbed; but the power of vision is considerably impaired, the *optic nerves* are frequently *atrophied; choked disk* also develops if the circumference of the brain does not expand in proportion to the accumulation of fluid; besides, ptosis, strabismus, nystagmus, etc., may be present. The hydrocephalic fluid, which is drawn off by lumbar puncture *intra vitam*, is water-white and contains no albumin or only traces of it.

Differential Diagnosis.—It is scarcely possible to confuse infantile hydrocephalus with any other affection. In cases of slight development, however, the question must always be decided whether abnormal shape of the skull might not be due to *rhachitis of the bones of the head*. In such cases, also, abnormally wide fontanelles and sutures occur, due to the fact that the cranial bones are retarded in growth in comparison to the contents of the skull. If we consider, further, that the face also remains small in rhachitis, owing to the deficient growth of the facial bones, and that the frontal and parietal eminences, the same as in hydrocephalus, are prominent, a certain similarity of these two cranial deformities cannot be denied. The differentiation is easy, however, if we consider that the nervous symptoms are entirely absent in the rhachitic skull, that the cranial measurements remain approximately normal, and that the bones of other parts of the body also show rhachitic changes. However, we must always bear in mind that hydrocephalus very often

[1] For exact tables of normal measurements of circumference and diameter of the skull, see Huguenin, Nervenkrankheiten, Ziemssen's Sammelwerk, second edition, vol. xi, p. 937.

occurs in association with rhachitis, and that a symptomatic hydrocephalus may, eventually, take place also in the course of rhachitis, due to intercurrent diseases, which are accompanied with general stasis, or to supervening acute meningitis, tuberculous tumours of the brain, etc.

Angeioneurotic Hydrocephalus.—Finally, we wish to state that Quincke has recently called attention to the possibility of the occurrence of a rapidly appearing and rapidly vanishing hydrocephalus, which he designated as "*angeioneurotic*" hydrocephalus. This form is said to be brought about, under the influence of those nerves which govern the secretion of lymph, as an acute increase of the normal secretion of the cerebro-spinal fluid. This would probably plausibly account for certain cases of paroxysmal, severe headache, for certain obscure pathological pictures which consist in *transitory* cephalic symptoms (vertigo, violent vomiting and psychical depression). I consider the right of existence of angeioneurotic hydrocephalus to be very probable and the following up of Quincke's suggestion to be very promising.

DIAGNOSIS OF FUNCTIONAL DISEASES OF THE BRAIN (SO-CALLED NEUROSES)

Preliminary Remarks.—Common to the so-called neuroses is that regular anatomical changes of the central nervous system, which are in a causal connection with the nature of the disease, are absent as yet, although the various affections of this class present quite typical functional disturbances of the central nervous system. Accordingly, neuroses are also designated as "*functional diseases of the brain.*" Although their diagnosis, owing to the absence of an anatomical basis, lacks a firm support, it can, nevertheless, mostly be made with great certainty, as the various neuroses represent strictly defined syndromes, the determination of which usually allows at once the recognition of the disease and the exclusion of the presence of other symptoms of the central nervous system in the given case.

HYSTERIA

The diagnosis of *hysteria,* as I wish to state in advance, cannot be learned from the descriptions of the pathological picture. It presupposes, rather, that the physician has ample opportunities to observe hysteric patients, to study the changing, yet, upon the whole, typical, clinical picture, to obtain a keen penetration for the diagnosis of the affection which cannot be controlled by autopsy results. Therefore, I shall simply outline the clinical picture, so far as it should be considered in the diagnosis, and discuss the differentio-diagnostical view-points more exhaustively.

Character of Hysteria.—*It is not possible as yet* to give an *exhaustive explanation of the conception of hysteria,* which would be of importance in defining the diagnosis. The former assumption that, in hysteria, it is a question of a functional disturbance of the *entire* nervous system, of the central, peripheral and sympathetic, especially of a "perverse reaction" of the same, has been more and more abandoned in recent times. Today, hysteria is mostly considered a *psychosis* in the broader sense. This conception of the nature of hysteria offers the advantage, at any rate, that origin and manifestations of the disease can be explained from a uniform view-point. To assume anatomical changes in the brain in hysteria is contradicted not only by the negative results of autopsies and anatomical investigations, but also by clinical observations, by the alternating clinical manifestations—the rapid appearance and disappearance of even the severest hysteric affections. Hysteria is, rather, the exquisite type of a

functional nervous disease in which either the sensory centres or the association areas in the brain are in a condition of irritable debility. This view, in my opinion, best accounts for the fact that sometimes anæsthesia, at other times hyperæsthesia, predominates, according to the prevailing intensity of the weakness, respectively irritability, of the ganglion cells in the sensory centres, and, furthermore, that hysteric patients are unable correctly to associate the impressions of sense, even if they are still perceived, i. e., to incorporate them into consciousness as images of memory, and to elaborate them mentally in the regular manner. One portion of the paralyses and convulsions in hysteria would be due to the irritable weakness of areas of the cerebral cortex which serve to produce movements, whereas another portion (in pronounced paralyses of will-power) would be due rather to the functional weakness of association tracts, i. e., to the deficient or absent action of the influences of will-power upon the motor centres of the cortex of the brain. Besides, to understand certain manifestations in the clinical picture of hysteria, it should be assumed, probably, that the inhibitory influences which emerge from the cortex of the brain and which regulate the movements and reflex processes, have also become deficient in this disease. It is evident, accordingly, that, here, only an alteration of such cerebral elements has been assumed as are connected with the corporal functions of movement, perception, secretion, etc. The higher mental functions, the association of previously obtained images of memory among each other and their elaboration in the act of thinking, need not be disturbed in the sense of an alienation, in such cases. Although the process of thinking, owing to the irritable weakness, which we must imagine as affecting the entire cortex of the brain, takes place in an enfeebled-slowed or exalted manner, yet it still remains within normal limits, so far as it does not concern new impressions of sense which are received from external sources, respectively from the *ego*. But, on the other hand, abulia and amnesia will not fail to appear in the higher grades of hysteria, as we know from experience to be the case. There still remains to discuss briefly, in accordance with the above theory, the occurrence of the *rapid onset* of hysteria and the *production of the attacks*. More marked psychical emotions are no longer overcome by a brain which has deteriorated by bodily weakening factors, which has attained a labile equilibrium owing to long-lasting mental alterations, and the power of resistance of which has thus become weakened; complete recovery fails to appear and the hysteric status becomes manifest; the better the soil is prepared, the less of a psychical emotion is required to cause such an effect, and *vice versa*. If the psychical shock is produced by a somatic trauma, we may expect that the hysteric manifestations will present themselves especially on that side which was affected by the trauma. If an "attack" is to make its appearance in hysteric individuals, it is necessary that the neurons, respectively the ganglion cells of the cortex of the brain, become charged to a certain degree, followed by explosive irritation. According to my opinion, it is quite feasible to imagine that, in a nervous system of the above-described condition, the irritations which come from external sources and extend centripetally, are not elaborated in the regular manner, but accumulate to

cause, not until a more extensive accumulation of individual irritations has taken place, a total discharge in the motor areas of the cortex and in the neurons, which becomes explosively manifest, according to the amount of accumulated irritations, either in a limited, partial irritation of the nerves, or in general, excessively intensified convulsions. The occurrence and the intensity of the convulsions are favoured by the fact that the normal inhibitions have also become insufficient in the brain of hysteric patients, as stated previously.

Ætiological Points of Support.—It will be seen from the above explanations that *ætiological factors* play a very important part in the diagnosis of hysteria. *Local* affections, especially those of the sexual organs, are, according to my experience and that of others, not really the basis of hysteria, but, at most, a predisposing factor. *Psychical* perniciousness, both violent emotions of short duration (fright, anxiety, etc.) and long-lasting depressing influences upon the mind, are, rather, incentive to the development of hysteria. Besides, education and heredity must often be considered, also, as very essential factors; but, furthermore, the origin of the disease is greatly enhanced by nervous emotions and metabolic changes, which are associated with the appearance of puberty, and also by diseases of metabolism and anomalies of constitution in general (anæmia, cachexia, adiposity, etc.). An appropriate treatment of such nutritive disturbances will, therefore, materially contribute to cause the disappearance of the hysterical manifestations. Hysteria is a disease which occurs almost exclusively in the *female*. Only rarely do we see exquisite clinical pictures and higher grades of hysteria in the *male*, according to my experience only when it is a question of effeminate characters, or if puberty was exceptionally turbulent.

The symptoms of hysteria, the most frequent of which and those which are most characteristic and usually permanently present are unneccessarily designated especially as "*hysteric stigmata*" (Charcot), belong to the motor, sensory, vaso-motor and psychical spheres of the nervous system.

Hysteric Paralyses.—The disturbances of motility are either complete or incomplete *paralyses*, or *spasms* of the most various kind and intensity. The *paralyses* can be recognized easily as being of an hysteric character, in contrast to paralyses depending upon anatomical changes in the brain and spinal marrow, in those cases in which their character manifests itself as "paralysis of will-power," "paralysis of function," in that only certain movements, for instance, walking and standing,[1] are impossible, whereas other functions are performed without difficulty by the same muscles. It

[1] These forms of hysteric paralysis were recently designated with the names "*astasia*" and "*abasia*." The patients thus afflicted are absolutely unable to walk or to stand; they collapse completely upon any attempt to stand erect. When lying down, neither a decrease of the motor power nor a disturbance of muscular perception or co-ordination can be determined. Astasia and abasia, the same as other hysteric paralyses of will-power, can occur and disappear suddenly. Sometimes they seem to be the only hysteric symptoms, i. e., other symptoms, such as disturbances of sensibility, etc., may be absent. At other times only one leg is paralyzed, which is then dragged by the patient when walking.

is more difficult to determine them as hysteric if it is a question, not of partial paralyses affecting certain combined movements, but of total paralyses in the form of *hemiplegias* or *paraplegias,* thus causing the differentio-diagnostical consideration of a cerebral or spinal affection.

Differential Diagnosis of Hysteric Paralyses.—Determining in this case is, above all, the condition of the other nervous symptoms which are synchronous with the paralysis. In *paraplegias* depending upon affections of the spinal marrow the bladder is almost always involved in the paralysis, with development of cystitis and bedsore—in hysteric paraplegia these severe accompanying symptoms of paraplegia are absent; in rare cases it may occur, it is true, that retention of urine takes place in hysteric patients (owing to spasm of the sphincter), but, on the other hand, permanent strangury or even paralytic cystitis do not take place, at least according to my experience. It is of less importance in the differential diagnosis that consecutive muscular atrophy and reaction of degeneration do not occur in hysteric paralysis, because both conditions are naturally absent also in many cases of spinal paraplegia. Hysteric hemiplegias, which are not so rare, are also distinguished by certain peculiarities from the non-hysteric hemiplegias caused by focal affections of the brain. Whereas, in the latter, paralyses of the facial and hypoglossal nerves so very commonly accompany the unilateral paralysis of the extremities, paralyses of cerebral nerves are surely absent in the majority of cases of hysteric hemiplegias. A spasmodic contraction of the musculatures of the tongue and mouth on the opposite (or even on the paralyzed) side was observed in some cases, besides the hemiplegia, causing a marked deviation of the affected corner of the mouth and of the protruded tongue (spasmus glosso-labialis hystericus). Furthermore, *sensibility* is materially disturbed in non-hysteric hemiplegias except in unusual cases, i. e., only in case of an unusual seat of the focal affection of the brain; whereas it is usually considerably impaired in hysteric paralyses, both the sensibility of the skin and that of the muscles, especially the perception of the electric contraction of the muscles. No differentio-diagnostical conclusion can be drawn from the condition of the tendon reflexes. They are very considerably increased in hysteric paralyses and in non-hysteric paralyses in some cases; then the paralyses present the character of spastic paralysis.

Of much greater importance than all the criteria mentioned so far is *the manner of the course of hysteric paralyses.* They are usually quite variable, i. e., the intensity and localization may change extremely rapidly, which is never the case in paralyses brought about by affections of the spinal cord and of the brain. But even if hysteric hemiplegias are obstinate, persisting in intensity for years, they behave differently from long-lasting non-hysteric hemiplegias. In the latter the motility of the paralyzed leg improves considerably more than that of the arm, whereas in hysteric paralyses the arm is more liable to regain its motility earlier than the leg. If contractures become associated with the paralyses, which may occur in both varieties, a difference manifests itself in so far as hysteric contractures do not conform as typically to the usual picture of contracture position in non-hysteric hemiplegias—extension contracture of the leg, adduction contracture of the upper arm, flexion contractures of the fingers—as the latter; that, for instance, the hand, accordingly, may assume a position of dorsal flexion. Finally, a determination of the very considerable resistance of hysterically paralyzed muscles to passive movements often decides in favour of the diagnosis of hysteria. If, furthermore, we are able to determine that the patients are able to move the paralyzed extremities when in the dorsal position, but cannot do so when standing, etc., in short, if we find indubitable "functional paralyses," any further differential diagnosis becomes superfluous; it is necessary, therefore, above all to investigate into the existence of such paralyses of will-power.

Besides, the differential diagnosis is materially facilitated, in by far the majority of cases, by the demonstration of other exquisitely hysteric manifestations of paralysis which are present besides paralysis of the extremities. Of these are to be considered, among others, those very frequent hysteric *paralyses of the vocal cords* (see

p. 89), paralysis of the œsophagus and *hysteric meteorism* depending upon intestinal paresis, a condition which may persist for some time and which may be followed by so enormous a distention of the abdomen (phantasma hystericum) that abdominal tumour, etc., must be considered in the diagnosis.

Hysterical Convulsions.—*Hysterical convulsions* are of much greater importance in the diagnosis of hysteria than paralyses. They are either clonic, or tonic, partial or general convulsions. Of partial convulsions are to be mentioned: Spasm of the muscles of mastication, of the throat and neck, rarely also of other muscles, hysterical stuttering, globus hystericus, spasms of deglutition (hydrophobia hysterica), spasm of the diaphragm (singultus) and spasm of the musculature of the bladder, increased peristalsis (manifesting itself in borborygmus and diarrhœa), and, furthermore, the roaring eructations, convulsions of crying, laughing, yawning and sneezing, hysterical hyperemesis, hysterical cough (distinguished by its superficiality and the short, continuously repeated concussions), nervous dyspnœa, hysterical trembling and shaking, etc. If the spasmodic innervation simultaneously affects a large number of co-ordinately acting muscles, the picture of so-called **chorea major** results, in which the patients execute larger, impulsive, co-ordinate movements, dance, climb, gesticulate, recite, continually emit roaring sounds, incessively run ahead until they fall, often completely exhausted, etc.; I have observed chorea major to occur principally in boys during the time of puberty.

Catalepsy.—If the condition of general convulsions takes place in such a manner that the muscles remain in a *moderate tonic contraction,* can easily be passively placed in any desired position (flexibilitas cerea), to be held in this position, even if it is most inconvenient to the patient, permanently, often for hours at a time, we speak of *catalepsy.*

Cataleptically contracted muscles react to the electric current with the usual contraction, but after the cessation of the electric stimulation they reassume the original (cataleptic) position. Such cataleptic conditions occur spontaneously in hysteria after psychical emotions or after hypnotization. Catalepsy does not take place exclusively in hysteria, but is also found in certain psychoses and is said to occur (personally I have never seen anything of the kind) in anatomically demonstrable affections of the brain; but most frequently it is an attribute of hysteria and can be utilized, jointly with other hysterical manifestations, in the diagnosis of this condition.

Hysterical Convulsive Paroxysms.—The hysterical predisposition to spastic conditions manifests itself much oftener than in chorea magna and catalepsy in the form of *general convulsive paroxysms,* which, although they may be entirely absent in some cases of hysteria, yet, in others, represent the most important mode of manifestation of the affection and intercalate between the permanent symptoms as "*hysterical paroxysms.*" They manifest sometimes a tonic, at other times an exquisitely clonic, character. In the former case the musculature of the back usually appears tense (tetanic), showing the picture of opisthotonus, of the "*arc de circle,*" so that only the head and the tips of the feet touch the bed. The attack may be ushered in by some kind of an aura: By a ball ascending from the abdomen, by palpitation of the heart, anxiety, followed by the

epileptoid period of the attack which at the onset may be entirely similar to the commencement of an epileptic attack. The hysterical convulsive attacks are very commonly accompanied with spasmodic, sometimes enormous, acceleration of respiration which often takes place in a gasping or sobbing manner. At the same time, consciousness is not entirely lost, but it is under the influence of hallucinations, the presence of which usually manifests itself in a frightened, threatening or enraptured facial expression and wild talking or shouting. The patients are extremely susceptible to "suggestions" in such conditions, they are highly accessible to the artificial causation of psychical compulsory conditions, and corporeal ones depending thereon; they allow themselves, eventually, to be put into catalepsy or into a condition of somnambulism, etc., by hypnosis. Such spasms are usually induced by pressure upon certain regions of the body—the ovarian region forms (although by no means exclusively) an especially effective, so-called "hysterogenous zone," the irritation of which more or less regularly recalls the hysteric attack, but sometimes causes an attack, which is in progress, to disappear. *Clonic hysterical convulsions,* if they are more or less well developed, are similar to the clinical picture of other neuroses, such as chorea, epilepsy, etc., and have, therefore, been designated by separate names, as chorea hysterica, myoclonia, hystero-epilepsy.

Hystero-Epilepsy.—As to the last-named hysteric attacks, "*hystero-epileptic*" paroxysms, they may, indeed, be so similar in all details to genuine epileptic attacks that the differentiation of both conditions may, sometimes, be quite difficult, in fact, as I must admit from personal experience, impossible; the latter case is very rare, however. The hysteric attack usually manifests itself as such, in contrast to an epileptic one, in that consciousness is scarcely ever lost *completely,* but only, occasonally, slightly dimmed; that, furthermore, convulsive movements of a more complicated nature and which are performed in a more co-ordinate manner (such as beating about with the arms, reaching for certain objects, rotary movements), become intermingled with convulsive spasms, and in that the facial expression does not show the dull tranquility of mental absence, but the prevalence of certain affective faculties (such as anger, fright or ecstasy). Genuine reflex pupillary immovability probably never occurs in an hysteric attack, as is the case in epilepsy, but, on the other hand, mydriasis caused by spasm or paralysis. Hystero-epileptics do not fall quite as carelessly, consciousness not being lost entirely, and do not hurt themselves as readily, as epileptics. The duration of a single attack is always short in epilepsy, lasts for minutes, whereas an hysteric attack lasts a quarter to half an hour and longer, and may recur rapidly, so the patients are sometimes for days in the continuous grasp of the convulsive attacks. It is important, differentio-diagnostically, that exquisitely hysteric symptoms persist during the intervals, that the epileptiform attacks alternate with others which can be easily explained as hysteric attacks. But, as stated, in some very rare cases the observation of the above-mentioned differentio-diagnostic factors is not sufficient to decide with certainty whether hysteria or epilepsy be present. This is not very remarkable, according to our view regarding the causation of hysteric convulsions (see above). We wish to state, particularly, that genuine, idiopathic epilepsy may be combined with hysteria.

Hysteric Disturbances of Sensibility.—*Sensory disturbances* are of no less importance in the diagnosis of hysteria than the motor symptoms. They refer to the whole of the organs of sense: The organs of vision and hearing, of smell, taste and touch (in the general meaning of the word), and they manifest themselves either in hyperæsthesias or in anæsthesias.

There is such an enormous multitude of the various pathological symptoms that an enumeration of the same, which would be only slightly exhaustive, is impossible —besides, would be of little value to the diagnostician, as deviations from the usual clinical picture are quite common, and the details are very changeable; general suggestions will, therefore, probably be sufficient in this instance. *Hysteric patients are, frequently, not at all conscious of their morbid disturbances, especially of the sensory ones, and the presence of these disturbances can sometimes be discovered only by an investigation which is specially instituted for the purpose.* It is necessary, therefore, in every doubtful case to look for the presence of these "stigmata."

Hyperæsthesia prevails in many cases. The slightest touch of the skin is painfully felt, usually only at certain, closely circumscribed, areas, more frequently on the trunk than on the extremities, and most constantly in the *lower abdominal region* ("ovaries") and in some of the *vertebræ*. In other cases the patients complain of severe pains, to which correspond no demonstrable changes of the affected parts—thus of tormenting headache, which occurs sometimes diffusely, at other times in the form of migraine, at other times, again (although not as often as is usually assumed), as a pain which is concentrated upon a small area of the vertex (*"clavus hystericus"*). In some cases there exist actual neuralgias or arthralgias (see Neuralgia). The list of pains in internal organs is especially large: Bladder pains, gastralgias, colics, uterine pains, angina pectoris and, connected with the altered reaction of the sensory nerves of the heart, retardation and irregularity of the pulse, etc.[1] This abnormal hyperæsthesia manifests itself in the organs of sense as photopsy, tinnitus aurium, acuition of taste and smell, etc.

The various *paræsthesias* occur quite usually in hysteria. The patients have sensations of itching, formication in the skin, strange sensations of taste and smell, etc., in all regions *of the body*.

Still more pronounced are the *anæsthesias* peculiar to hysteria, which affect either the skin or the organs of sense. Slight, as well as painful, touching of the skin is no longer noticed—complete *analgesia* is present which is either strictly limited or affects the entire surface of the body. In the latter case it may occur that a very small area is not included in the analgesia, and this fact points quite particularly to the hysteric character of the latter. Thus I remember that in one case the entire surface of the body was analgesic to such an extent that an alcohol flame brought in contact with the skin burnt the latter without causing any perception of pain; and, at the same time, a smaller area of the forehead showed normal sensibility. In another case, one general perception, that of titillation, was retained, with total anæsthesia of the entire surface of the body—a fly, which crawled over the anæsthetic cheek of the patient, was annoying her, the betrayer of hysteria! Besides anæsthesia of tactile sensations (in the narrower sense), the perception of other sensory qualities, especially the perception of changes of temperature, may be fully preserved. In

[1] Sometimes hysteric patients feel their pains, which are obviously produced by conditions of psychical irritation, so intensely when moving about that they decline any kind of movement. These conditions, in which, as has become more and more known, "hallucinations of pain" prevail, are designated as "*akinesia algera*."

other cases this, also, is altered, as well as the muscular sense, so that the patients, upon closing their eyes, have no idea as to the position of their limbs, and an actual hysteric ataxia may manifest itself. *Sensory anæsthesias* are also frequent traits in the picture of hysteria: Diminution of hearing, anæsthesia of impressions of taste and smell, functional disturbances of the organ of vision.

Condition of the Eyes.—Loss of sight is found, in the first place, most frequently unilateral, rarely bilateral; it persists for a short while only, and quite suddenly the former faculty of sight returns; this loss of vision is usually designated as *retinal anæsthesia.* Furthermore, *concentric limitation of the field of vision* and synchronous hemianopsia were observed, also *disturbances of the colour sense* (such as diminution of the same), as well as partial and total colour blindness. Sometimes the levator palpebræ muscle is affected by transitory paralysis (ptosis). Manifestations of a so-called *retinal hyperæsthesia* may also take place, characterized by marked susceptibility to light, lack of perseverance in nearby occupations, caused by rapid fatigue of the internal recti oculi muscles and of the accommodation muscle, and piercing pains in the fundus of the orbit. Tonic spasms of the orbicular palpebral muscle (blepharospasm) may also occur. Sometimes a *spasm* affects only the rectus internus muscle, or conjugate deviation of the eyes takes place in hysteric attacks.

As previously stated, the regions of disturbances of sensibility are sometimes circumscribed, at other times diffuse. *But, in many cases, anæsthesia is limited strictly to one half of the body,* and this condition is especially pathognomonic of hysteria. *Hemianæsthesia,* in such cases, may not only affect the skin, but also the mucous membranes, muscles, joints and organs of sense of one side. As anæsthesia extending over the entire sensory region of one half of the body does not occur equally as complete in any other affection accompanied with hemianæsthesia, it may actually be considered as an important diagnostic characteristic of hysteria. However, it is often necessary for the physician to discover even this total hemianæsthesia, as the patients themselves have no idea of the presence of anæsthesia; on the contrary, they complain of pains in the affected half of the body. A further expedient to recognise hysteria is the so-called *"transference."*

If a plate of metal is placed upon an anæsthetic area of patients with hemianæsthesia, sensibility soon returns in this area, while anæsthesia is produced over an exactly corresponding area of the other half of the body; the same result is obtained if the plate of metal is placed, not first upon the anæsthetic, but directly upon the normally sensitive area of the skin. The same conditions prevail in an analogous treatment of anæsthetic mucous membranes, organs of sense and even of hysteric contractures and paralyses, etc. I was not able, in spite of a great deal of scepticism, to convince myself that these remarkable symptoms are simply the effects of suggestion, besides, indications of transference are found also under normal physiological conditions.

Vaso-Motor and Secretory Disturbances.—Less frequent than the symptoms of disturbances of sensibility are hysteric alterations of the *vaso-motor* and *secretory* nerves. *Vascular innervation,* above all, may be *disturbed in hysteria*; this fact manifests itself in abnormal pallor or redness of the skin, more rarely in the occurrence of "hysteric," sometimes "blue"

sion become manifest; the patients are no longer able to form ion; finally, complete loss of energy and abulia are present, or, on ther hand, entirely one-sided concentration of the will-power in a cer- direction prevails, so that they try to attain the ends they have in iew, which are usually of a very paltry nature, with a great deal of obstinacy. In the meanwhile hallucinations and forced ideas become predominant, precipitating the patients to commit perverse, in fact base, deeds. It is not uncommon that hysteria passes into pronounced derangement of the mind: Melancholia, mania, a condition of "folie raisonnante" or insanity.

Differential Diagnosis.—When discussing the various symptoms to be considered in the diagnosis of hysteria we have explicitly entered upon the difference between hysteric paralyses and those which are due to affections of the spinal marrow and of the brain. The differential diagnosis in these cases may sometimes, at least for a while, be quite difficult, also in cases in which some hysteric symptoms manifest themselves apparently quite isolately, and may be accounted for in some other manner. Often it is particularly difficult, also, to recognise hæmorrhages from internal organs to be hysterical ones, and thus harmless in comparison to hæmorrhages brought about by anatomical changes in the various organs; in this respect we must refer to the diagnostic explanations in the chapters on Respiratory, Gastric, etc., Diseases.

Finally, one word regarding the differentiation between hysteric and *hypochondriacal* conditions! There can be no doubt that, in the clinical picture of hysteria, the depression and concentration of thoughts upon the personal condition of body and mind prevails to such an extent that the patients fully create the impression of hypochondriacs. It will soon be seen, however, upon closer investigation, that the psychical disturbance in hysteria is less one-sided, and less forcibly restricted to the morbidly felt condition of the own body and mind than in real hypochondriacs, who represent an independent psychical affection. Furthermore, in hysteria the symptoms of the alteration of the psychical condition are always intermingled with corporeal disturbances, convulsions, pareses, etc. Besides, hysteria is much more accessible to suggestion than hypochondriasis. The latter forms the fundamental feature in the clinical picture of neurasthenia, the discussion of which we shall enter upon now.

NEURASTHENIA, NERVOUSNESS

Neurasthenia.—Affections diagnosticated under these so frequently used terms are, in my opinion, only *varieties of hysteria* which represent either slightly developed grades of this affection, or pathological conditions modified by the sex of the patient. It is true, the diagnosis of neurasthenia has recently become a fashionable diagnosis, and certain differentio-diagnostic factors have been made, with a good deal of perseverance, to become prominent in order diagnostically to separate hysteria and neurasthenia. But I have not yet been able to convince myself that these two affections represent two diseases that are different in *character*, although I fully

œdema and spontaneous gangrene, relatively often in hæmorrhages which may take place from the gums, respiratory tract, gastric mucous membrane, and which may often give rise to considerable differentio-diagnostic difficulties. Furthermore, *anomalies of secretion* are frequent symptoms of hysteria, such as abnormal diminution or increase of the salivary, lachrymal and sudoriparous secretions, galactorrhœa, colica membranacea and, above all, disturbances of the urinary secretion: Ischuria, polyuria (urina spastica), anuria with vicarious vomiting of profuse, watery (urate-containing) fluid. However, I must, from my experience, advise the greatest caution in the assumption of such cases of hysteric anuria, which do not terminate fatally as might be expected (with the clinical picture of uræmia).

It does not appear necessary to me, in the interest of the diagnosis of hysteria, to enumerate further symptoms observed in this affection, especially as, owing to their protean character, even the most exhaustive description of the pathological picture is not complete. Only one point should be specially referred to, viz., *the altered psychical conduct of the patients.*

Psychical Disturbances in Hysteria.—*We are able, upon closer investigation, to demonstrate an alteration of the psychical conditions in all cases of hysteria.* It concerns, as previously stated, principally those functions of the brain which are in connection with corporeal conditions, but indications of disturbances of higher psychical functions are also noticeable, and they become more pronounced so soon as the hysteric condition attains a more marked degree. In milder cases disposition and sentiments are pathologically altered, the inhibition of affective faculties is insufficient. These patients are very apt to become ill-tempered, they are extremely sensitive, irritated by trifling matters; they are highly whimsical, subject to sympathies and antipathies, with a participation of sexual sensations. Conditions of depression and sensations of anxiety alternate with periods of uncalled-for exaltation; the frame of the mind is influenced also, and, above all, by the conduct of the surroundings, in that the latter either pay too much attention to, or wonder too much at the manifestations occurring in, the patient, or (the more frequent case) do not sufficiently consider their importance, according to the opinion of the patients. To attain their ends, hysteric patients, either consciously or instinctively, commit deeds which are contradictory to their former moral principles; often they lie and deceive, simply to create an impression with their morbid manifestations, and without consideration they give way to their unrestrained impulses. Gradually the range of their ideas becomes narrower, and, at last, it concerns nothing but their own morbid *ego*; subject to such fancies, the patients exaggerate the presentation of their corporeal ailments. Conversely, vivid sympathies with certain therapeutic procedures may give rise to inconceivable recoveries (of severe paralyses of long standing, etc.). Suggestion may be employed most advantageously in this affection, and particularly the effect of suggestion, which is surprising in most cases, may be utilized to arrive at the diagnosis of hysteria. Gradually the ***higher*** psychical functions also become more affected: Absence of memory

and indecision become manifest; the patients are no longer able to form an opinion; finally, complete loss of energy and abulia are present, or, on the other hand, entirely one-sided concentration of the will-power in a certain direction prevails, so that they try to attain the ends they have in view, which are usually of a very paltry nature, with a great deal of obstinacy. In the meanwhile hallucinations and forced ideas become predominant, precipitating the patients to commit perverse, in fact base, deeds. It is not uncommon that hysteria passes into pronounced derangement of the mind: Melancholia, mania, a condition of " folie raisonnante " or insanity.

Differential Diagnosis.—When discussing the various symptoms to be considered in the diagnosis of hysteria we have explicitly entered upon the difference between hysteric paralyses and those which are due to affections of the spinal marrow and of the brain. The differential diagnosis in these cases may sometimes, at least for a while, be quite difficult, also in cases in which some hysteric symptoms manifest themselves apparently quite isolately, and may be accounted for in some other manner. Often it is particularly difficult, also, to recognise hæmorrhages from internal organs to be hysterical ones, and thus harmless in comparison to hæmorrhages brought about by anatomical changes in the various organs; in this respect we must refer to the diagnostic explanations in the chapters on Respiratory, Gastric, etc., Diseases.

Finally, one word regarding the differentiation between hysteric and *hypochondriacal* conditions! There can be no doubt that, in the clinical picture of hysteria, the depression and concentration of thoughts upon the personal condition of body and mind prevails to such an extent that the patients fully create the impression of hypochondriacs. It will soon be seen, however, upon closer investigation, that the psychical disturbance in hysteria is less one-sided, and less forcibly restricted to the morbidly felt condition of the own body and mind than in real hypochondriacs, who represent an independent psychical affection. Furthermore, in hysteria the symptoms of the alteration of the psychical condition are always intermingled with corporeal disturbances, convulsions, pareses, etc. Besides, hysteria is much more accessible to suggestion than hypochondriasis. The latter forms the fundamental feature in the clinical picture of neurasthenia, the discussion of which we shall enter upon now.

NEURASTHENIA, NERVOUSNESS

Neurasthenia.—Affections diagnosticated under these so frequently used terms are, in my opinion, only *varieties of hysteria* which represent either slightly developed grades of this affection, or pathological conditions modified by the sex of the patient. It is true, the diagnosis of neurasthenia has recently become a fashionable diagnosis, and certain differentio-diagnostic factors have been made, with a good deal of perseverance, to become prominent in order diagnostically to separate hysteria and neurasthenia. But I have not yet been able to convince myself that these two affections represent two diseases that are different in *character,* although I fully

acknowledge that other views may be held, even if I were not to consider it right to use the designations neurasthenia and hysteria promiscuously.

Neurasthenia occurs much more frequently in the male than in the female; in the former it rarely attains those higher grades which are designated as hysteria, and which occur rapidly in women after the "condition of functional weakness of the nervous system" that designates the character of neurasthenia, once has developed. It is rare, therefore, that we speak of hysteric men and still rarer of neurasthenic women. The causes of neurasthenia are the same as those of hysteria: Development of puberty, masturbation, high-graded, frequently repeated excitements, constitutional anomalies, etc. But certain factors are added which are much more effective in the male, especially the abuse of alcohol, sexual excesses, mental overexertion and the want of recreation of the *exhausted* activity of the nerves. The same as in hysteria, the irritable debility of the central nervous system manifests itself in disturbances of the psychical, sensory, motor and vaso-motor spheres. Only, they all remain upon a lower grade of development, are less apt to change into higher degrees of general nervous disturbance, but, on the other hand, the different symptoms are generally distinguished by great obstinacy and change less often than in pronounced hysteria. An interesting variety of neurasthenia has recently been described repeatedly, the "*circular*" form of neurasthenia, an analogy of *folie circulaire*, in which conditions of depression and exaltation, "bad and good" days alternate with great regularity.

Psychically we again meet with irritability, sensibility, caprice, depression, which we have encountered in hysteria. To these are added sensations of distress of various kind (agoraphobia), ideas of compulsion, although of a more harmless character than in pronounced mental diseases, *folie du doute*, etc., and, above all, a hypochondriac frame of mind. Burdened with this alteration of his psychical life, the neurasthenic gradually loses his former mental elasticity and ability to work; he is more subject to fatigue in thinking, even in reading and writing. His will-power gradually relaxes and also the ability to abnegate his own self in favour of others and to allow the preponderance of more ideal sentiments, until, finally, actual abulia and dulling of sentiment take place.

To be mentioned among the *disturbances of sensibility* are, above all: Headache, giddiness, pains and hyperæsthesias in the back and in the extremities, especially, also, the painfulness of some vertebræ, and, furthermore, paræsthesias of various kinds (prickling, burning, tinnitus aurium, sensations of pressure and weakness of the eyes, etc.). More developed anæsthesias do not occur quite as frequently, and they never increase to high degrees of general or unilateral anæsthesia which distinguish hysteria, the more developed, modified form of the affection, especially in the female.

The same applies to disturbances of the *motor* sphere. Here, too, we encounter only the lower grades of motor debility: The facility to become tired, muscular weakness, sensation of faintness in the legs, tremor, etc.; convulsive contractions of individual muscles occur also, whereas *attacks* of convulsions and pronounced paralyses are absent in the clinical picture of

neurasthenia. But, on the other hand, we meet with vaso-motor and secretory disturbances of various kind, the same as in pronounced hysteria: A dry or a greatly perspiring skin, coldness of the hands and feet, alternating with suddenly appearing heat, salivation, etc. Brought about by the hypochondriac ideas and due to the irritable weakness of the respective central neurons, numerous varieties of *subjective disturbances of internal organs* appear, which, often, are elaborated systematically, by "autosuggestion," into actual "imaginatory" morbid pictures in the sentimental sphere of neurasthenic patients. Among these are *tabetic-like symptoms*: Pains in the back, girdle sensation, paræsthesias in the legs, etc., *cardiac neuroses* (palpitation of the heart, cardiac unrest, a sensation of suspension of the action of the heart with sensations of frightful distress, in which the frequency of the pulse may be greatly accelerated), and, furthermore, *genital debility* with frequent pollutions, sometimes accompanied with complete impotency, *gastric and intestinal neuroses*, with a sensation of bulimia, loss of appetite, nausea, cardiac pressure, vertigo, especially during the time of digestion (nervous dyspepsia), cardialgia with fainting spells, sensation of distention of the abdomen, constant constipation or nervous diarrhœas and colics. An object of special complaint is *insomnia*, which is either due to the sensations that torment the patient, or the result of overirritation and relaxation of the activity of the brain in general, and which exerts the most pernicious influence upon the course of the affection in that, with the loss of sleep, the patient has been deprived of the best means of recuperation of the nervous system and of the introduction of a healthy reaction, and thus the irritability of the nerves becomes more aggravated.

Neurasthenia, the same as hysteria, also develops acutely in consequence of an extraordinary mental overexertion and emotion. Usually it develops chronically in that the psychical constitution of the patient, mostly predisposed to weakness by inheritance, gradually deteriorates, owing to an unsuitable mode of living, bad nutrition, venereal excesses, constitutional anomalies (allegedly to gout, in particular) and infectious diseases, or to insufficient schooling and overexertion of the mind, and the brain, especially the cortical elements, gradually enters upon the condition of exhaustion and irritable weakness. Neurasthenia, in some cases, becomes concentrated upon one special functional sphere, for instance, the sexual organs, so that the latter attain a condition of weakness owing to excesses or to chronic gonorrhœa; in such cases a *"sexual neurasthenia"* is usually diagnosticated as a special form of neurasthenia.

The diagnosis of neurasthenia is not difficult, as a rule. It must be considered the first and most important rule never to make it until all the organs have been most carefully examined for an affection which is characterized by anatomical changes and precise clinical symptoms, i. e., *to diagnosticate neurasthenia only when no organic disease accounting for the nervous symptoms can be demonstrated,* if, therefore, nothing can be found, clinically and anatomically, that is contradictory to the assumption of an exclusive and at that a functional affection of the nervous system. If we thus proceed very cautiously, always considering this rule, we are cer-

tain to avoid a confusion of neurasthenia with tabes, incipient severe cerebral affections, non-nervous gastric and intestinal diseases, with angina pectoris caused by atheroma, aneurysm or adipose heart, with slight uræmic symptoms, etc. It is not necessary to enter upon a detailed differential diagnosis; and it is especially unnecessary, according to our conception of both affections, exhaustively to discuss apparent differences between neurasthenia and hysteria.

TRAUMATIC NEUROSIS

Traumatic Neurosis.—The name "traumatic neurosis" has recently been applied to clinical conditions in which it is also *a question of modified varieties of hysteria, respectively neurasthenia, which differ from the usual forms only in the ætiology, in that they are apparently the immediate effects of traumatism, especially of marked concussion (for instance, in railway accidents, "railway spine," "railway brain," etc.).* The connection of the above-named neuroses with traumatism and, evidently, also with the fright accompanying it, has been elucidated principally by the increasing frequency of railroad accidents. But the knowledge of these pathological conditions was increased, above all, by the fact that persons who were affected by these accidents have of late become entitled to claim indemnities, and, consequently, a physician had to give his opinion in regard to them.

The development and the clinical picture of traumatic neurosis are quite varying as to the details, so that it is not possible to outline a picture which covers every case.

After the action of the traumatism was immediately followed by stupor or unconsciousness, general paralysis and collapse (symptoms which correspond to the clinical picture of concussion of the central nervous system) or, possibly, after no manifestation pointing to severe affection of the nervous system had become prominent at first, a condition of general nervousness makes its appearance, due to the mental shock. This nervousness manifests itself as restlessness, irritability, gloomy disposition and concentration of the thoughts upon the accident suffered, loss of energy and, eventually, weakness of intelligence also. Besides, *vertigo* is mostly present, and violent *tremor* occurs upon any sort of psychical emotion and bodily exertion. In the majority of cases *insomnia* is present and a variety of *disturbances in the sensory sphere: Pains* of a vague character or concentrated upon certain parts of the body, as the head, the joints (articular neuralgias), but localized especially upon the part of the body that was affected by the traumatism. Furthermore, *paræsthesias* and *anæthesias* are found in the regions of the skin and of the nerves of the special senses: Hemianæsthesias, analgesia, dulness of smell, hearing, and taste, deficiency of sight, and, which is usually considered as being particularly typical, *concentric limitation of the field of vision.* In the motor region, also, various deviations from the normal condition manifest themselves; primarily, *paralyses,* or at least *pareses* (paraplegias, monoplegias, and hemiplegias, although without affecting the region of the facial and hypoglossal nerves,

abasia, and astasia), in which the most pronounced paralytic symptoms are manifest in the extremity which was affected by the traumatism; the same is the case in sensory disturbances. Disturbances of gait are quite common occurrences: Stiffness and slowness of the gait becomes manifest; sometimes it is like the ataxic, at other times like the spastic, gait of patients affected by diseases of the spinal cord. Speech and voice may also be impaired (aphonia, stuttering, mutism), fibrillary contractions of the muscles may take place, sometimes only after exertion or after application of the electric current. Besides, rigidity and contractures of the muscles, *convulsions* (among others, *tic convulsif*), or general convulsions in the form of *epilepsy,* are also observed. The cutaneous reflexes are often diminished, the *tendon reflexes,* eventually, increased, rarely diminished or lost. In some patients, finally, nervous symptoms relating to the heart and to the vascular apparatus are present, especially *palpitation of the heart, which in some cases is supervened by a gradually developing hypertrophy of the heart, acceleration of the pulse,* which is eventually increased by pressure upon painful areas of the body, more rarely *arrhythmia.* Slight disturbances of the bladder and sexual functions are also occasionally observed in the course of traumatic neurosis, as well as vaso-motor and secretory disturbances, the same as in hysteria and neurasthenia: Slight flushing of the face and of the chest, urticaria factitia, the hair becomes gray and falls out, etc.

To insure the diagnosis, an experiment may be made with the administration of 100 grammes of grape sugar. If (alimentary) glycosuria takes place about one to two hours after ingestion, this, in cases of doubt, will point to the presence of a traumatic neurosis in that it has been found that the production of an alimentary glycosuria is generally far easier accomplished in neuroses than under normal circumstances. However, this diagnostic expedient is not of very great value.

If we review the long list of symptoms which are considered attributes of traumatic neurosis, the above description will show that they are either of a purely subjective, or of a functional nature. It is a question, principally, of hysterico-neurasthenic phenomena, and there is no urgent reason in such cases to assume the presence of a special disease, "traumatic neurosis." It would be advisable rather to diagnosticate "a psychosis, hysteria, respectively neurasthenia, due to traumatism." These disturbances, which develop without pathologico-anatomical changes in the central nervous system, are opposed by others which are undoubtedly due to anatomical changes of the nervous system caused by traumatism; such as neuritis, hæmorrhage, multiple sclerosis, tumour of the brain, etc.

Differential Diagnosis.—In the diagnosis of nervous disturbances depending upon traumatism we must decide, in the first place, whether it is permissible, *without any doubt,* to connect them with the effect of the traumatism and, furthermore, whether *simulation* be present, as, of course, is exceedingly often the case in these morbid conditions. It is usually difficult to exclude the latter, as the simulating subject knows, as a rule, that a tenacious adherence to statements regarding pains, insomnia, mental depression, loss of memory, etc., will be of advantage to him. If the clinical

picture presents symptoms which under no circumstances can be simulated, such as changes in the electric reaction of the muscles (abnormal diminution of electric, especially of galvanic, irritability in the sphere of some nerves, longer-lasting fibrillary convulsion of the muscles after the action of the faradaic current, as observed by Rumpf), clonic convulsions of some muscles, pupillary difference, marked trophic disturbances, permanent, considerable acceleration of the cardiac action, or a greater frequency of the pulse upon the touching of painful pressure-points, or, finally, even a hypertrophy of the heart that develops under the eyes of the physician, simulation can be excluded at once. However, such cases are comparatively *rare*; usually all objective points of support for the diagnosis are absent, and a careful examination is required, as well as the application of several artifices, to differentiate between hysteria caused by traumatism and a simulation which is carried through systematically. It is impossible to give general rules to unmask the malingerer. The diagnostic success in this respect rather depends mainly upon the individual sagacity and experience of the physician. However, not enough stress can be laid upon the fact that the physician should not be induced, by the finding of one or the other symptom which is really present or by the determination that the same is "simulated," to make or to reject the diagnosis of traumatic neurosis. The *entire conduct* of the patient must always be observed, and it must be investigated with reference to the presence of a psychical disturbance in the meaning of hysteria or neurasthenia. We must always bear in mind that complaints about painful sensations, that cannot be controlled by an objective investigation, and a certain exaggerated emphasis of some morbid phenomena, are some of the characteristics of such a neurosis. In most cases it is advisable, therefore, first to observe the patient for some time before deciding whether a genuine simulation, a conscious, egotistic exaggeration, be present, or whether the latter be based upon a hysteric or neurasthenic condition caused by the traumatism.

EPILEPSY, FALLING SICKNESS.

It is well known that epilepsy is characterized by paroxysmally occurring *unconsciousness, with more or less pronounced typical general convulsions.* It is either a simple symptom of various severe cerebral affections (of focal affections, especially of brain tumours, which are located at the convexity), or a functional neurosis to which no demonstrable anatomical changes in the brain correspond. The latter is designated as primary, *idiopathic epilepsy,* the former as secondary *symptomatic* (Jacksonian [1]) epilepsy. The attacks which accompany the latter form are also

[1] The designation of "partial" epilepsy, i. e., the limitation of epileptic convulsions to individual muscles or muscle groups without loss of consciousness, as expression of the anatomical lesion of circumscribed cortical areas, with the name of Jacksonian epilepsy is no longer permissible, according to our present experience. For we find, on the one hand, in epilepsies depending upon cerebral lesions, loss of consciousness, in idiopathic epilepsy [illegible] consciousness in exceptional cases,

called *epileptiform*, to distinguish them from those of idiopathic epilepsy. The following explanations concerning the diagnosis of epilepsy refer exclusively to *idiopathic epilepsy*; regarding the diagnosis of symptomatic epilepsy, I refer to the discussion of the same in the preceding chapters.

Nature of Epilepsy.—It would be of importance for the diagnosis of epilepsy if we were fully enlightened regarding the *nature* of the disease. Numerous theories and experiments were not wanting in this respect; however, quite a diversity of opinion still prevails.

We must bear in mind, principally, that the patients may be absolutely well in the intervals between the paroxysms, and that the anatomical findings are absolutely negative in the majority of cases. We are compelled, therefore, to assume that occasionally occurring, rapidly disappearing, *functional disturbances in the central nervous system* are the cause of epilepsy. For there can be no doubt that the central nervous system, and especially the brain, is affected in epilepsy. This is indicated by unconsciousness accompanying the convulsions, by the frequent complication of psychical disturbances, and by the fact that pronounced, anatomically demonstrable affections of the brain are followed by epileptic attacks, which sometimes are not at all different from attacks of genuine idiopathic epilepsy. The latter fact and also the result of recent experiments which were made regarding epileptic convulsions, have gradually confirmed the opinion that the starting-point of epileptic convulsions should be looked for in the *cortex of the cerebrum*, and especially the *cortical motor regions* are areas the irritation of which produces the epileptic attack.

This view has been experimentally supported, especially by the fundamental observations of Hitzig and Fritsch and, later, by the investigations of Unverricht. Hitzig and Fritsch determined in their celebrated experiments regarding the electric irritability of the cerebrum that we are able, by electric irritation of the cortex of the brain, to produce well-characterized epileptic attacks, i. e., paroxysmally occurring convulsions which commence in the circumscribedly irritated musculature, then extend to the same side and later to the entire musculature of the body and which may be repeated several times, either with or without further irritation; the pupils were dilated *ad maximum* in such cases. These facts were confirmed later by Unverricht; it was found that, upon circumscribed electric irritation of the cortex of the brain, the spasm of the various muscles, or muscle groups, occurs in the sequence which corresponds to the topographic arrangement of the centres of the cortex, in a similar manner as we see the convulsions take place also in human epilepsy, at least in one half of the body. It is absolutely necessary for the experimental accomplishment of a complete paroxysm that the motor areas of the cortex are intact; extirpation of some portions of the same allows us to eliminate convulsions of certain muscle groups at will. After complete diffusion of these convulsions over one half of the body (the one opposite to the irritation), they extend to the other. If we extirpate the entire motor region of one hemisphere, the convulsive attacks occur only unilaterally; in the same manner it was determined in epileptic human beings that the clonic and tonic convulsions, after having occurred for years symmetrically on both sides of the body, became limited to one side when an apoplexy had paralyzed one half of the body. They may also be produced by irritation of the posterior regions of the cortex, in which dilatation of the pupils and of the palpebral fissure occurs first, then lateral deviation of the bulbi and nystagmus and, finally, the epileptic

---

whereas, on the other hand, we encounter general convulsions, even the initial cry, in the former, and a restriction of the convulsions to one half of the body, or even to a few muscles, in idiopathic epilepsy. *Therefore, I consider it to be more correct generally to apply the name Jacksonian epilepsy to "symptomatic" epilepsy, no matter in what form the attacks manifest themselves.* The principal merit of Hughlings Jackson was, in reality, not the discovery of limited epileptic convulsions, but the determination of the connection between convulsions and demonstrable affections of circumscribed cortical areas.

convulsion itself. Among the interesting results of Unverricht's experiments we mention especially that there is a certain area of the cortex from which a long-lasting *suspension of respiration* can be originated, and, furthermore, that changes also take place in the *vascular apparatus* which are independent of the muscular spasms and which during the attack are produced first by stimulation of one, then by that of the other, half of the brain. We are able here to determine a certain order of the changes of pulse and blood pressure: At first acceleration of the pulse and increase of the blood pressure, then retardation, then again acceleration of the pulse, followed by a return to the normal condition. If experimentally produced epileptic attacks follow each other in rapid succession, there arises in the animal a form of *status epilepticus*.

The opinion which prevailed formerly that the convulsions of epilepsy originate in the pons, i. e., in a convulsion centre situated in this location, does not appear to be very probable. It seems that only general tonic convulsions can be produced experimentally from this area, but certainly no epileptiform spasms of a chronic character. However, this is not meant to convey that the tonic convulsions in an epileptic attack are bound to originate in an irritation of the pons and that only clonic convulsions can be produced from the cortex; for the clonus called forth by irritation of the cortex increases to tonus which actually represents only a higher grade of clonus. I do not quite understand, according to what we know regarding the course of the motor tracts and the results of experimental investigations of this question, why, on the other hand, the possibility should not be admitted that convulsions similar to epileptic convulsions can be originated from the infracortical portions of the central nervous system (theory of the "mixed genesis of epilepsy"). However, we must consider, as I believe, the genuine epileptic attack which, besides tonic and clonic convulsions, manifests itself in *unconsciousness*, to be the exclusive result of irritation of the cerebral *cortex*.

It is not easy to decide what causes *unconsciousness* in an epileptic attack. Formerly it was ascribed to cerebral anæmia due to a spasm of the cerebral arteries respectively of the vaso-motor centre in the medulla oblongata. However, the supposed vaso-motor spasm would cause any other condition rather than adiæmorrhysis; besides, Todorski has recently proved experimentally that an increase of blood pressure and increased afflux of arterial blood to the cerebrum takes place in the course of an epileptic attack. As in experimental epilepsy, which is produced by irritation of the cerebral surface, at least a suspension or disturbance of consciousness of the trial-animals is observed (but no anæmia of the brain), and as, furthermore, quite a number of affections of the cortex, as we know from clinical experience, may cause unconsciousness, simultaneously the epileptic convulsions, it is probably to be assumed as the most likely theory that such an irritation of the cortical-motor areas, if it attains a more marked degree, is connected with a commotion of larger cortical areas embracing the association centres, respectively of the entire cortex. But for the present we are not able to explain how and why this is brought about, nor do we know why the cortex is placed in a condition of excessive irritation only temporarily and in certain, often very long, intervals. All theories concerning this subject are nothing else, as yet, but absolutely unproved hypotheses.

The symptoms of epilepsy are so pronounced and distinct in well-developed cases that the diagnosis does not meet with any difficulties. However, it is often difficult to recognise the rudimentary forms of the disease as epileptic and differentio-diagnostically to distinguish symptomatic forms of epilepsy from idiopathic epilepsy.

Epileptic Aura.—The usual picture of epilepsy is ushered in, in the majority of cases, by an *aura*, with nervous manifestations, which as a prodromic precede the epileptic attack proper, mostly immediately, rarely for some length of time, and which, according to their occurrence in the sensory, motor, vaso-motor, or psychical regions, are designated as sensory,

motor, etc., auræ. It recurs in the same patient mostly quite regularly, in the same manner and with the same symptoms, of which the following are the principal ones: Titillation, dragging pains, sensations of light and smell, tinnitus aurium, sensation of weakness, tremor, twitchings, sensation of cold, vertigo, hallucinations, etc. The duration of the aura is usually very short; it lasts from seconds to minutes, rarely for hours, and in a great number of cases it is *entirely absent*, i. e., the attack begins suddenly without premonition, being fully developed, characterized by its two well-known attributes, *loss of consciousness* and *spasm*.

Epileptic Attack.—The attack frequently begins with a loud cry, the patient falling to the floor *unconscious*; the spasms appear with a *tonic convulsion*, lasting from ten to thirty seconds; the entire body is in a condition of spasmodic rigidity in which opisthotonus and extension of the extremities are marked; with this the fingers and especially the thumbs show spasmodic flexion. The head is drawn back, the bulbi are turned upward and laterally, the pupils are dilated, the respiratory muscles markedly contracted. The deadly pallor of the face, which as a rule is present at the onset, gives place to a gradually increasing cyanosis, partly due to the cessation of respiration, partly to the compression of the jugular veins (in consequence of the spasm of the muscles of the neck). The stasis may become so marked that hæmorrhages occur in various portions of the body, a diagnostically important symptom, as under some circumstances it indicates the attack for some time afterwards, which may have occurred without the presence of witnesses.

Now, while unconsciousness continues, the period of *clonic spasms* of the face and extremities follows, which may run their course rapidly and severely so that grave injuries may occur (biting the tongue, luxations, fractures of bones, etc.). Usually the saliva appears at the mouth in the form of white, rarely blood-tinged, foam, as deglutition is no longer possible; flatus, fæces, urine and sperma may be voided. The reflex activity is completely annihilated, especially, as a rule, the reactions of the pupils to light. The pulse shows no constant condition, in the tonic stage it is most frequently small, to become more frequent and fuller in the clonic stage. The respiration is gasping, irregular, rapid; the skin, towards the close of the attack, is covered with profuse, sticky perspiration. The duration of this clonic stage of the spasm, which is especially characteristic of epilepsy, varies within wide limits—from one half a minute to five minutes; only exceptionally lasting longer than this.

Effects of the Attack—Psychico-Epileptic Equivalents.—When the convulsions cease, when cyanosis and dilatation of the pupils relax and respiration commences to be regular again, the patient awakens from his severe unconsciousness and his condition is normal again after a few minutes, but usually he shows for a long time more or less marked after-effects of the attack, i. e., the patients remain for some time in a somnolent condition, occasionally they are delirious or they present a picture of actual psychical alienation, the so-called "*post-epileptic insanity.*" However, conditions of mental disturbance are found in epileptics also independently of convulsive attacks, or in place of the latter (*psychico-epileptical "equiv-*

alents"), and common to these and to the "post-epileptic" psychical disturbances are: Impairment of consciousness, weakness of memory (especially during the time after the attacks), impulse to forcible actions under the impression of anxious sensations, psychical depression with predominance of perverse impulses, hallucinations, etc. The duration of these epileptic equivalents lasts from minutes to hours, rarely from days to weeks. After these severe mental disturbances, which occur in paroxysms, have terminated, the patients are usually without any remembrance of the events that took place during the attack. Regarding the details of the symptomatology of psychico-epileptical equivalents, their signification and their relation to other mental operations, I must refer to text-books of psychiatry.

Somatic Effects and Equivalents.—Of somatic disturbances subsequent to *epileptic attacks* the following are important: Albuminuria, which, however, according to my experience and that of others, is by no means constant, polyuria, and, very rarely, glycosuria. An increase of the temperature of the body by one to two degrees F. after the attack can rarely be determined. Violent attacks of perspiration have been observed as a *somatic equivalent* of epileptic attacks (and consciousness was sometimes retained, at other times impaired while they lasted), also *paroxysms of salivation* in place of the epileptic attacks.

If the attacks follow rapidly one upon the other, so that the after-effects of the first attack have not entirely disappeared when the second one sets in, it occurs frequently that a condition develops which, with excessive rises of temperature, leads to a fatal termination and which has been designated "*status epilepticus*" (*état de mal*). An analogous condition can be produced experimentally, as we have seen.

Varieties of Epilepsy.—It is of great importance for the diagnosis that, besides the above-described, fully developed, severe epileptic attacks, *slight rudimentary* and *irregular attacks* occur, the existence and course of which must be known to the physician if he desires to avoid serious diagnostic errors. As to the *slight rudimentary attacks,* they manifest themselves (besides a short, incomplete disturbance of consciousness) either in very slight twitchings of some muscles so that the attack sometimes creates only the impression of a slight *tremor,* or in paroxysmal losses of consciousness, without any convulsion of the musculature. In fact, the rudimentary form of epilepsy may be present in the form of simple *attacks of vertigo* (vertigo epileptica). We are able to recognise them as being epileptic if we consider that they recur in intervals and are ushered in by the indication of an aura, that disturbances of vascular innervation become very markedly prominent, that the position of the body has no effect upon the vertigo, and that the latter generally makes its appearance without any cause, quite abruptly. If attacks occur in the meanwhile, with loss of consciousness lasting for minutes, the diagnosis of epileptic vertigo becomes still clearer. In the *irregular* forms of epilepsy one of the principal phenomena of the epileptic attack becomes subordinate to the other in the clinical picture or it may even be entirely absent, so that, for instance, only the coma or only the convulsions become manifest. I believe that these dif-

ferences in the course of an epileptic attack depend entirely upon the intensity of the cerebral irritation that is present; the more pronounced the latter, the more decided is the unconsciousness.

Differential Diagnosis.—If we consider the above-named characteristics, the diagnosis of epilepsy can generally be made easily and correctly. However, we must always bear in mind that epilepsy is never anything else than a *symptom-complex* which represents an independent disease and can be diagnosticated as such only after the existence of those diseases can be excluded in the given case, which also present epileptic attacks among their symptoms. It is necessary, therefore, in *every* instance, even when the epileptic paroxysms appear to be quite clear, previous to making the diagnosis of idiopathic epilepsy, to consider carefully whether there is no reason for the assumption of *symptomatic* (Jacksonian) *epilepsy.*

Jacksonian Epilepsy.—We must consider, above all, whether there are no symptoms present which point to an anatomically demonstrable, direct or indirect affection of the cerebral cortex, especially to tumour or abscess, multiple sclerosis of the brain, progressive paralysis, etc. *The manner of the course of the epileptic attack is of no determining consequence.* There can be no doubt that the extreme types of the two clinical pictures, that of symptomatic and that of idiopathic epilepsy, are apparently quite different: In the one, suddenly occurring loss of consciousness, during which the patient falls with a cry, momentary tonic convulsion and, following the latter, clonic twitchings which extend over the entire body—in the other, in Jacksonian epilepsy, convulsions of some muscles or of an extremity, the course of which is observed by the patient, fully conscious! However, these are only the *usual,* pronounced types; besides, we observe in symptomatic epilepsy cases in which the convulsions are general and consciousness is lost, and, on the other hand, we note in idiopathic epilepsy that the spasms are restricted to a few muscles or to one half of the body and that the attacks terminate without any impairment of consciousness. Neither is the aura a differentiating symptom of these two varieties of epilepsy, as it also occurs frequently in Jacksonian epilepsy, either as a sensation of weakness preceding the convulsions in the limbs, which later are principally attacked by the spasm, or in other parts of the body, or in the form of pains, paræsthesias, hallucinations of vision, etc. Not even the initial cry is absent in symptomatic epilepsy, although this symptom is considerably rarer in the latter than in the idiopathic form. The *conduct of the patient during the intervals* is of much greater importance in the differential diagnosis. If we do not consider the psychical disturbances, which may be well developed in the intermissions in both morbid conditions, there are principally the permanent paralyses, hemiplegias, monoplegias, contractures and hemianæsthesias, violent headaches, vomiting and the development, which is of such importance diagnostically, of neuroretinitis or choked disk, etc., which point with certainty to the fact that an anatomical change in the brain is present, the symptoms of which then embrace, among others, epileptic attacks. If, on the other hand, not a single symptom favours the assumption that an anatomically demonstrable affection of the brain is the cause of the epilepsy, the diagnosis of idio-

pathic epilepsy may be made, the more so if the most significant ætiological factor of the same, a *hereditary epileptic or neuropathic taint, is evidently demonstrable* in the given case, and if the epilepsy has commenced early, i. e., not after the twentieth year of life.

Reflex Epilepsy.—Positive points of support for the diagnosis of genuine epilepsy are furnished by certain irritative conditions of the nervous system which precede the onset of the affection and which seem to be able reflexly to cause epileptic attacks ("*reflex epilepsy*"). It is possible that, after prolonged irritation of peripheral nerves by cicatrices, foreign bodies, tumours, inflammatory exudates, by intestinal parasites, abdominal herniæ, affections of the sexual organs, etc., epileptic convulsions may be called forth, the origin of which by means of the nerves of the periphery of the body cannot very well be denied. The intimate relation between epilepsy and those lesions and irritations of the peripheral nervous system is favoured, primarily, by clinical experience, namely the fact that the epilepsy, after removal of such irritations as affect these nerves, by excision of cicatrices, removal of foreign bodies, etc., may disappear without leaving any trace. But, furthermore, a number of experiments are in favour of this view; thus, to mention the best known of these experiments, Brown-Séquard succeeded in producing epilepsy in guinea-pigs by severing the sciatic nerve. The epilepsy took place spontaneously or by irritation of certain portions of the periphery of the body (so-called epileptogenous zones), and was even transmitted to the progeny. Marked psychical emotions and traumatism of the head may also give rise to the occurrence of epilepsy.

However, all these ætiological factors of epilepsy play more or less only the part of *causative* factors, *in that they affect a brain which is prepared and suitable for such a severe reaction to the irritation.* Often we are able in the given case at least to presume the cause of this abnormal, more developed capability of the brain to reaction. In a relatively large number of cases it is inherited, in others acquired—by intoxications (alcohol, lead, etc.), by constitutional diseases, especially diabetes mellitus (*diabetic epilepsy*), by deficient blood supply to the brain due to atheroma (*senile epilepsy*) or by an adipose heart with retardation of the pulse, and, furthermore, by infectious diseases (scarlatina, typhoid, etc.).

French investigators (Voisin and others) have recently maintained an interesting hypothesis regarding the origin of epileptic attacks. The urine of epileptic patients, injected into the veins of trial-animals, proves to be especially toxic after the cessation of a series of attacks, whereas hypotoxicity of the urine can be determined previously to, and during, the periods of attacks; it appears that, during the stage which intervenes between the attacks, the toxicity is the same as in healthy individuals. If, then, the toxicity of the urine decreases, we may assume a retention of the toxic substances in the blood; with it the prodromes of the epileptic attack set in: Dyspepsia, psychical irritability, etc. If the intoxication of the blood and, what coincides with it, the hypotoxicity of the urine, passes beyond a certain degree, an epileptic attack is called forth. After a series of attacks, the toxic substances are excreted *en masse* (hypertoxicity of the urine); if this excretion does not take place, post-epileptic mental disturbances or the dangerous status epilepticus set in. If these assumptions and results of investigations will be further confirmed, it would furnish us with a most interesting insight into the cause of epileptic attacks. It remains difficult to understand, however, why the kidneys are temporarily unable to excrete toxic substances, and it is still more difficult to conceive why, after the cessation of the attacks, these organs should be enabled to do so in an equally high degree, as *rapidly passing* disturbances of the renal secretion (irritation of the splanchnic nerve?) are said to occur owing to congenital or acquired factors, etc.

We have stated in the previous chapter (p. 727) that the differential diagnosis between epilepsy and *hysteria* may sometimes give rise to insurmountable difficulties, and it was emphasized at the same time how to

differentiate, in a measure, between a hysterico-epileptic and an epileptic attack.

Simulated Epilepsy.—As epilepsy is comparatively often *simulated* for selfish purposes, it is of practical importance to discuss how feigned attacks can be recognised as such, in contrast to genuine epileptic attacks. It is generally possible to unmask the malingerer at the first glance, because a strict adherence to the typical course of an epileptic attack is successfully carried through only by the smallest number of simulating subjects. However, cases do occur in which the differential diagnosis presents some difficulties. To the presence of epilepsy, in contrast to stimulation, point, in such cases, cyanosis, loss of reaction of the pupils during the attack, albuminuria, suffusions of the skin, the hæmorrhages in the retina and in the true vocal cords (as residues of past epileptic attacks) and, finally, the "post-epileptic" insanity.

Eclampsia.—Furthermore, convulsive attacks which take an *acute* course, which have loss of consciousness in common with epileptic attacks, and which are otherwise like the latter in all details, but which do not leave a permanent predisposition to convulsive attacks, have been differentiated from epilepsy under the name of "*eclampsia.*" I consider the *separation* of the latter from epilepsy, especially from "reflex epilepsy," an *artificial one which cannot be carried through in a diagnostical respect.* Only that much may be maintained: If epileptiform attacks occur in small children, especially often during dentition, in the presence of intestinal parasites, in gastro-enteritis, in febrile affections, etc. (*infantile eclampsia*), or if such attacks take place in anæmic, nervous adults, and especially in women during pregnancy or *intra partum* (*eclampsia parturientium*) (uræmia, of course, must be excluded in such cases), we may diagnosticate with a certain degree of probability, or—better to express it—we may entertain the hope, that the epileptic attacks in question will be of a *transitory* nature. For usually, under such circumstances, not more than *one* epileptic attack or at most a few attacks which rapidly follow each other will occur; it happens only in the rarest cases that the attacks which, according to the entire situation, must be primarily interpreted as eclampsia, become a permanent affection, i. e., that they pass into epilepsy proper.

Uræmia.—*Uræmic attacks* which occur in the course of renal affections are nothing else, according to their character, but epileptic attacks. This was proved, among others, by Landois, by a direct application of chemical substances contained in the urine to the surface of the brain. However, a confusion of uræmic with epileptic attacks is not very likely to occur in practice. The vomiting, the persisting coma, the results of the ophthalmoscopical examination (albuminuric retinitis), the demonstration of characteristic casts in the urine, etc., protect from diagnostic errors. At the same time, it is advisable, to avoid serious mistakes in diagnosis, *regularly* to undertake an ophthalmoscopical examination, and a microscopical investigation of the urine, upon the occurrence of epileptic attacks.

To confuse other convulsive conditions (trismus, anæmic convulsion of the face, spasms in the region of the accessory nerve, etc.) with epileptic convulsions, is impossible with even the slightest attention and upon longer observation of the patient.

CHOREA (CHOREA MINOR, SYDENHAM'S CHOREA, ST. VITUS'S DANCE)

A physician, after once seeing a case of pronounced chorea with its characteristic muscular unrest, with the inco-ordinate movements affecting a larger group of muscles, and with the psychical disturbances (in the meaning of mental weakness and increased irritability of the mind), cannot possibly mistake the disease. A detailed description of the symptoms, thus to facilitate the diagnosis of the affection, is of little value. Chorea must be seen; the picture of this affection impresses itself very easily, so that he who has closely observed the choreic movements in *one* case, will subsequently not be embarrassed correctly to recognise even indications of chorea as such. We wish briefly to mention only a few of the main traits of the disease. Without prodromal phenomena, or after a change of mental disposition and a relaxation of the mental faculties, rheumatic pains or disturbances of appetite and sleep have preceded, the pathognomonic disturbances of movement occur: Uncalled-for muscular contractions, which cannot be suppressed at all or only for a very short time, and which interfere obstructingly with the intended movements so that more delicate manipulations (writing, threading of a needle, etc.), walking and other co-ordinate movements become impossible. Especially characteristic are also the grimace-like contortions of the face and the disordered movements of the tongue which impede the speech. The latter faculty is also changed in that phonation of the patients, owing to inco-ordinate movements of the laryngeal muscles, has become impaired, and that sudden inspirations interrupt them during the process of speaking.

In contrast to the muscles with voluntary innervation, the activity of the musculatures of the heart, bladder and rectum, and also that of the muscles of respiration and deglutition, is not impaired. It is only exceptionally that a more marked frequency of the pulse or arrhythmia of the action of the heart is noted.

In spite of the restless contractures of the muscles, a sensation of *fatigue does not* occur; the muscular unrest disappears entirely during natural sleep or in sleep which is produced artificially; on the other hand, in waking it is increased by every psychical emotion.

The *mind* probably is more or less disturbed in every instance—from my own experience I know of no exception. *The depression of mental faculties,* an impairment of memory as well as of the entire process of thinking, always predominates. Besides, an increased irritability of the mental sphere may eventually manifest itself; pronounced psychoses also occur occasionally in chorea.

Disturbances of sensibility are absent; only the nerve trunks and, above all, some vertebræ are often sensitive to external pressure. Conditions of electric reaction are mostly normal; sometimes, as I am able to confirm from my own experience, an excessive irritability to the induced current can be determined. Such deviations from the normal condition cannot be utilized in the diagnosis. The *action of the reflexes* is unchanged, the ten-

don reflexes are normal, weaker, or, as in one of my cases, intensely increased. Some authors have found the *pupils* permanently dilated; personally, I cannot confirm the constant occurrence of mydriasis in chorea.

The temperature of the body is normal, *also the amount of urea* in the urine, whereas the excretion of chlorides in the urine is decreased, and albuminuria is occasionally noted at the height of the disease.

Chorea is essentially a disease of advanced childhood (the time from the second dentition to the end of puberty), and it occurs oftener in females than in males. Infectious diseases, especially measles, scarlatina and rheumatism, create a decided predisposition to the occurrence of chorea, also irritations of peripheral nerves and marked psychical emotions, such as fright, but, above all, and undoubtedly, pregnancy, and the first one more than a subsequent one.

Varieties of Chorea.—A special form of chorea, *Huntington's chorea*, is observed comparatively rarely; it is *hereditary* in certain families and makes its appearance almost always in the third decade of life. It is distinguished from ordinary chorea principally by its *chronic, progressive course*, and also by the fact that, although not without exception, the involuntary movements are not increased by the voluntary ones (respectively by intention of will), but *decreased*. It appears to be incurable and usually leads, in its later stages, to psychical weakness, to progressive dementia; it seems that epilepsy is a predisposing cause of the disease. To conclude from the results of autopsies, in hereditary, progressive chorea it is a question of nuclear infiltration of the cerebral cortex, with atrophy of the brain.

Chorea electrica is considered another variety of chorea; it is sometimes observed in children and is distinguished from ordinary chorea in that the individual convulsions, similar to those that are produced by electric stimulation, occur *lightning-like*. Sometimes they affect only some muscles, at other times a great portion of the musculature of the body. It is very doubtful whether this disease has anything at all in common with the ordinary form of chorea; it is probably identical with so-called myoclonia.

The frequent restriction of the convulsions to *one half of the body* (*hemichorea*) and the comparatively frequent complication of chorea with *endocarditis* and *joint affections*, are of importance, especially in the pathogenesis of the disease.

Nature of Chorea.—The last-named complications indicate that a certain connection exists between chorea and acute articular rheumatism. On the other hand, the frequent occurrence of hemichorea, the, according to my view, constant psychical disturbances in chorea and, finally, the fact that chorea occurs as a symptom subsequent to focal affections of the brain (ushering in the cerebral affection, or following hemiplegia, "pre- and post-hemiplegic chorea") are factors which locate the seat of chorea in the brain. As a matter of fact, the most various findings have been determined in the brain of persons that have succumbed to chorea. These findings were either of a diffuse nature (anæmia or hyperæmia of the brain or spinal cord), or they affected some areas of the brain in particular. Of the latter, those anatomical changes are of importance, above all, which were found in the thalamus and its surroundings, and in the tegmentum towards the red nucleus. But capillary extravasations, inflammatory changes, etc., have often been found also in the spinal cord, particularly in the cervical portion of the same. A case of Eisenlohr's deserves especial mention, in which a sclerotic area was found in the lateral column of the cervical portion of the spinal cord, as an isolated change in the central nervous system. But, on the other hand, no anatomically demonstrable change in the nervous system (neither in the central nor in the peripheral) could be determined in other cases, in spite of the most scrutinizing microscopical and macroscopical examinations. According to my view, we should always consider chorea to be essentially a *co-ordination disease*. In keeping with what I have explained on p. 542, etc., regarding the prob-

able course of the co-ordination tracts, it is not astonishing that *anatomical changes* taking place in the most varying locations in the central nervous system (from the periphery to the cortex) may give rise to chorea. But it is also obvious that those co-ordination tracts, the affection of which is followed by chorea, may be altered functionally not only by evident pathologico-anatomical changes, but also by *chemically acting* substances, thus, for instance, by mercury (I know cases of chronic (industrial) mercury poisoning, the course of which ran under the exquisite picture of chorea), by a specific infectious noxa, which acts similarly to the virus of articular rheumatism, and, occasionally, by acute articular rheumatism itself. That the latter may be the case is proved by instances in which chorea developed intercurrently in the course of acute rheumatism, and disappeared before the termination of the original disease, or by other instances in which both affections actually alternate, and, furthermore, by the fact that cases of chorea become more frequent during epidemics of articular rheumatism. However, it would not be correct, according to my opinion, in *all* cases in which no anatomical changes can be demonstrated in the brain and in which, on the other hand, recent endocarditis is found, besides chorea, to derive the latter from an infection with the virus of articular rheumatism. The justification of such an assumption is contradicted by the absence of fever in chorea, and, furthermore. by the fact that such specifics which promptly act against articular rheumatism, are almost always ineffective in cases of the neurosis, chorea. I also consider it a mistake to try to connect the origin of chorea with a transmission of products of a complicating endocarditis, because chorea occurs very rarely in older individuals in whom valvular defects are so very common, nor is it observed in the clinical picture of septicopyæmia in which embolizing endocarditis forms one of the most important symptoms. I do not mean to deny that emboli into those portions of the brain which are in a direct connection with the cause of chorea, especially into the posterior thalamic artery, may occasionally give rise to hemichorea, and this fact, of course, does by no means contradict our views regarding the nature of the disease.

The above-described factors which concern the pathogenesis of the disease must under all circumstances be considered in the diagnosis of chorea, if we mean to arrive at an understanding of the origin of chorea in the individual case. The simple determination of the presence of chorea is easy in all cases; for it is scarcely possible that chorea may be confused with another affection if the attending physician is at all acquainted with the form of manifestation of motor disturbances in paralysis agitans, in disseminated sclerosis, tetany, etc., i. e., in diseases which have pronounced disturbances of motion in common with chorea. But it may occasionally occur that chorea cannot be differentiated from *athetosis*. This is not very remarkable, because both diseases may pass directly into one another, and athetosis does not as yet represent a sharply defined neurosis, as will be seen from the following discussion.

ATHETOSIS

Characteristics and Differential Diagnosis of Athetosis.—It has been agreed to apply the name of *athetosis* to a condition of muscular unrest in which involuntary, moderately rapid but continuous movements take place, which show a certain regularity and which the patient is not able to suppress at all or only for a short time. These movements occur most frequently and most pronouncedly in the hands and fingers; the latter are constantly pronated and supinated, extended and flexed again, spread open, etc.; the foot and toes, trunk and head are less frequently affected. The movements are not as unlimited, hasty and atypical as in *chorea*, nor do they occur, as in this affection, in special connection with intended movements, so that the abnormal

movements show less the character of disturbance of co-ordination than is the case in chorea. However, the signs of such a disturbance are by no means entirely absent in the clinical picture of athetosis; the muscular unrest may also, the same as in chorea, be intensified by involuntary movements and may cease entirely during sleep. The differentiation of athetosis from *tremor* rarely ever presents any difficulties. It is true, common to both affections is the monotonous, continuous form of the involuntary movements; but in athetosis they occur in marked excursions, in the various forms of tremor they manifest themselves in more or less rapid, slight oscillations.

Various Forms of Athetosis.—Athetosis is usually observed unilaterally (hemiathetosis), occasionally also bilaterally (athetosis bilateralis). It is sometimes only the symptom of a well-characterized affection of the brain, rarely of the spinal cord (symptomatic athetosis), at other times it is an independent disease of unknown origin (idiopathic athetosis). In the latter case it is a question of a, mostly bilateral, muscular unrest (without hemiplegia and other manifestations of nervous disease), which occurs in previously healthy adults, but is usually present from earliest childhood or it is congenital and persists during life. Idiocy and, possibly, also epilepsy and hysteria are sometimes accompaniments. The condition in symptomatic athetosis is generally that a unilateral muscular unrest follows cerebral focal affections with hemiplegia, particularly after affections of the optic thalamus and corpus striatum, and especially also after infantile cerebral paralysis. This "post-hemiplegic" athetosis is generally considered to be identical with so-called post-hemiplegic chorea. But idiopathic, bilateral athetosis is also very closely related to chorea. For although, as stated, certain characteristic symptoms can be emphasized in both diseases, they are by no means always well developed so that at one stage of the disease the case may be classified as chorea, at another as athetosis.

Case of Transition of Athetosis into Chorea.—The following case which occurred in my practice proves most conclusively that both diseases, idiopathic athetosis and chorea, may even merge into each other, and that *it is more advisable for the present not to attempt a distinct nosological separation of the two affections.*

The patient was a labourer, thirty-seven years of age, who for four years suffered from muscular unrest following, as he claims, a severe cold; it consisted in rarely occurring convulsions in the region of the left facial nerve and in the muscles of the trunk, with continuous, uniform movements of the arms and legs: regularly alternating supinations and pronations, flexions and extensions of the fingers, flexions and abductions of the legs, flexions and extensions of the toes. The movements ceased entirely during sleep but increased upon voluntary movements, and slight disturbance of co-ordination manifested itself in the latter case. The pupils were not dilated and painful pressure points at the vertebral column were absent. The sensorium was unimpaired, *the boundaries of the heart were normal, the sounds of the heart were pure.* Four weeks after his admission to the clinic a considerable increase of the muscular unrest occurred, and the regular type of the involuntary movements ceased. They were then enormously intensified by active and passive movements—the former picture *of athetosis had merged into that of chorea with its disturbances of co-ordination, the irregular muscle contractions, etc., which were well marked in all details.* At the same time, an *endocarditis* occurred with systolic and diastolic murmurs and, later, a disturbance of the psychical conduct, delusions of persecution. The chorea gradually improved during the course of the following two months, so that the patient was able to walk without assistance; he left the hospital with a myocardic implication.

PARAMYOCLONUS MULTIPLEX—MYOCLONIA

The name paramyoclonus multiplex is applied to an affection which was first described by Friedreich, in 1881, and which is characterized in that a number of muscles of the trunk and of the extremities are *symmetrically* or asymmetrically affected by short, lightning-like, clonic convulsions, which may sometimes increase to tetany of short duration, and which generally have no or only very slight locomotor

effects. Mostly they do not occur isochronously, but arrhythmically, sometimes with moderate frequency, at other times exceedingly often (up to 100 and above per minute). If the patient performs an active movement or if his attention is distracted, a diminution of the convulsions occurs, and, *vice versa*, an increase of the same, upon mental emotions; they cease during sleep. Reflex irritability is increased, the patellar reflexes, in particular, have become intensified; the other nervous functions are normal throughout. Fibrillary twitchings and slight disturbances of sensibility are sometimes observed, besides the spasm.

The affection may be considered a functional disease of the nerves. The neurotic character of the same is indicated in that the convulsions are partly brought about subsequent to a marked psychical emotion, especially fright (Friedreich designated the disease directly as a "*neurosis of fright*"), in other cases they were greatly influenced by mental emotions and could be rapidly cured by electricity. Nor was it possible in Friedreich's case, the only genuine case of paramyoclonus which came to autopsy, to demonstrate any pathological changes either in the muscular or in the nervous system. The question is only whether paramyoclonus is to be considered an independent disease or whether it should be classified as *hysteria*. It is certain that in hysteria, besides exquisitely hysteric stigmata, the picture of paramyoclonus sometimes appears as a hysterical symptom, but it is also a fact that cases of the latter exist in which no other sign points to hysteria, in spite of a long duration of the disease. The fact that those patients who were affected with this disease were, almost throughout, men of an advanced age, is also against the simple subsumption of myoclonia under hysteria; besides, it depends upon the extent of our conception of hysteria.

If we adhere strictly to the picture as described by Friedreich, it is not possible to confuse paramyoclonus with other known neuroses. *Choreic* convulsions bear the greatest resemblance to the same. The character of these convulsions, as the expression of deficient co-ordination, their intensification by impulses of will, and an eventual connection of the affection with articular rheumatism, decide in favour of chorea in cases of doubt.

Tic Convulsif.—The relations between paramyoclonus and *tic convulsif* require a separate discussion. Whereas, in paramyoclonus, only the muscles of the trunk and of the extremities are, almost without exception, affected by the convulsions, there occur some few cases in which the sphere of the facial nerve is also affected. A difference between ordinary tic convulsif and the facial twitchings which occur in the clinical picture of paramyoclonus could then be found only in the fact that the twitchings in the latter case, as in paramyoclonus generally, are most marked during rest, whereas in tic convulsif they diminish during such periods. In so-called *tic général*, the "*maladie des tics convulsifs*," the same as in paramyoclonus, the spasms are also calmed by distracting the attention of the patient and by pronounced impulses of the will, and the affection is probably closely related to paramyoclonus in other respects as well. However, a confusion is scarcely possible, in that "*maladie des tic convulsifs*" is usually not characterized by separate movements, but by *combined* movements which take place forcibly, to which are added the, likewise compulsorily occurring, utterance of inarticulate sounds or short words (of an obscene nature—coprolalia, or, compulsorily repeated—echolalia), and the intermingling of morbid primordial delirium, symptoms which are specific of *maladie des tics convulsifs*.

TETANY

The name *tetany* comprises *tonic, intermittent, spasmodic conditions* which take place without loss of consciousness and which are mostly accompanied with pain. They are characterized by certain peculiarities which distinguish them from other similar spasms; these symptoms, pathognomonic of tetany, are briefly as follows:

1. Pressure upon the principal nerve trunks (or vessels) can increase the existing convulsions, or it may be able to call forth convulsions during the periods which are free from attacks (Trousseau's phenomenon).

2. Furthermore, *the electric irritability of the nerves*, especially to the galvanic current, is, almost without exception, *markedly increased* (Erb), so that even relatively weak currents are able to produce cathodal closure tetany or even *anodal opening tetany*, in fact *cathodal opening tetany*; the increase of irritability by the faradaic current is less constant and less considerable. It seems that the electrical irritability of the *sensory nerves* is also increased in the majority of cases (Hoffmann).

3. Finally, Chvostek has found that the *mechanical irritability of the nerves* is increased in tetany, so that on tapping the nerves with the percussion hammer, sometimes even upon stroking with the finger, spasms occur in the corresponding muscles; the mechanical irritability of the *muscles* themselves is *not* increased.

However, Chvostek's phenomenon, most manifest in the region of the facial nerve, is found also in other nervous affections, in fact, sometimes even in healthy individuals; but it is certain that it is never found as frequently and of as equal an intensity as in tetany. Chvostek's phenomenon cannot be considered a simple expression of increased reflex irritability, as the reflexes are by no means regularly intensified in the same, although occasionally a considerable increase of the patellar tendon reflex is observed in patients afflicted with tetany. As to the cause of Trousseau's symptom, there is no question that it is brought about mainly by pressure upon the nerve trunks, and only indirectly by pressure upon the large arteries, as is probably definitely proved by the experiments on dogs of von Frankl-Hochwart.

The muscle spasms affect mostly the extremities of *both sides*, and that preferably the upper ones. As a rule, the flexors are in tonic contraction; most frequently the foot assumes the plantar flexion, the hand the position of the writing hand (respectively "the obstetrical hand"). But quite a number of other spasmodic positions occur also (spasmodic spreading of the fingers, clinching of the hand, etc.); the forearm is bent towards the upper arm, the latter pressed against the trunk. The patients are not able during the spasm either to flex or to extend the firmly contracted muscles. Occasionally we observe also spasms of the muscles of mastication, or those of the eyes (spastic strabismus) or of the muscles of the neck, chest and back; spasms of the tongue, larynx, pharynx, and diaphragm are rare; a tonic spasm of the diaphragm was also recently observed by me in an attack of tetany. The *frequency* of the convulsions varies considerably in the different cases; sometimes the attacks follow each other in rapid succession; at other times the intervals between the attacks last for days. The *duration* of the attack extends over minutes or hours, in rare cases even over days; they are called forth by mental emotions, voluntary movements, by irritation of some painful vertebræ, or, as previously stated, by pressure upon the nerve trunks of the arm; but mostly they occur without any demonstrable *cause*. In the above-mentioned case of tetany that was associated with spasm of the diaphragm, the paroxysm was brought about by deep inspirations during the examination of the lungs.

Subordinate Symptoms.—*The following subordinate symptoms of tetany are observed*: Preceding the spasms, often for a considerable length of time, are paræsthesias, pains, anæsthesias, increase of electric irritability of the gustatory and auditory nerves, headache and vertigo, and, furthermore, *trophic and secondary anomalies*, i. e., rather regularly, marked perspiration, especially following the attack, sometimes polyuria, albuminuria and glycosuria, and, also, herpes zoster, œdema,

loss of nails and hair, etc. Finally, *epileptic* convulsions and especially also, occasionally, *psychical* disturbances of various kinds and degrees may be associated with tetany. *The temperature of the body*, usually normal, has been found in some cases to be feverishly increased or even subnormal.

Ætiological Diagnosis.—A consideration of the *ætiology* of the affection is of importance in the diagnosis. Tetany has been observed in the course of *infectious diseases* (scarlatina, measles, malaria, enteric fever, and others) and subsequent to *intoxications* (after poisoning with ergotin, chloroform, lead, etc.), possibly also as an expression of alcoholism and uræmic intoxication. The connection of the disease with *pregnancy, puerperium* and *lactation*, is undoubted, also with *rhachitis* (as has been positively demonstrated by the most recent scientific investigations), and, which appears particularly interesting, its causal connection with *extirpations of goitre* is likewise certain (N. Weiss). In the latter respect it has been found that an extirpation of the gland gives rise to tetany only when nothing, or only a small, functionally insufficient portion, of the organ remains, whereas partial extirpations of goitre, both according to clinical and to experimental experiences, do not produce tetany (von Eiselsberg). *Affections of the stomach and of the intestinal canal* also appear to favour the occurrence of tetany; it is noted to take place after gastric and intestinal catarrhs especially in children, and in gastrectasis in adults. However, the last-named causal connection is certainly not a frequent event. During the last fifteen years I have not seen one single case of pronounced tetany which could be ascribed to the presence of gastrectasis. This gastric form of tetany is said to occur reflexly by tugging of the gastric nerves or by auto-intoxication due to the development of toxic substances in the putrefying gastric contents, or by both conditions simultaneously. Finally, tetany is frequently a disease which occurs in *epidemics*, which make their appearance especially often during the months of February and March. The affection then attacks preferably certain tradesmen (shoemakers and tailors), youthful male individuals; this variety of the occurrence of the affection is not explained as yet. Refrigerations, overexertions and psychical emotions may favour the causation of the disease; the latter occurs decidedly oftener in some places than in others. The original cause of epidemic, idiopathic tetany is unknown as yet; it is more than probable that an infectious virus may be surmised for idiopathic, epidemic tetany.

Differential Diagnosis.—Only few diseases are to be considered in the differential diagnosis. Tetany can scarcely be confused with *tetanus*, which presents an entirely different general picture. Rigidity of the neck, opisthotonus, early occurring trismus, the enormous reflex irritability, are morbid manifestations which are characteristic of tetanus, but which are absent in tetany. The differentiation of tetany from certain forms of *hysteria* may be less easy, in that the tonic spastic positions of the hands and arms may simulate tetany. Determining in such cases in favour of the diagnosis of hysteria are: The occurrence of the spasm on only one side, which is seldom observed in tetany, the abrupt change of position of the spasmodically contracted member, the psychical alteration, the intermingling of other hysteric phenomena, for instance aphonia, with the picture of the attack, the decided effect of suggestion, and, finally, the absence of Trousseau's and, above all, of Erb's phenomena. The diagnosis of tetany becomes more difficult if some traits of its clinical picture are developed atypically or not at all; we are entitled then to leave the diagnosis in suspense. Thus it may occur that continuously tonic spasms of the extremities develop in small children (designated specially *arthrogryphosis infantum*), which, however, owing to their continuous character, in contrast to the intermittent spasms of tetany, do not appear to belong to the latter.

On the other hand, it may even occur, sometimes, that the spasms *are entirely absent*; then the affection lacks the most important, most conspicuous, diagnostic symptom. But if, in such cases, Trousseau's and Chvostek's phenomena are developed or if the electric irritability is increased, and if, at the same time, a firm ætiological base is present also, thus, for instance, total extirpation of the thyreoid gland previous to the occurrence of these symptoms, we are justified, in spite of the absence of spasms, to assume at least a "*tetanoid*" condition. We may expect in such cases that the appearance of the characteristic spasms will sooner or later complete the pathological picture.

SHAKING PALSY, PARALYSIS AGITANS, PARKINSON'S DISEASE

The diagnosis of paralysis agitans which, for the time being, for want of positive anatomical autopsy reports (we do not refer to chronic neuritis which was determined in some cases), must be included in the neuroses, is not difficult if it is a question of well-developed cases, and if the physician knows the clinical picture of the disease from former observations. The more or less continuous *tremor*, the retardation of movements, the *rigidity of the muscles*, the *attitude of the body in rest and in motion*, and, finally, the *lack of mimic expression of the face of the patient*, give such a typical appearance to the affection that it is usually recognised at the first glance, and is scarcely ever confused with other nervous or muscular affections.

The malady occurs almost exclusively in more advanced age. Nothing certain is known ætiologically; traumatism, severe mental emotions, especially fright, may give rise to the occurrence of paralysis agitans. In one of my cases the affection appeared in a man at the moment when he saw his child run over by a wagon. *Anatomical* changes of the central nervous system have recently been found repeatedly, and that in the form of perivascular scleroses in the anterior, and partly in the posterior, portions of the spinal cord. To conclude from the existing, positive, anatomical findings (Redlich, Sander and others), it is a question of *senile* processes—*arteriosclerotic processes* and *glia proliferations*—which may develop excessively in patients suffering from paralysis agitans, and which affect not only the white, but also the gray, substance of the spinal cord. If the above-named changes develop in the anterior horns, it is probable, as assumed by Sander, that the contact of the motor neurons with the dendrites of the anterior horns is impaired, and that, owing to this persistent interruption of the conduction of motor innervation, tremor results. If the process advances still further, a diffuse destruction of the nerve fibres will take place in the gray substance, in the anterior lateral columns and also in Goll's columns, thus accounting for the more severe disturbances of motor innervation and the tabetiform symptoms which have recently been observed as secondary signs in several cases of paralysis agitans.

Tremor.—The following details should be noted in a diagnostic respect. The *tremor* usually commences in the hands (preferably in the right hand) and is most developed in the same. The fingers are then kept in a half-flexed position in all joints, the thumb is half opposed and trembles in this position simultaneously with the fingers, or it is continually contracted in this opposed position and relaxes (pill-rolling movement). The tremor gradually extends from the hands to the arms and legs, to the trunk and head. The trembling movements are usually communicated to the latter by the general shaking of the body, but sometimes they are independent

oscillations, as I have seen. Speech may become tremulous, owing to tremor of the vocal cords; it is usually hesitating and monotonous, probably on account of the rigidity of the laryngeal muscles. The trembling movements in paralysis agitans occur comparatively slowly (four to five times per second) and uniformly; they become more marked by *psychical emotions* and by changes of the weather, as I have distinctly seen in one of my cases, but not on closing of the eyes. *Energetically performed, voluntary muscular contractions generally cause a diminution of the tremor,* but forced, active movements and such as require close attention, for instance, writing, do not seem to increase the tremor. D. Gerhardt has recently called attention to the fact that the oscillations in paralysis agitans are transitorily aggravated immediately after the arm has been brought into a resting position, subsequent to a voluntary movement. The trembling movements extend over the entire body in the majority of cases; in other cases only two extremities, or even one only, or one half of the body are affected; almost without exception they cease entirely during sleep.

Muscular Rigidity.—A second manifestation, *rigidity of the muscles*, associates itself with the tremor. It is at least quite as important for the diagnosis of paralysis agitans as the tremor, as the latter is very slight in some cases or may *even be entirely absent for a long period*, i. e., it does not occur until the later stages of the disease. The rigid expression of the face, the bent attitude of the body, the *forward inclination of the head and trunk*, the permanent position of the extremities, especially of the fingers, in a flexed position, the difficulty to perform passive movements and the difficulty with which the patients are able to perform complicated active movements, such as rising from a chair, change of posture, etc., are the expression of this muscular rigidity. The latter also gives rise to a further, very characteristic symptom of paralysis agitans: In that a patient, afflicted with this malady, owing to the muscular rigidity, is not able rapidly to change the centre of gravity of his body, it becomes impossible for him, on forward movements of the body, to stop suddenly. He is, therefore, threatened, in walking, to fall over forward (*propulsion*); this condition is even more marked in backward movements (*retropulsion*), so that he may even fall. Propulsion and retropulsion are especially well marked if a slight push is given to the patient, either forward or backward. "*Paralysis*" of the muscles is but slightly indicated, as a rule; the motor power, although somewhat reduced in general, and although the apparently normal, strong muscles become rapidly exhausted, yet actual paralysis, as might be expected from the name of the disease, is only noted in the most advanced cases, and even in these it is comparatively slightly developed.

Sensory disturbances, apart from periodical, spontaneously arising, pains, are not observed in paralysis agitans, neither is there an alteration in the *excretion of urine and fæces*; the reflexes are nearly always normal, as well as the electrical contractility of the nerves and muscles. The patients commonly complain of a disagreeable, marked sensation of heat; the temperature of the body, however, is not raised, whereas the temperature of the skin actually was frequently found to be several degrees higher (up to $8\frac{2}{5}$° F.). The pulse frequency remains unaltered; in some cases there is an increase in the secretions of sweat and saliva. The psychical condition of the patient is usually not altered; but the patients are much tormented by internal unrest and by insomnia; in some cases the signs of senile dementia develop.

Differential Diagnosis.—A confusion of paralysis agitans with other similar morbid conditions does not occur readily. If only one symptom of paralysis agitans, the tremor, is regarded in the diagnosis, senile tremor, alcoholic tremor, saturnine tremor, mercurial tremor, etc., or the tremor in multiple sclerosis of the brain and spinal cord, progressive paralysis, etc., may have to be considered in the individual case. The observation of the, simultaneously present, muscular rigidity, the characteristic posture of the body, the rigid expression of the face, the phenomena of propulsion and retropulsion, etc., at once removes all diagnostic doubts. Usually,

however, the *character of the tremor* is sufficiently characteristic to be utilized in the diagnosis: The tremor in sclerosis, like the tremor of paralysis agitans, takes place in relatively long waves, but in the latter it is continuous. It persists also during rest, and on energetic contraction of the muscles it becomes rather fainter; in this connection, therefore, it varies from the tremor in multiple sclerosis, in which a stoppage occurs during rest, to reappear at once when the patient attempts muscular movements ("intention tremor"). I should like, however, especially to remark that deviations from this fundamental type of tremor in both diseases, according to my experience, do not at all permit us at once either to exclude or to assume one or the other of these affections on this account. Similar conditions prevail in the differential diagnosis between paralysis agitans and dementia paralytica, traumatic neurosis and hysteria. Confusions are possible only when the tremor is predominant in these conditions. But such mistakes in diagnosis are always due to the fact that the affection has not been observed accurately, or that the physician is not familiar with the condition. Confusion with simple senile tremor is most likely to occur, which, it appears, actually represents an affection closely related to paralysis agitans. Mistakes in questionable cases are best avoided if we adhere to the strict rule not only to observe individual symptoms, but to consider the entire clinical picture of the disease, which is so characteristic in paralysis agitans.

MYOTONIA CONGENITA—THOMSEN'S DISEASE

Myotonia, which became known by Thomsen's description about twenty years ago, is a rare affection, but it is characterized by very marked symptoms. It is a question of a hereditary affliction present in some families, which shows itself in the fact *that the muscles, after they have remained at rest for some time, upon voluntary movements assume a condition of tension with increasing tetanic stiffness.* This latter condition becomes more and more aggravated at the beginning of the movements and renders them very difficult; the patients have the sensation of marked resistance in every muscular action. *This difficulty of movability disappears in the course of active movements of the muscles, so that the latter may then be used quite without hindrance,* the patients having the sensation of normal movements. This pathological condition of the muscles is exaggerated by forced exertion and psychical emotions and also under the influence of cold. All muscles of the body may be affected sometimes, at other times only individual muscle groups, most rarely the muscles of the eye and the respiratory muscles. The disease may be looked upon as *myopathic,* perhaps due to a condition of disturbed metabolism, which opinion is favoured by the intact condition of all nervous functions, by the microscopical changes in muscle particles which have been excised, and by the completely negative findings in the examination of the medulla oblongata, of the spinal cord and of the peripheral nerves, including the intramuscular portions (as is proved by a case of Dejerine and Sottas upon which an autopsy was held).

Objectively we can observe: Remarkably strong development of the musculature, without increased or, on the contrary, with decreased strength of the same, and a normal condition of the cutaneous and tendon reflexes. The *conditions of mechanic and electric reactions* ("myotonic reaction"), the details of which were disclosed by Erb, are pathognomonic. The mechanic and electric irritability of the motor nerves is not increased generally, but rather decreased (only a summation of irritations calls forth tonic, persisting contractions). *On the other hand, however, the irritability of the muscles has been intensified in all directions.* Stronger *faradaic* currents produce marked, and, which is essential, *sluggish contractions of the muscles, persisting*

after the cessation of the current; weak irritation causes only short, lightning-like spasms. The anodal closure contraction may predominate on application of the *galvanic* current; but tonic, persisting contraction of the muscles becomes manifest above all; upon a stable action of the current we observe (although by no means regularly) "undulating contractions which follow each other rhythmically, which pass successively over the muscles and quite correctly originate in the cathodes and are directed towards the anodes." *Mechanical* irritations, i. e., percussion of the muscles, in fact, often even pressure with the finger, provoke a sluggish tonic contraction of the affected muscle, persisting after the irritation has ceased. A microscopical examination of excised muscle particles revealed hypertrophy of the muscular fibres with increase of the nuclei and partial obliteration of the striation.

The above-described, characteristic conditions of reactions, which are pronounced only in myotonia, allow of the positive recognition of Thomsen's disease; however, a pressure of the hand is generally sufficient at once clearly to show the presence of the affection, in that the patient is not able rapidly to let go of the hands, and, this being accomplished, he encounters difficulties again to open the hand, which is still partially closed in a tonic spasm. Myotonia cannot be confused with another nervous or muscular affection; although tonic contraction may be simulated in voluntary contraction of the muscles, yet the pathognomonic electric conduct of the muscles will at once and finally remove any doubt as to the existence or the non-existence of myotonia.

SO-CALLED ANGEIONEUROSES AND TROPHONEUROSES

This designation subsumes certain clinical pictures which manifest themselves principally in changes of the blood supply and disturbances of nutrition and growth, and which characterize themselves as nervous affections in that anatomical changes of the vessels are not found and that undoubtedly nervous symptoms are associated with them. Therefore, they are usually considered as the expression of affections of the vaso-motor and trophic nerves. Aside from Graves's disease, more infrequent pathological conditions, which in part have not been noticed until quite recently, must be considered in this case. They are either independent diseases, or nothing but symptom-complexes which occur in the train of known nervous affections, but which may become so prominent in individual cases that they attain a certain independence and, designated by a special name, are diagnosticated as such. The special morbid pictures are strictly defined clinically, but their origin is by no means clear as yet. It is to be desired and to be hoped that physiology will elucidate the effect of the nervous system upon metabolism in general, and upon nutrition and growth of the organs in particular. The prospects are favourable, according to the latest discoveries of Gaule and others, that this will be the case. Not until then will it be possible to obtain a closer insight into the cause of those affections, and to secure their permanent classification.

PROGRESSIVE FACIAL HEMIATROPHY AND HEMIHYPERTROPHY

Facial Hemiatrophy.—*Progressive unilateral atrophy of the face*, a rather rare disease, is characterized by progressive wasting of the soft parts, in some cases also of the bones, of one half of the face. Besides the latter, the tongue and the palate may also be affected by this atrophy.

The skin of the patient shows distinct disturbances of nutrition on the affected side of the face: Loss of pigment in some places and abnormal accumulation of pigment in the form of white or brownish-yellow patches, thinning and desquamation, and, furthermore, decrease of sebaceous secretion and loss or decoloration of the hair. Owing to the wasting of the underlying layer of fat, the affected half of the face loses its natural fulness, shrinks and contrasts markedly with the bloom-

ing appearance of the healthy half of the face; this is still more so if the bones and muscles are involved in the atrophy. The electric irritability of the muscles is usually unchanged, also the sensibility of the skin. The vessels, curiously, do not take part in the atrophy; and phenomena which point to a disturbance of innervation of the cervical sympathetic nerve, i. e., lower temperature, alteration in the size of the pupils, etc., are almost never observed. Mendel's demonstration of a chronic neuritis of the fifth nerve in a case of facial hemiatrophy, which came to autopsy, the occurrence of partial facial atrophy, corresponding to the area of distribution of individual branches of the trigeminus, and the latest physiological investigations regarding the function of the trigeminus ganglion, allow of the assumption that an affection of the trigeminus is the cause of progressive facial hemiatrophy.

The affection can only be mistaken for *congenital asymmetry of the face*, in which, however, the above-mentioned signs of nutritive disturbance, especially of the skin, and the progressive character of the disease after the body has attained full growth, are absent.

In contrast to the just-described condition of facial atrophy, a *unilateral facial* hypertrophy has been observed in some rare cases, in which the affected half of the face increased greatly in volume, the tongue and palate also taking part in the hypertrophy. As signs of the abnormal cutaneous nutrition appear: Increase of the sebaceous secretion, more marked growth of the hair upon the affected side, pigmentation of the skin, etc.

ERYTHROMELALGIA

By erythromelalgia we understand a symptom-complex, the principal components of which are pain, reddening and swelling of the ends of the extremities. The affected areas convey the sensation of heat, they pulsate, sweating occasionally, and, in some cases, show a tendency to hæmorrhage and to the formation of small nodules. Not always are the symptoms of pain, redness and swelling uniformly developed; often only the pain is prominent, which may be either severe or insignificant. Prolonged walking, standing, and the influence of the heat cause the swelling as well as the pain to become more marked, so that the patients anxiously avoid walking, etc. Only the toes or the heels are affected, as a rule; more rarely the fingers or the hands and the feet at the same time. The affection is very tenacious, occurring in paroxysms, or is continuous, causing more difficulty in summer than in winter.

The diagnosis of this condition will rarely present any difficulties; the only question that may arise in the individual case is, whether erythromelalgia is to be looked upon as a substantive "angeioneurosis," or as a symptom of another disease of the nervous system, such as apoplexy, tabes, multiple sclerosis, neuritis, hysteria. It is always well to inquire accurately whether the latter conditions are present, before we conclude to make a diagnosis of an idiopathic erythromelalgia. The condition is a vaso-motor nerve affection, which, as Auerbach has lately found, may depend upon disease of the posterior roots.

ACUTE ANGEIONEUROTIC ŒDEMA, ACUTE CIRCUMSCRIBED CUTANEOUS ŒDEMA

By this condition we understand an acute, circumscribed, œdematous swelling of the skin, occurring in attacks at various parts of the body, developing without pain or itching, and rapidly, i. e., in the course of a few hours, disappearing completely. Occasionally the œdematous swelling also affects the mucous membranes of the larynx and pharynx, and, as it appears, also of the stomach and intestines, which fact is indicated by periodic vomiting, gastralgia, etc., which are combined with the cutaneous swelling; hæmorrhages into the mucous membranes and effusions into the joints occur in exceptional cases.

This affection can only be confounded with urticaria or with exudative erythema, with which it is, undoubtedly, closely allied, in that it is probably only a question how deeply the œdematous infiltration extends in the given case. Other varieties of

cutaneous œdema in affections of the kidney, heart, etc., rarely require any differentio-diagnostic discrimination, on account of the fleeting character of the angeioneurotic œdema. It must also be taken into consideration in the diagnosis that the affection in question occurs in nervous individuals, being found in the course of hysteria, exophthalmic goître, etc.

INTERMITTENT ARTICULAR DROPSY

Hydrarthrus nervosus intermittens is a very rare, stubborn affection, characterized by swelling of the joints (especially of the knee joint) owing to effusion of fluid, running its course without fever or reddening of the skin and usually also without pain, occurring in regular intervals and disappearing after a few days (from one half to one week). The same as the other forms of angeioneurosis this affection may also appear as a substantive condition or it may be a symptom of other well-known neuroses, such as hysteria, epilepsy, Graves's disease. An observation of Féré on a hysterical morphine-taker is very interesting: During the withdrawal of the drug diarrhœa and a very marked, painful swelling of the knee joints appeared daily at a certain hour, disappearing in a few moments after a hypodermic injection of morphine, without leaving a trace.

SYMMETRICAL GANGRENE, RAYNAUD'S DISEASE

appears as a *morbus sui generis* or as a part phenomenon of hysteria, tabes dorsalis, syringomyelia, etc. Emotional disturbances, especially fright, appear to favour the development of this affection which appears in paroxysms. It is ushered in with paræsthesia in the fingers or in the toes, which become waxy pale (corpse finger, regional ischæmia), pains occurring at the same time; these areas become cyanotic after a little while and, finally, of a bluish-black colour. During this stage a retardation of the phenomena may occur; in other cases, however, the characteristic symptoms of gangrene develop at symmetrical areas of the hands and feet, and that usually only at the end phalanges. Symmetrical gangrene has also been observed in the soft parts of the femurs, ears, etc. The *nervous* character of the affection is indicated by the facts that the gangrene may occur in paroxysms, that it appears symmetrically and that various nervous phenomena, disturbances of sight, hearing and taste, also signs of an affection of the sympathetic nerve, or the symptoms of a disease of the spinal cord, epilepsy, neuritis, etc., may coexist. Of course, the usual causes of gangrene, such as diabetes, must be excluded before "angeioneurotic" gangrene dare be diagnosticated.

ACROPARÆSTHESIA

occurs preferably in anæmic women, especially during the climacterium, but it is also found in tubercular individuals. The effect of preceding refrigerations and exhaustive manual labour (sewing, washing, etc.) appear to be the principal predisposing causes of the affection. Paræsthesias of all kinds (formication, numbness, itching, tingling, etc.) in the finger points, more rarely in the toes, and most marked in the morning before and after rising, increasing later and developing into marked pains, are the principal symptoms of the affection. With this there may be anæsthesia or hyperæsthesia, and stiffness in the hands, but, above all, also symptoms which point to a disturbance of innervation in the course of the sympathetic nerve: Pallor or cyanosis of the affected ends of the extremity, abnormal sweat secretion, curvature of the nails, conspicuous gloss of the skin, etc.

The diagnosis "acroparæsthesia" may be made only after apoplexy, tabes dorsalis, or other affections of the brain or spinal cord have been excluded, which, with other symptoms, give rise to paræsthesia of the extremities, such as tetany, hysteria, ergotism, etc.; and even then it must not occasion surprise if later on it is found that "acroparæsthesia," which has been diagnosticated, is only a precursor of one of the previously mentioned, severe affections of the nervous system, or represents the initial stage of acromegaly, which is now to be described.

ACROMEGALY, PACHYACRIA

Following Pierre Marie, the name acromegaly is employed to designate an affection characterized by an *abnormally increased growth of the ends of the body*, especially of the feet, hands, nose, and of the bones of the face (particularly of the inferior maxilla). The feet, on account of the giant growth, become abnormally bulky and large, the hands become enormous, "paw-like," the fingers sausage-shaped, the nails broad and flat; but, in comparison to the enormous increase of the fingers, they remain relatively small. From the wrist joint upward, the hypertrophy is no longer developed, as a rule. In the lower extremity, besides the feet, the bones of the lower leg, especially the malleoli, are hypertrophic; occasionally also the patella takes part in the enlargement. In the face the protruding areas are the seat of the hypertrophy: The nose, occasionally also the ears, and, further, the bones of the cheek, the chin with the lower lip, and the entire inferior maxilla; the tongue may reach double the size of the normal organ. The bones of the neck and the upper portion of the thoracic vertebræ take part in the hypertrophy, causing a kyphosis in the affected parts; the larynx may assume unusual dimensions, the voice may become deep and rough. The hypertrophy affects the bones, above all, however, the deeper layers of the skin; the muscles at times retain their normal strength, at other times they early become weak and flaccid. The heart is often enlarged, the circulation impeded and the tendency to engorgement is very usual. Almost constantly we observe a loss of sexual power and the *cessation of menstruation*. Sensation is intact, but widely distributed *pains* and paræsthesia are frequent at the onset and also in the later course of the disease, and, simultaneously with these attacks of pain in the extremities, a paroxysmal swelling of the same is observed, which later may become permanent; we may, therefore, assume that the extraordinary growth is due to the influence of vaso-motor disturbances. Increased secretion of sweat, polyuria, *diabetes*, and *neurasthenico-hysterical* manifestations (analgesia, etc.) appear as secondary phenomena in the morbid picture, the same as, finally, cerebral symptoms (choked disk, optic atrophy, hemianopsia, paralysis of the muscles of the eye), which may be referred to pressure of the enlarged *hypophysis* [pituitary body] in this vicinity. There can no longer be a doubt that in a very large number of cases of acromegaly alterations of the hypophysis (usually tumours of the same) appear, which most likely are in connection with the development of the affection. On the other hand, it cannot be denied that typical cases of acromegaly occur without any change of the hypophysis and, *vice versa*, tumours of the latter are noted which do not give rise to acromegaly. It is not permissible, therefore, to regard tumours of the pituitary body as the cause of acromegaly; it would be just as reasonable to assume that hyperplasia and tumour formation of the pituitary body is a symptom which is *co-ordinate* with the other phenomena of acromegaly, and that the foundation of the disease depends upon a constitutional disturbance which manifests itself especially in abnormal processes of growth.

ARTHROPATHIC HYPERTROPHY

From the form of acromegaly which has just been described, Pierre Marie has lately separated a special variety of giant growth, in which particularly the bones and the joints at various parts of the body are affected by hypertrophy. This gives rise to difficulty in active and passive movements, to arthritic pains, and œdematous swelling in the surroundings of the joints. The end phalanges of the fingers, especially, are most markedly thickened (drum-stick fingers), the nails are brittle and show longitudinal striæ, the wrist joints are markedly affected and deformed. The soft parts take part in the hypertrophy only secondarily, whereas, in acromegaly, the soft parts and bones are uniformly affected. The increase in volume in the latter malady occurs particularly in the face (nose, inferior maxilla and lips), whereas in "*osteoarthropathia hypertrophicans*" the increase in volume is absent or, if present, never affects the lower jaw. The cervical kyphosis which is characteristic of acromegaly, does not occur in osteoarthropathy; if kyphosis develops here, it affects the

lower dorsal and lumbar regions exclusively. The symptoms which point to disease of the pituitary body are also absent in osteoarthropathy. Marie believes the development of the disease to be in genetic connection with *pulmonary affections* (*osteo-arthropathie hypertrophicans "pneumique"*), and the drum-stick fingers which occur in phthisical patients and in those affected by bronchiectasis, should be included in the category of "osteoarthropathy." The latter, for the present, appears to me to be very questionable, as the implication of the joints, the friability of the nails and, especially, the further spread of the process, are absent in this thickening of the finger ends, at least according to my experience.

Acromegaly may be confounded with *elephantiasis*, in which, however, the thickening only affects the skin (especially the subcutaneous cellular tissue), not all the soft parts, and especially not the bones. More difficult is the differentiation from *arthritis deformans* especially in those cases that run their course resembling the picture of osteoarthropathy, in which the joints are especially affected by hypertrophy. In arthritis deformans there is also painfulness and difficulty in the movement of the joints, and particularly also the decided enlargement of the circumference of the osseous arthritic ends; but in arthroosteopathy various bones: the radius, tibia (also in their diaphyses), the sternum, the ribs, etc., are implicated simultaneously, and the terminal phalanges of the fingers are changed in a particularly characteristic manner. Acromegalic changes which simulate acromegaly are also observed in *syringomyelia*; however, these never reach such a marked degree of development, nor is their distribution such as in acromegaly. The hypertrophy is limited to individual parts in syringomyelia, to the toes and fingers, and, with this, the well-known characteristic symptoms are developed: Thermo-anæsthesia and analgesia, etc. From *myxœdema*, which is now to be described, acromegaly differs in that, besides the soft parts, the bones are also affected, which is never the case in myxœdema.

MYXŒDEMA

By *myxœdema* we understand an elastic, œdematous swelling of the skin, going hand in hand with cachexia and nervous disturbances, the origin of which is in connection with a *loss or an arrest of function of the thyreoid gland*. The connection of the affection with an absence of function of the thyreoid gland is beyond all doubt. We know that the thyreoid gland is absent or atrophied in myxœdema, and, moreover, that total extirpation of the thyreoid gland in animals, respectively human beings, either gives rise to tetany (see Tetany) or it produces the symptom-complex of myxœdema. It is assumed, therefore, that, on account of absence of function of the thyreoid gland, a toxic substance accumulates in the organism which has a deleterious effect on the nervous system.

The œdema of the skin differs from ordinary œdema in that the cutaneous swelling is firmly elastic and does not disappear upon pressure, affecting particularly the eyelids, the nose, the cheeks, the lips and the neck, but, besides, also the trunk and extremities. The hair falls out, secretion of sweat is diminished, the skin is dry and rough. In one of my cases there developed a desquamation of the skin, resembling ichthyosis. The difference between the myxœdematous and the ordinary œdematous consistence of the skin may be looked for in an accumulation of mucin in the subcutaneous cellular tissues in myxœdema. Thickening of the mucous membranes and interstitial inflammations of the internal organs (kidneys and liver) are also observed occasionally; the voice becomes monotonous and rough.

In connection with the cutaneous œdema a general *cachexia* and a tendency to hæmorrhages arises (*cachexia strumipriva*). The temperature of the body is usually subnormal.

The third phenomenon in the pathological picture consists in *disturbances on the part of the nervous system*: Headache, sensation of fear, hallucinations, somnolence, apathy and stupidity. The diminution of intelligence, simultaneously with the swelling of the facial skin, is the cause of the conspicuous change in the physiognomy of the patients, who, in contrast to their previous appearance, take on an expression of imbecility. Paraparesis has also been observed lately.

Chvostek's symptom of tetany, increase of the mechanical irritability of the nerves, seems to be present in some cases of myxœdema, in others, exophthalmos and enlargement of the thyreoid and a tremor resembling alcoholic tremor—symptoms which point to a connection of myxœdema with tetany, on the one hand, and with *Graves's disease*, on the other. The latter disease occasionally changes into myxœdema or may become associated with it.

GRAVES'S DISEASE (BASEDOW'S DISEASE, GOITRE EXOPHTHALMIQUE)

The diagnosis of *Graves's disease* is based primarily upon the determination of the three cardinal symptoms of the affection—*increased and accelerated action of the heart, exophthalmos and goître.*

Acceleration of the pulse is the most constant manifestation of this trio of symptoms, and usually that which appears earliest. The number of pulse beats amounts to 80 to 120, in rare cases more, up to 200, and varying at different times. The *impulse of the heart* is *intensified,* diffuse; the carotid arteries are distended and pulsate markedly, also, sometimes, the arteries of the head; the pronounced pulsation of the arteries is then perceived by the patient as a troublesome hammering in the head. The arteries of other regions of the body, thus those of the abdomen and thigh, are sometimes also found markedly distended and throbbing. The veins of various regions of the body, especially of the neck, are also distended, and an exquisite venous pulse has often been observed in the jugular veins. The increased action of the heart is associated with a *tormenting palpitation of the heart*; the latter occurs sometimes in attacks which are accompanied with sensations of fear and which may present the clinical picture of a true angina pectoris. If the intensified action of the heart has persisted for some time, a diffusion of cardiac dulness manifests itself—as the expression of consecutive (left-sided) hypertrophy of the heart (which, as occurred in one of my cases, may be demonstrable post mortem), or of a dilatation of the heart, fatigued by overexertion. In such cases we hear, upon auscultation, loud systolic murmurs which are usually of an accidental nature, but often they may be due to complicating valvular defects.

Goître.—The second symptom, which usually associates itself later, sometimes not until months have elapsed, is the *enlargement of the thyreoid gland,* which, however, need not be very markedly developed and may be entirely absent in rare cases. *The consistence of the gland is soft, elastic;* this is the rule, at least; but it seems that symptoms of Graves's disease may be observed in all forms of goître. At any rate, the fact that a hard goître is present in doubtful cases, should be no reason to reject a diagnosis of Graves's disease if the other symptoms of the disease are present. The goître of Graves's disease does not usually exert pressure upon the adjacent regions, because, aside from exceptional cases, it attains only a moderate size and remains soft. It is characteristic of the goître, furthermore, *that the vessels of the thyreoid glands expand* and, besides, appear tortuous and pulsate; upon palpation of the vessels we often feel a thrill, to which loud, especially systolic, vascular murmurs correspond on auscultation.

Exophthalmos.—The changes in the eyes, which are so important in the diagnosis of Graves's disease, usually develop latest. The most remarkable of these is *exophthalmos,* which develops, as a rule, synchronously in *both* eyes; the degree of development is sometimes different in both eyes. It occurs occasionally that exophthalmos appears first on one side and does not become bilateral until later; rarely it remains unilateral. The peculiar physiognomic expression of the patients is caused, besides by the exophthalmos, by the wide opening of the palpebral fissure and also by the *insufficiency and rarity of the involuntary winking of the eye* (so-called Stellwag's sign). *The failure of the upper lids to move downward when looking downward,* known as Gräfe's sign, was noticed very early in the majority of cases. This symptom, independently from the exophthalmos and the degree of the latter, often precedes other signs of Graves's disease, and, on the other hand, it may disappear in the course of the affection. In markedly developed exophthalmos, owing to the insufficient covering of the cornea by the lids, a drying of the corneal epithelium occurs sometimes, with loss of the same, also secondary infection, with ulceration of the cornea and destruction of the entire eye.

The movements of the eyes are little or not at all impaired, as a rule; often there exists an *inability to keep the eyeballs converged* (Möbius's sign), so that, upon looking at a nearby object, one eye deviates. The other functions, as acuity of vision, accommodation, width and reaction of the pupils, etc., are undisturbed; mydriasis especially cannot be determined. Ophthalmoscopical examination has sometimes revealed a pulsation of the arterial vessels of the retina.

The above-named three cardinal symptoms usually develop gradually, i. e., in the course of several months, but sometimes very acutely in a few days. It is not necessary, as will be noted from the preceding description, to find them all combined, to establish a diagnosis of Graves's disease; exophthalmos especially is absent comparatively often, goître rarely.

Minor Symptoms.—We observe in the different cases, besides the three principal symptoms, a number of more or less prominent *nervous manifestations* of a general character: Vertigo, headache, increased irritability and restlessness, insomnia, hysteric symptoms (e. g., astasia and abasia) and actual psychical disturbances (delirium, hallucinations, insanity, etc.). Two of these nervous symptoms have been especially noted recently. Their occurrence is considered so specific of Graves's disease that some authors even place them in line, regarding their diagnostic importance, with the three cardinal manifestations. They are the *tremor* and the *diminution of the resistance of the skin to electric conduction.*

Diminished Resistance to Conduction; Tremor.—Regarding the *diminution of the resistance to galvanic conduction,* careful researches of various investigators have proved that it is neither a question here of a constant manifestation, nor of one which is only peculiar to Basedow's disease, but that the reduction of the resistance to electric conduction, which, it is true, is frequently determined in these patients, should probably be ascribed only to a more marked inclination of the patients to perspire, and to the condition of the skin changed thereby. The *tremor* in exophthalmic goître, however, appears to be a symptom based upon the character of the affection itself. It is characteristic that the oscillations follow each other very rap-

idly (8 to 9 per second), similar to the condition in alcoholic tremor, but decidedly in a much more rapid manner than in senile tremor, in paralysis agitans and in multiple sclerosis, and, furthermore, that the fingers do not tremble isolatedly in tremor of the hands, but are put in motion by the trembling wrist. A trembling of the eyeballs and of the upper eyelids has also been described. There is no question that tremor in Graves's disease is a frequent, although by no means constant, manifestation, and it remains the merit of Charcot and of P. Marie to have called attention to this phenomenon.

Changes of the Skin.—*Alterations of the condition of the skin* should be especially mentioned among the other minor manifestations which are observed in the clinical picture of Graves's disease. Of usual occurrence is the *sensation of heat* which molests the patients (the temperature of the body is almost always normal, but sometimes there occur, according to my experience and that of others, rises of temperature from 1 to 2 and more degrees F.). The skin is frequently flushed, and is very apt to change its colour, or it reacts to slight irritations with a long-lasting hyperæmia or formation of urticaria (urticaria factitia, dermographism). Very marked accumulation of pigment is often found in the skin, sometimes only in those places which are normally rich in pigment, as in the nipples, in the axillary cavities, etc., at other times in unusual places; sometimes almost the entire skin is brown, as in Addison's disease. Less often than abundance of pigment we observe in Graves's disease loss of pigment, i. e., patches of vitiligo which occur sometimes over the entire body, at other times only in the neck, face, etc., and, furthermore, loss and whitening of the hair. *Sclerema* of the skin has often been observed by me and by others, also erythema, œdema and gangrene of the lower extremities. Characteristic of the disease is also the inclination to profuse perspiration, *hyperidrosis;* circumscribed and semilateral perspiration was also seen in several instances.

Other anomalies of secretion occur also: Salivation, profuse lachrymation, and polyuria; it is interesting, theoretically, that sometimes sugar was demonstrated in the urine and, also, alimentary glycosuria. Occasionally it occurs that a pronounced inclination to hæmorrhages manifests itself in exophthalmic goître.

The digestive organs are affected, above all, by paroxysmal, profuse *diarrhœas,* which are accompanied with colicky pains, and, besides, with vomiting, bulimia and polydipsia.

My patients complained in several instances of "rheumatic" pains, especially in the joints; "intermittent articular dropsy" was also observed in the course of Basedow's disease.

The alteration of the metabolism, which was lately demonstrated with certainty as occurring in Graves's disease, is highly interesting. It was noted for quite a long time that patients afflicted with this malady may emaciate alarmingly rapidly, in spite of overabundant administration of food; an inclination prevailed, therefore, *a priori* to regard an increase of the processes of decomposition as the cause of the emaciation. However, a more thorough study of the effects of the thyreoid gland upon metabolism was not brought about until it was learned that adiposis can

be successfully combated by the administration of thyreoid-gland preparations. Magnus-Levy, to whom primarily we are indebted for the positive demonstration of the extraordinary increase of metabolism by thyreoid preparations, found, upon administration of thyreoid tablets, an increase of oxygen consumption to twice the normal amount, and he determined, furthermore, that *a very material increase of oxygen consumption takes place in patients with exophthalmic goître* (5.0 to 6.5 c.c. of oxygen against 3.5 to 4.5 per kilo body weight and minute in healthy individuals). Accordingly, it is very probable that in Graves's disease the affected thyreoid gland exerts a considerable influence upon metabolism in that it increases the same, and that there exist substances, formed in the body itself, which, when entering the blood, are able chemically to provoke a far-reaching alteration of the metabolism.

Upon longer duration of the affection gradually the severest form of cachexia develops, to which the patient succumbs if the fatal termination has not been brought about previously by intercurrent diseases (especially pneumonia and tuberculosis or cardiac inefficiency).

In some cases symptoms occur which point more directly to an affection of the central nervous system: Hemiplegias and paraplegias, pareses of the facial nerve, nuclear paralysis of the muscles of the eye, partial or diffused muscular atrophies, spasms in the extremities, exquisite hysteria, epilepsy.

Origin of the Disease.—It would be of decided importance for a precise definition of the diagnosis, especially of the interlocking of the individual manifestation of Graves's disease, and for the separation of the accidental symptoms which are founded upon the nature of the affection, if we would know the cause of the same.

Its pathogenesis, however, was entirely obscure until recently, and, even now, I do not consider the doctrine of the nature of Graves's disease to be by any means clear and final. It is well known that the assumption of an *affection* of the *cervical sympathetic nerve*, which was formerly almost generally considered to be the cause of exophthalmic goître, does not satisfactorily explain even the occurrence of the three cardinal symptoms, in that, unless we proceed very artificially in the diagnosis, we must either assume for the different fibres of the sympathetic nerve paralysis (consequence: Dilatation of the vessels—development of goître), or permanent irritation (consequence: Tachycardia, palpitation of the heart, and, to some extent, exophthalmos), or functional integrity (of the fibres which cause dilatation of the pupils). I believe it would be more natural, therefore, under all circumstances to connect the origin of Graves's disease with an affection of the *central* apparatus of the *medulla oblongata* which preside over those nervous functions, which was already done by Geigel, Senior, and others. Filehne has furnished an experimental support to this supposition in that he succeeded, by injuring the restiform bodies (the transitory station of the respective nerve tracts) in the rabbit, to produce the three cardinal symptoms artificially (although not always all of them at the same time). Moreover, an affection of the medulla oblongata, as the original cause of Graves's disease, is favoured by clinical experience, thus the nuclear paralyses which were positively demonstrated in the course of the disease, the spasms, the paralyses and atrophies of the musculature, melituria, etc., in one of my cases of exquisite Graves's disease there existed, besides spasms in all four extremities, slight disturbances of the hypoglossal nerve and dysphagia. However, the autopsies performed on the bodies of patients who succumbed to Graves's disease have as yet given very few positive findings regarding anatomically demonstrable changes of the medulla oblongata: and, on the other hand, the absence of symptoms of Graves's disease in the various affections of the medulla oblongata constitutes the rule. If, therefore,

we are not satisfied with the assumption of a *functional* bulbar disturbance in the conception of a neurosis, similar to chorea, epilepsy, etc., we must admit that the theory of the oblongata being the causative factor is by no means satisfactory.

The majority of physicians were right, therefore, when they abandoned it and adopted the *thyreoid theory*, which was first established by Moebius. According to the latter, "*Graves's disease*" *is an intoxication of the body by a morbid activity of the thyreoid gland.*" There can be no doubt that this assumption has many points in its favour, and it is well adapted plausibly to explain many obscure points in the clinical picture of exophthalmic goître. The alteration of metabolism upon abundant administration of thyreoid preparations, and in patients affected with Graves's disease upon adopting the same plan, is established, i. e., there occurs an increase of metabolism, especially of the consumption of oxygen, whereas, conversely, a diminished decomposition of matter can be determined in myxœdema. This renders it quite obvious that patients with Graves's disease emaciate and, on the other hand, patients with myxœdema gain in weight, and, also, that a reduction of albumin disintegration up to 25 per cent of the entire transformation of nitrogen occurred in patients with Graves's disease after strumectomy, to be replaced by an increase of N-excretion when the excised thyreoid gland was administered after strumectomy (Matthes). It is logical, accordingly, to assume that *hyperthyreoidism* is present in Graves's disease and hypothyreoidism in myxœdema, all the more so because there are other contrasting differences between these two affections: In the former, enlargement of the thyreoid gland, tachycardia, psychical irritability, hyperidrosis, increase of the temperature of the body—in the latter (myxœdema), atrophy of the thyreoid gland, slowing of the pulse, indolence and actual imbecility, deficient perspiration, subnormal temperature. However, if we consider that the administration of thyreoid-gland preparations never produces all, in fact not even the cardinal, symptoms of Graves's disease, and that the anatomical changes in the thyreoid gland in the latter affection render hyperplasia of the tissues probable only at the onset and later a qualitatively altered activity of the gland, the assumption of a simple (quantitative) hyperfunction of the thyreoid gland as the cause of the symptom-complex of Graves's disease cannot be upheld. Therefore, we should think of a toxine which is developed by a qualitative activity of the gland and which, introduced into the blood from the thyreoid gland, would cause an intoxication, especially of the nervous system. However, so long as we have no positive knowledge regarding the physiological significance of the activity of the thyreoid gland, and are not able experimentally to obtain any insight into the action of the toxine of Graves's disease, nothing remains but, for the present, to abandon a positive view regarding the occurrence of exophthalmic goître.

Differential Diagnosis.—The diagnosis of exophthalmic goître does not present any difficulties if the three cardinal symptoms of the affection and the tremor are present at the same time. If only two of the symptoms are developed, however, or if only one can be demonstrated during the initial stage of development of an exophthalmic goître, the diagnosis of the latter can, eventually, not be made at all or, as a rule at least, not with certainty.

Usually, if only one symptom is present, it is a question of *tachycardia*, as this phenomenon usually represents the first morbid symptom of incipient Graves's disease. But the moderate acceleration of the activity of the heart is such a frequent symptom, depending upon various circumstances, primarily upon anæmia, that the presence of this manifestation alone cannot be utilized in the diagnosis. If, besides, tachycardia and palpitation of the heart occur only in paroxysms, with long-lasting intervals of normal activity of the heart, it would point against incipient exophthalmic goître.

It is necessary also, even if an *acceleration of the pulse and goître* are present at the same time, to be careful in establishing the diagnosis of Basedow's disease. For the development of the goître is so often observed in anæmic young girls, that the combination of the symptoms goître and acceleration of the pulse (the latter of which, as is well known, is a very common phenomenon of anæmia) occurs frequently enough without an exophthalmic goître being present. However, the presence of the latter is probable if acceleration of the pulse is very considerable (100 and above) and if it develops, synchronously with the goître, within a few days, as it does now and then, if the goître is soft and if the vessels of the thyreoid gland in such a case appear distended and tortuous.

The diagnosis becomes certain if tachycardia or goître and tachycardia are supervened by *changes in the eyes.* Graefe's symptom (see p. 760) especially is of great diagnostic significance, because it always appears early, in fact may precede all other symptoms of Graves's disease. If exophthalmos is well developed, it serves to furnish a firm support to the diagnosis. But we must always bear in mind that a primary goître of a different origin, owing to pressure from the adjacent cervical nerve trunks, in rare cases may produce a clinical picture that simulates the syndrome of Graves's disease. But the exophthalmos in such cases is developed very slightly or not at all, whereas a *change in the width of the pupils* (which is *absent* in Graves's disease) can be determined besides the acceleration of the pulse; furthermore, the above-named subsequent symptoms of pressure of the goître upon the adjacent regions are mostly developed on *that* side only, which corresponds to the greatest development of the goître.

The various other symptoms of Graves's disease: *Tremor, profuse secretion of perspiration,* emaciation, alterations of the skin, diarrhœa, febrile temperature of the body, etc., may contribute to render the diagnosis more certain, but to the careful diagnostician they will never be anything else but supplementary diagnostic factors.

DIAGNOSIS OF DISEASES OF THE MUSCLES

FOLLOWING the diseases of the nervous system, we wish to discuss the *affections of the muscles.* One of them, *progressive muscular dystrophy,* owing to its close diagnostic relation to spinal progressive muscular atrophy, was enlarged upon when treating the diagnosis of affections of the spinal cord (which see). Of the remaining affections of the muscles, two are of great practical significance, so-called *muscular rheumatism,* and *acute multiple myositis.*

ACUTE AND CHRONIC MUSCULAR RHEUMATISM

Clinical Picture.—This affection, which is extremely common, manifests itself in violent pains in the affected muscles when they are actively or passively moved, or when external pressure is exerted upon them. The pains increase very considerably if the affected muscles are extended passively or contracted markedly by means of the faradaic current, or if a portion of them is grasped between the fingers and squeezed slightly. According to my experience, the disease may begin with pain in the tendons, and the process may extend to the substance of the muscles; mostly only a single muscle is affected, sometimes an entire muscle group.

The function of the muscle is very much impaired or entirely suspended. To avoid an increase of the pains, any movement of the affected part of the body is carefully avoided, which causes the patients, according to the seat of the muscular rheumatism, to assume an attitude of the body which, as a rule, is very characteristic. Thus, the head is held in an oblique position in unilateral rheumatism of the cervical muscles (*rheumatic torticollis*); in rheumatism of the lumbar musculature, especially of the quadratus lumborum (*lumbago*), the trunk is held in a rigid position. Respiration, sneezing, coughing, etc., are painful in rheumatism of the *muscles of the chest,* so that the patient respires only superficially and suppresses these reflex movements as much as possible. It seems that occasionally the larynx is also affected by rheumatism, causing hoarseness, immobility of the vocal cords and disturbances of phonation.

Whereas the diagnosis generally does not present any difficulties if we consider the above-named characteristics of the affection, it is sometimes possible that neuralgia, or, in rheumatism of the thoracic muscles, pleuritis sicca are to be considered in the differential diagnosis. The pain in the muscles upon compression of the same between the fingers, the disappearance, or at least the temporary decrease, of the pains after faradization and the marked increase of the same upon certain movements which contract or expand the muscle, guard against confusion of the affection with *pleurisy,* even if no dulness or, temporarily, no friction sound can be demonstrated in the latter. The presence of *intercostal neuralgia* is indicated in a doubtful case in that the diffusion of the pain is strictly limited to the dissemination of an intercostal nerve, that quite circumscribed areas of the same are painful upon touching the intercostal space, and, furthermore, that the pains, although they

become more violent upon more extensive respiratory movements, etc., may increase also in the intervals, without any apparent cause, to actual paroxysms of pain. Also the fact, although this does not occur often, that the pain in intercostal neuralgia is diminished by strong pressure (in contradistinction to rheumatic affection of the muscle) is directly against the presence of muscular rheumatism in a given case.

Nature of the Affection.—We might suppose that a disease which is as frequent as muscular rheumatism ought to be well known regarding its character and that thus the diagnosis would gain in certainty. But this is not the case as yet; however, observations which I have recently made in more than 100 cases, make it appear probable that the disease, at least in the majority of cases, is of an *infectious* nature. This view is supported by a number of reasons: The increase of the number of cases of muscular rheumatism at certain times, the onset of the disease with general manifestations which may precede the muscular pains for some time, the flightiness and the diffusion of the latter over several muscles, the fever (in one third of the cases), and the involvement of internal organs, especially of the endocardium, in the morbid process. The transition of muscular rheumatism to acute articular rheumatism is quite common, so that, possibly, the infectious noxa which causes muscular rheumatism represents only the attenuated virus of articular rheumatism, at any rate is closely related to the latter.

Chronic Muscular Rheumatism.—The pathological picture of *chronic muscular rheumatism* is much less precise. The pains, which generally cause the diagnosis of chronic muscular rheumatism to be made, are weaker, more migratory, and depend mostly upon changes of the weather. However, this diagnosis mostly lacks a firm support unless the connection with muscular rheumatism of originally acute onset is undoubted; it is usually a meaningless diagnosis of embarrassment which gives a name to pains which cannot be defined more precisely.

ACUTE MULTIPLE MYOSITIS

This affection has been repeatedly described since the reports of E. Wagner, Hepp and Unverricht, in 1887, of a disease which until then was almost unknown, viz., *acute polymyositis,* so that there cannot be any question as to its existence. I have personally observed, recently, four cases of polymyositis which were ætiologically connected with each other and which pointed to the infectious character of the disease.

Pathological Picture.—Before entering upon the diagnosis it is advisable minutely to discuss the pathological picture according to the reports existing so far, as the disease is very little known as yet.

It commences usually, sometimes suddenly at other times gradually, with *certain general symptoms*: Headache, sensation of weakness, nausea, chills (in one of my cases), pains upon urination, eruptions, which were quite varying in several cases, and consisted in erythemata, roseola, urticaria, herpes or, as occurred in two of my cases, psoriasiform exanthem with marked desquamation of the epidermis. An enlargement of the spleen was also demonstrated several times in cases with an acute course. *Fever* was always present, of medium intensity with corresponding acceleration of the pulse; intercurrent chills occurred in two of my cases. The affection obtains its peculiar character from the *local symptoms,* which take place either at once with the general symptoms or a little later. They are painful sensations in the extremities and in the trunk, in part of a spasmodic nature, which greatly impede the movements of the patients. *The affected muscles are exceedingly sensitive to pressure, especially at the insertions of the muscles into the tendons,* as in the case of Plehn and in my own cases;

besides, consistent infiltration of the muscles can be demonstrated unless a concurrent *œdema of the skin* renders palpation impossible. The affection does not take place at the same time in all the muscles which are affected by the disease, but it extends, sometimes more rapidly, at other times more slowly, from muscle to muscle. *The œdema of the skin* which is associated with polymyositis and which is accompanied with transitory redness of the skin, is particularly characteristic. The *joints* may also be inflammatorily infected. In two of my cases, in which the muscles of the femur were very markedly attacked, a *thrombus of the femoral vein* developed which had not led to softening but to consolidation of the thrombus, as was proven by a case that came to autopsy. Generally the disease terminates fatally when the phenomena are more pronounced; however, according to my experience, it occurs that the disease, even if it is very severe, i. e., if it is accompanied with diffusion of the inflammation over almost all muscles, with venous thrombosis, articular inflammations and chills, finally terminates in recovery after months.

If myositis extends upon *the muscles of the pharynx and those of respiration*, dangerous functional disturbances may result, such as dysphagia and respiratory insufficiency; the affection of the *musculature of the tongue* may greatly impair speech, that of the eye muscles may affect the bulbi. With a long duration of the disease the swellings recede, and a pronounced atrophy of the affected muscles develops. More marked *sensory disturbances and painfulness to pressure of the nerve trunks are absent.* The electric irritability of the muscles was found diminished in cases in which it could be tested, the sensorium was always unaffected in my patients as well as in those of others. A great inclination *to perspire* was frequently observed.

The course of that case in my observation which terminated fatally may be briefly described, owing to the rarity of the disease.

Case of Subacute Polymyositis.—The patient, E. M., twenty-two years old, was admitted to the clinic on June 8, 1892, and died on July 30, 1892.

History.—Both parents died of dropsy. Two children alive and healthy. The patient had pneumonia when nineteen years old, but was otherwise healthy until June 3, when she became sick with *sudden chills, nausea, headache and abdominal pains*, which were especially marked during walking, standing and urinating; the stools were regular. The patient was confined to her bed on June 5, when the abdominal pains diminished. On June 5 the *face* commenced to swell; the skin was tense and after June 7 covered with *red patches.* Owing to the increasing debility, *pains in the limbs*, in the sacrum and abdomen, the patient came to the Julius Hospital; she complained also of loss of appetite, increased thirst, slight vertigo and headache. Her condition on June 8, on admission to the hospital, was as follows: Robust body and healthy complexion. Numerous irregular *reddish-brown patches*, from the size of a lentil to that of a dime, which were partly confluent, projected slightly over the level of the skin and became pale upon pressure, were present on the face; similar sparse patches were also found on the skin of the shoulders, chest and upper arms, later also upon the hands. The eruption was non-irritating. Œdema and jaundice were not present, but there were glandular swellings along the trapezius muscle of the right side.

The lungs did not show dulness or *râles*; slight laryngitis was present, the heart normal, the pulse regular and slow. Spleen and liver were not enlarged and could not be palpated; *pressure upon the left lumbar region* caused pain.

The diagnosis was *laryngitis, bronchitis* and *lumbago.* During the following days the patient complained of pains at the end of urination, painfulness upon pressure upon the *bladder region,* marked leucorrhœa. Urine which was voided after preceding irrigation of the vagina was cloudy, although not decomposed, it was acid and contained, according to the microscopical examination, numerous pus corpuscles and very motile bacilli, so that the presence of a *cystitis* was certain. On June 26 a more or less continuous fever of 102.2° F. to 104° F. commenced, which lasted until the patient succumbed, whereas previously only occasional rises of temperature to 101.3° F. were present, but otherwise no fever; on some days the fever rose to even 105.8° F. and above, and the pulse frequency was from 120 to 140. Bronchitic râles were present with normal percussion of the lungs. Repeated examinations of the sputum did not show any tubercle bacilli. On June 29 the patient complained of spasmodic sensations in the chest and abdomen, which impeded respiration. On June 29 she experienced *drawing pains when standing on the leg and upon sitting, besides the lumbar pains which were previously present.* The abdominal wall was sensitive to pressure. This was supervened, on June 30, *by extremely violent pains in the muscles of the calf and in those of the left side of the abdomen upon pressure.* The *bronchitis* was more developed, the spleen not enlarged. Two attacks of dyspnœa occurred during the day, with gasping, accelerated respiration; cyanosis was absent.

The swelling of the left calf was undoubtedly pronounced on July 1 and 2; the contours of the ankle were obliterated. *The pain was restricted exactly to the musculature of the calf.* The skin covering the same, and the joints were not sensitive to pain; the right lower leg was painless and slender, three centimetres thinner than the left one. *The tendon insertions in the region of the popliteal space* were especially painful, the skin covering the same could be folded without causing pain, the *adductors* of the femur were *painful,* the quadriceps muscle was not. *Pressure upon the different nerve trunks caused no pain,* whereas percussion of the tibia did, however only in the gastrocnemius muscle and not in the areas upon which the percussion was performed. The sensation of prickling was felt in the left arm, but disturbances of motility and sensibility were entirely absent.

The region of the left crural vein was very sensitive on July 5, and *thromboses could be distinctly demonstrated*; an œdematous swelling of the entire lower left extremity was present. The eruption appeared also upon the hands on this day, but it was not irritative. *On July 7 the abdominal rectus muscles were sensitive on both sides,* also the abdominal oblique muscle of the left side. The skin covering these muscles was not sensitive to pain. Marked bronchial catarrh was present, but the patient always enjoyed a good appetite. On July 12 *pains occurred in the right leg,* especially in the right calf upon pressure; vomiting had occurred for several days previously. A chill manifested itself on this day, which was repeated on July 13. The urine did not contain any albumin for several days. During the week preceding July 25 dyspeptic manifestations predominated, and the tonic condition of the patient deteriorated considerably. The pains in the right leg increased continually at the same time and an œdematous swelling of the same became apparent. The gastrocnemius muscle was especially sensitive to pain, but also the skin covering the same; the abdominal muscles were no longer painful. The eruption had assumed the appearance of psoriasis. On July 27 a dulness developed in the left lower lobe followed by rapid collapse. Patient succumbed on July 30. The autopsy, which was performed, fifteen hours post mortem, by Professor Rindfleisch, showed: *Left-sided sero-fibrinous pleurisy, serous mediastinitis, almost complete compression of the left lung, circumscribed hyperæmia with incipient pneumonia of the right lung, fatty infiltration of the liver, parenchymatous myositis; venous thrombosis, commencing in the saphenous vein and extending into the vena cava; the thrombotic masses were firm, not softened. The mucous membrane of the urinary bladder was quite markedly injected; spleen and kidneys were normal.*

The passage of the autopsy report which refers to myositis and to the thrombosis, reads as follows: "The *inferior vena cava* contains a coagulum which is coarse externally, but lacunarily reduced internally; the thrombosis extends to the bifurcation. The internal iliac vein contains an old, softened coagulum which continues into the hypogastric vein. The saphenous vein is filled with a moderately old coagulum;

the thrombus of the crural vein is red in the centre, surrounded externally with a dark covering. The *adductor magnus muscle* is discoloured to a whitish-gray in its anterior upper circumference, in contrast to the more normal colour of its lower part. Distinct œdema of the muscle is present, and water wells from its cut surface; the *rectus abdominis* muscle is discoloured to a pale gray. The subcutaneous connective tissue of the *region of the knee* is markedly œdematous; the muscles do not appear quite as gray and as pale as the adductors. The upper muscular layer of the *gastrocnemius muscle* shows atrophy and œdema; the flesh of the muscles is friable to the touch and the fasciæ can be easily recognised, *but no pus is present*; the parenchyma shows a remarkable aptness to tear. The microscopical examination of the gastrocnemius shows traces of fatty degeneration in the fresh preparation; parenchymatous myositis is noted in the left adductor, but no change in the right one; the pectoral muscles and those of the scalp and of the eyes, the musculature of the tongue and the muscles of the neck are also free from change."

The examination, by von Rindfleisch, of the muscles and nerves placed in Müller's solution showed the following result after four months: "The *muscles* contain, besides fully normal bundles, such others as are formed by 10 to 20 muscle fibres, in which the latter are altered in a varying degree of intensity. *First degree*: Brittleness of the contractile substance, inclination to transverse disintegration into short pieces which appear to be almost as long as they are broad; besides, very accentuated striation. *Second degree*: Granular cloudiness up to disappearance of striation and gradual decrease of thickness; besides, proliferation of the sarcolemmic nuclei. *Third degree*: Total disappearance of the contractile substance, leaving a sarcolemmic tube rich in nuclei."

As to the *nerves* that were found in the region of the affected musculature, they remained *fully intact* on microscopical examination. Small trunks of 3–10 primitive fibres could either be followed for a long distance, or they were found on transverse and oblique cut sections. Weigert's solution allowed the medullary sheaths to stand out in the usual manner, and it was not possible, especially, to demonstrate an increase of the nuclei and cells and of the neurilemma in hæmatoxylin preparations. The axis cylinders could be well seen, they stained and were absent nowhere.

Differential Diagnosis—Trichinosis—The diagnosis of this affection is not difficult considering the, upon the whole, well-developed clinical picture and the unquestionable concentration of the disease upon the muscles, provided certain maladies can be excluded, the course of which is accompanied with similar symptoms. The same as it occurred to me, the question will have suggested itself to others whether *trichinosis* might not be present. The *differentiation between both affections simply from the symptom-complex is actually impossible.* Only a consideration of the ætiology and the microscopical examination of the fæces or of an excised muscular particle will decide with certainty which of the two diseases should be diagnosticated. It is true, some phenomena in the pathological picture from the beginning point more to the presence of trichinosis than of polymyositis, thus the preponderance of digestive disturbances (vomiting and diarrhœa) and the concentration of the œdema upon the face and eyelids at the onset of the disease. But, otherwise, the local manifestations in the muscles, the swelling and the hard infiltration, the painfulness to pressure, the cutaneous œdema, the dysphagia, the dyspnœic attacks, the bronchitis and its consequences, and, in fact, also the accompanying symptoms, such as perspiration, fever and eruptions, are the same in both affections, so that polymyositis has been justly designated as "pseudotrichinosis" (for particulars see Trichinosis).

Polyneuritis is another disease the symptoms of which are very closely related to polymyositis. Common to both are pains and, eventually, paræsthesias, secondary atrophy of the muscles, and perspirations; cutaneous œdema and fever also occur in multiple neuritis. However, disturbances of sensibility and paralyses (with the reaction of degeneration) and also the painfulness of the nerve trunks predominate in polyneuritis, whereas in myositis the nerve trunks are not sensitive even to pressure and the exquisite painfulness on pressure is localized exclusively upon the muscles, especially upon the points of insertion of the tendons, and the process in progression affects one muscle after the other.

Other Forms of Myositis.—Another form of inflammation of the muscles, *purulent myositis*, as it occurs in the train of phlegmons and suppurations of bones and joints, is distinguished from polymyositis in that it is restricted to a relatively small area and does not, as the latter affection, show a decided tendency to dissemination over many muscles, in fact usually it covers the entire muscular system. *Myositis following infectious diseases,* which is generally rare, such as myositis in the course of enteric fever, scarlet fever, and cryptogenetic septicopyæmia, which consists either in serous or in purulent infiltrations of the muscles, is sometimes to be considered in the differential diagnosis; but the differentiation never causes any real difficulties if we consider the preceding or still existing special infectious disease. This applies also to *syphilitic myositis,* which, at least in its diffuse form, might be confused with polymyositis in that it is also accompanied with pains and may affect several muscles. The fact, however, that this affection apparently leads preferably to contracture and that, besides, pronounced manifestations of severe syphilis are present, will soon guide the diagnosis in the right direction. Moreover, a mistake is absolutely impossible as soon as distinct gummata develop in the muscles.

MYOSITIS OSSIFICANS

Myositis ossificans is an extremely rare affection in which the greatest part of the muscles of the body slowly but progressively become subject to calcification and ossification; it is absolutely impossible to mistake the same, at least not after calcification has once taken place in the muscle. During the first days of the various attacks, in which it is a question at first of an *interstitial* inflammation of the muscles and of a distention of the affected portion of the muscles, which is of doughy consistence to the touch, it may be possible, it is true, that the affection can be confused with incipient polymyositis, because during this period of development of myositis ossificans, the same as in the other disease, painfulness of the affected muscles, cutaneous œdema and fever are present. However, these manifestations are of a very transitory nature in myositis ossificans; very soon a nucleus with usually serrated periphery and gradually increasing in circumference and hardness, develops in the affected muscle. Later this nucleus becomes coalescent with the adjacent bone, divests the muscle of its contractility and presents itself, upon palpation, distinctly as a bone mass. The patients at the same time become entirely stiff, lose the ability of any active movement in the region of those portions of the body that are affected by the morbid process and, if the musculature of the thorax is affected, they have the sensation of being constricted by a coat of mail which impedes respiration. According to what I have personally seen of well-developed myositis ossificans, I do not consider it possible that it can be confused with any other disease.

SUNSTROKE AND HEAT EXHAUSTION

Sunstroke.—Sunstroke is an affection due to exposure, during the hot season, to the sun, combined with high humidity. The clinical phenomena are prostration and high fever, rapidly followed by coma. The affection is more frequent in the male sex. Alcoholism is one of the predisposing causes; aged persons are more liable to be affected; it is common in the case of soldiers on the march. Workmen engaged in their occupations, being exposed to the direct rays of the sun, are often attacked. The white race is particularly liable to this affection.

Pathology.—Granular degeneration of the ganglion cells of the brain and spinal cord has been noted. The brain and its membranes, the lungs, and the abdominal viscera often present marked degeneration. Some of the internal organs may show parenchymatous degeneration, particularly the liver and kidneys. The vena cava and right auricle are sometimes found distended with blood that is partially coagulated, being dark-red in colour. Blood concentration gives rise to polycythæmia. Leucocytosis is often noted.

Symptoms.—The attack may be preceded by prodromes, or it may begin suddenly. The prodromes consist in restlessness, insomnia, dyspnœa, nausea and vomiting, thirst, and some rise in temperature. These phenomena are gradually aggravated, the temperature reaching extreme grades, sometimes to 115° F. and even higher. The body then becomes red, perhaps livid, dyspnœa being pronounced. The pulse is full and bounding, the pupils are contracted. As complete coma occurs, the Cheyne-Stokes type of respiration is often present. Convulsions take place and just before death relaxation of the sphincters is noted. Even with extreme hyperpyrexia, recovery may occur. One attack predisposes to successive attacks upon exposure.

The prognosis is unfavourable in alcoholics and in individuals suffering from chronic affections of the heart, lungs and kidneys.

Heat Exhaustion.—Heat exhaustion is due to exposure to great heat. It is characterized by prostration, with normal or subnormal temperature. It occurs in the working classes, particularly in stokers, bakers, foundrymen, etc.

Symptoms.—As a rule, prodromes are present. They consist in vertigo, headache, tingling and irregular pains in the extremities, often nausea and vomiting. Headache becomes marked as the prostration increases. The pulse is rapid, from 120 to 140. The temperature is afebrile, frequently subnormal. Recovery is the rule.

Difficulties in diagnosis can scarcely arise. In sunstroke there is always the exposure to the rays of the sun. However, in all cases apoplexy and uræmia must be carefully excluded.]

DISEASES OF THE BLOOD AND OF METABOLISM—CONSTITUTIONAL DISEASES

I. DISEASES OF THE BLOOD

ANATOMICO-PHYSIOLOGICAL INTRODUCTION

THE blood, according to its arterial or venous constituents, represents a bright or dark red, opaque fluid mass, consisting of a liquid part, the *plasma*, and the *morphotic elements* suspended in it, the red and white blood corpuscles and the blood plaques; the colouring matter to which the red appearance of the blood is due is the *hæmoglobin*.

The *specific gravity* of the blood varies in wide limits, from 10.46 to 10.66, the average being 10.55. The height of the specific gravity is primarily dependent upon the red colouring matter, and for this reason it diminishes in diseases in which there is a marked diminution of the hæmoglobin, in conditions such as chlorosis, to 10.30 and below. The *degree of alkalinity* of the blood under normal circumstances averages that of a 3-per-cent soda solution. The entire quantity of blood amounts to about one thirteenth of the entire body weight, therefore in a medium weight of 55 to 70 kilogrammes, to about 5 litres.

The *blood plasma* contains, besides 90 per cent of water, the following solid constituents: *Serum albumin*, *globulin* (each about 4 per cent), and *fibrinogen* (about 0.2 per cent), from which fibrin is formed on clotting by the action of the *fibrin ferment* which arises from decomposition of the white blood cells after the blood has left the vessels. The contents of the blood of fats and carbohydrates (0.1 to 0.2 per cent grape sugar) is dependent upon nutrition; further constituents of the fluid elements of the blood are salts (especially sodium chloride and the end products of metabolism), which are dissolved in the blood and excreted by the kidneys (urea, uric acid, etc.).

Of great importance for pathology is the conduct of the morphological constituents of the blood, which lately has been accurately investigated and which requires a special description.

I. Red Blood Cells, Erythrocytes.—The *red blood cells*, "*erythrocytes*," are flat, circular, biconcave disks, averaging 7.5 micromillimetres in diameter (see Fig. 67, 1, 9, 13). Normally they do not contain a nucleus and consist of a protoplasmic stroma the gaps of which are filled with hæmoglobin.

The *hæmoglobin* contains an albuminous body (globin) and an iron-containing, organic colouring material (hæmatin), which crystallizes in rhombic prisms and with O_2 forms oxyhæmoglobin. In the spectroscopic picture the latter shows two absorption lines in yellow and green, the (reduced) hæmoglobin shows a broad washed-out absorption strip. The hæmoglobin has the faculty to combine with oxygen in so loose a manner that oxyhæmoglobin very easily decomposes into hæmoglobin and O_2, i. e., that oxygen may be rapidly given off in the circulating blood to the tissues of the body. Hæmoglobin is of the greatest physiological importance, therefore, as an oxygen carrier in the living organism.

The number of the erythrocytes is quite constant; a cubic millimetre contains normally, in man five millions, in woman about four and one half millions. Their upper surface is relatively large which makes them particularly favourable for the taking up and giving off of O_2. Of gases there are in the blood O_2, CO_2, and N. Oxygen as well as carbonic acid are only to the slightest degree physically combined, for the greatest part they are chemically combined. The taking up of O_2 in the blood occurs in the alveoli in the manner that O_2 is chemically combined with the hæmoglobin; the oxygen which is given off to the tissues from the capillaries is utilized for physiological combustion, which gives rise to the formation of CO_2 that collects in the tissues, being reabsorbed by the blood to be given off in the alveoli to the pulmonary air.

Formation of Erythrocytes.—The *formation* of the red blood corpuscles occurs in embryonic as well as in post-fœtal life from nucleus-containing, coloured blood cells, the *hæmatoblasts*. They are regularly contained in the red bone marrow of the flat bones, of the sternum and of the ribs, the base of the skull and the vertebral bodies. The red blood cells which at first still contain the nucleus, lose it later by caryolysis, i. e., by dissolution of the nucleus (according to the opinion of some investigating authors by expulsion) then to enter the circulating blood as non-nucleated red blood disks. We must at present adhere to the view that *in post-fœtal life the red (non-nucleated) blood cells are formed exclusively in the red bone marrow, namely from the nucleus-containing red blood cells which are constantly present in those regions.* The latter cells, the hæmatoblasts, appear, at least in embryonic life, to arise from lymphocytes by hæmoglobin production, at first as megaloblasts, which perhaps are originally the mother cells for the normoblasts. However, after the latter have been formed, *they increase by mitosis, constantly producing new normoblasts.* Probably in embryonic life, besides the bone marrow, also the lymph glands have an erythropoietic function; in the adult organism probably only the *bone marrow*, in which also the transformation of normoblasts into non-nucleated blood disks occurs by caryolysis. Under normal conditions only *non-nucleated* blood cells enter from the bone marrow into the blood. So soon as an appreciable quantity of nucleated red blood cells is found in the blood, pathological conditions are present: Infections, intoxications, inanition, or, if we are dealing with large amounts of nucleated blood cells, severe anæmias, and even of the severest character, are present (see Anæmia).

Decomposition of the Erythrocytes.—The red blood cells are permanently utilized normally and are substituted by fresh material coming from the bone marrow, so that the number of erythrocytes remains at an almost constant height. The *life-period* of the individual specimen is estimated to be from three to four weeks. The blood cells unquestionably disintegrate to the greatest part in the *liver*; we are justified in this assumption as a very small number of blood cells are determined in the venous blood of the liver, and, above all, for the reason that the daily amount of biliary colouring matter which is produced in large amounts in the liver, undoubtedly is formed from hæmoglobin. Oxyhæmoglobin, by this process, is decomposed into an iron-containing colouring substance, *hæmatin*, and a colourless albumin body of globulin-like quality, "globin." The hæmatin, $C_{32}H_{32}N_4O_4Fe$, is further changed, by taking up water ($+2H_2O$) and giving off iron, to bilirubin (isomeric to hæmatoporphyrin, $C_{32}H_{36}N_4O_6$). The iron which becomes free is utilized to the greatest extent in the liver, partly in inorganic, partly in organic, combinations, being retained in the liver cells and leucocytes (hæmatogenous siderosis) and later probably used for the formation of new, hæmoglobin-containing, red blood cells in the bone marrow. It is furthermore probable that the aging erythrocytes, before they are decomposed into globin and hæmatin, are taken up in the liver capillaries by leucocytes. Such leucocytes containing red blood corpuscles are found in the liver, in the pulp of the spleen and in the bone marrow. The greater the number of blood cells which decompose, the more copious will the decomposed, iron-containing material from the blood cells be accumulated in the liver (see Pernicious Anæmia).

II. **White Blood Cells.**—The *white blood cells* (*leucocytes*) represent colourless, membraneless cells with one or several nuclei and a protoplasm which is very variously constituted. Since the introduction of new methods of staining in examining the blood, the fact has been developed that there are not only two varieties of leuco-

cytes, as was assumed for a long time, but that there are numerous kinds, differing markedly from each other, in normal blood. We best differentiate two principal varieties—*mononuclear* and *polynuclear* (or *polymorphonuclear*) cells which both show various secondary subvarieties (compare Figs. 68 and 69).

(a) *Mononuclear Forms*: Cells with one nucleus and varying amounts of basophilic, non-granular protoplasm (see Fig. 68).

1. *Lymphocytes*, characterized by a *large*, round, *concentrically* arranged nucleus and a narrow protoplasmic ring. The latter, as well as the nucleus, reacts *basophilically*, especially markedly the protoplasm which shows no granulations. The lymphocytes possess no *amœboid movements*. The greatest number of them scarcely attains the size of the red blood disks (see Fig. 68, 1, 5, 11); rarely are *large lymphocytes* found, especially in the blood of children (see Fig. 68, 2, 6, 7, 8, 12, 13). The number of lymphocytes amounts to about 25 per cent of the white blood corpuscles. Some individual examples, especially of the larger forms, show small loose particles of the protoplasm (see Fig. 68, 7, 8).

2. *Large mononuclear leucocytes*, decidedly (two to three times) larger than the lymphocytes, differing markedly from the large lymphocytes in that the large, usually oval nucleus is mostly *excentrically* situated and that the non-granulated protoplasm is relatively plentifully developed (see Fig. 68, 3, 9). This and the nucleus react, as the lymphocytes, *basophilically*, but, in contrast to the latter, the protoplasm stains less readily than the nucleus. Transitional forms between the first and second variety are not observed, and for this reason, according to Ehrlich, the "large mononuclear" cells are strictly separated from the lymphocytes, all the more as "transitional forms" from the large mononuclear to the polynuclear are observed (i. e., large cells with neutrophilic granulation and with inlets in the nucleus) (see Fig. 68, 4, 10). The number of the large mononuclears in normal blood is always small (about 1 per cent).

(b) *Polynuclear Forms* (see Fig. 69): Cells with several small nuclei or usually with a polymorphonuclear figure, i. e., with marked indentations of the nucleus, so that the nuclear segments partially hang together by threads of communication. The polymorphonuclear ("polynuclear") leucocytes are further characterized by *amœboid movements*. The protoplasm is *granular*. The granules show ever-varying conditions regarding staining; thus giving rise to the differentiation of the following three varieties:

1. *Neutrophilic polynuclear leucocytes* (Fig. 69, 1, 5, 9), usually designated as "polynuclears," characterized by the close granulation of the protoplasm and the affinity of the same to neutral staining material. They form in the normal blood about 70 per cent of the white blood corpuscles.

2. *Eosinophile cells* (Fig. 69, 2, 6), characterized by their size and by the *coarse* granules of protoplasm which stain intensely by acid-colouring material (eosin). They resemble the neutrophilic polynuclear cells and, like these, are markedly contractile; their number amounts to about 3 per cent of the white blood corpuscles.

3. *Basophilic leucocytes*, "*Mast Zellen*" (Fig. 69, 8) are sparsely found in normal blood (0.5 per cent of the leucocytes) and characterized by intense basophilic reaction of the granules of the protoplasm and by the very slight property of tinction of the nucleus; the granulation does not colour with the triacid stain. The mast cells appear in triacid preparations as light, polynuclear, non-granular cells.

Formation of White Blood Corpuscles.—We have already seen that there is no complete unanimity of opinion regarding the development of the erythrocytes. The opinions differ widely in respect to the *formation of the leucocytes*, and it is very difficult to maintain a firm stand regarding this point as investigation is still being carried on in this respect. The following brief description, therefore, cannot take into consideration all the varying and markedly differing opinions regarding the development and nature of the white blood corpuscles. We owe our exact knowledge to a number of very excellent investigators, among whom there shall only be mentioned—Virchow, Koelliker, Max Schultze, Neumann, Heidenhain, Arnold, Uskoff, Rieder, Engel, etc., and, above all, however, the great investigator in hæmatology, P. Ehrlich, and, among the latest authors, Askanazy, Pappenheim, and Rubinstein.

Virchow's view that the lymphocytes are the young, the leucocytes the old, of the cell forms, the latter arising from the former, can no longer be accepted in its simple, strict conception, after the results of the investigations of the last decades, especially since the introduction of the tinctorial methods of examination by Ehrlich. Moreover, it is better to adhere to the view that the lymphocytes, the leucocytes and the hæmoglobin-containing cells represent distinctly separated stages of development of cells which probably are only alike in their first stages of development—namely, cells with non-granular, weakly basophilic properties and *one* round nucleus. From these there develop, in the *bone marrow*, the *myelocytes*, in that the protoplasm becomes granular (neutrophilic or eosinophilic), whereas the nucleus still retains its round form; in the later stages of development the granulation becomes more marked, the nucleus flattened, sinuate and, finally, fragmentary (polymorphonuclear leucocytes with neutrophilic or eosinophilic reaction). Being matured in this way, the cells enter the circulating blood as "polynuclear" leucocytes. Some few cells, before maturing, i. e., still basophilically non-granular and supplied with *one* nucleus, enter the blood as so-called "mononuclear" leucocytes, which then mature in the blood, becoming polynuclear leucocytes. Those cells which have begun to mature in the bone marrow, remain in the bone marrow up to their complete maturity, becoming polynuclear leucocytes, so that the blood does not normally contain myelocytes. The matured polynuclear cells, however, enter the blood upon chemotactic, physiological irritation in relatively small, usually quite constant numbers, under pathological conditions in frequently very large amounts ("leucocytosis"). The defects produced in this way of polynuclear cells capable of passing into the blood, are compensated by the formation of new primary stages of the same, the myelocytes, and by the further development of the latter to polynuclear cells in a correspondingly greater amount (see chapter on Leucocytosis).

In the *spleen* and *lymph glands* leucocytes are not produced, at least not in noteworthy amounts (even not in cases of marked necessity of the same, in leucocytosis). On the other hand, the lymph glands (the spleen only to a subordinate degree) are the points of origin for the formation of *lymphocytes*, which, as is well known, represent one quarter of the white blood cells in normal blood. They probably also, similarly to the myelocytes, are formed in the bone marrow from large, basophilic, mononuclear cells which, similar to the early stages of the leucocytes, generally do not enter the blood, or at most only as "large lymphocytes" in children, and under pathologic conditions in lymphatic leucæmia, in which they are met with in the blood (especially numerous in the acute form of this disease). The same as the myelocytes in their further development mature into polymorphonuclear leucocytes, "age," so is this aging process also noted in the nucleus of the leucocytes, in that it, later, undergoes lobulation and fragmentation (Rieder's cells), without the protoplasm losing its basophilic, non-granular character.

The nuclear changes—lobulation and fragmentation into several nuclei—therefore, point to the physiological age of the cells; just so may we assume, in general, that a slighter staining quality (*amblychromasia*), especially of the cell nuclei, characterizes the cells as incomplete, less developed, in contrast to the intensively staining ("*trachychromatic*"), matured forms.

The same as the hæmatoblasts and the non-nucleated red blood corpuscles, so are also *myelocytes and the polynuclear cells produced almost exclusively in the bone marrow;* only separated myelocytes are found (not introduced by the blood current) in the spleen and lymph glands. *On the other hand, the point of production of the (small) lymphocytes is in the lymph glands.* Naturally, small amounts of lymphocytes are also met with in the bone marrow; these, however, must be looked upon principally as having been carried in by the blood stream, and only partially, probably, are they also formed in the marrow.

The origin of the very small number of the mast cells, which under normal conditions are found in the blood, has not yet been determined with certainty. Most investigators assume that they originate in the connective tissue; others believe that they develop from lymphocytes.

Functions of the White Blood Corpuscles.—It must be stated that they play an important part in coagulation of the blood; it has been determined that the leuco-

cytes represent structures that are injured with extraordinary ease; when they adhere to foreign bodies, among which the desquamated endothelium of the damaged vascular wall, atheromatous areas of the same, etc., must be included, they decompose and are partly dissolved. This also occurs in blood which flows out on venesection, in that here, too, a destruction of the white cells may be noted, according to their individual resistance sooner or later, occurring in human blood in from three to four minutes. In this destruction of the leucocyte a ferment is formed, thrombin, which separates the albumin body fibrinogen, contained in the blood plasma, into a soluble globulin-like, albumin body, fibrin-globulin, and into the coagulating fibrin.

A further very important function is due to the leucocytes as structures which have the property to render innocuous certain deleterious substances which may threaten the life of the organism; in this respect particularly bacteria are to be regarded, the toxines of which exert a chemotactic action upon the leucocytes, i. e., drawing them to the bacterial focus. This property of attracting the leucocytes to those areas at which the bacteria have begun to show their toxic action, becomes possible from the fact that the leucocytes, and especially the polynuclear, possess amœboid contractility, in consequence of which they easily pass through the wall of the vessel and are able to migrate. After the emigrated leucocytes have reached the region of the bacteria, the action of the products of secretion of the white blood cells, the *alexines*, takes effect, by which the bacteria are destroyed, or at least their action diminished, and they are now taken up into the cell body of the leucocyte and carried away (Metschnikoff's "*phagocytosis*"). Further particulars regarding the part which bacteria play in immunization, will be found in the chapter on Infectious Diseases and in the description of leucocytosis (p. 800). The property of the leucocytes to take up foreign bodies does not only show itself in phagocytosis which affects bacteria, but also in the fact which has been variously determined, that small granules of all kinds—fat, pigment, etc.—become embedded in the protoplasm of the white blood cells; that also erythrocytes are taken up into the interior of the leucocyte has been previously mentioned.

Decomposition of Leucocytes.—As the erythrocyte, so also does the leucocyte constantly decompose in the living organism, new-formed cells taking their place. The masses resulting from the decomposition, as Ehrlich has shown, are taken up by the parenchyma of the spleen, and this, for the most part, gives rise to the splenic tumour which occurs in so many infectious diseases.

III. **Blood Plaques.**—As third constituent of blood, besides the red and white blood corpuscles, blood plaques occur, which were first described by Bizzozero, consisting of small, colourless, sticky or finely granular plaques of varying size (about three to four micromillimetres), forming usually smaller or larger masses. Whereas formerly they were looked upon as descended from decomposed white blood cells, especially as nuclear rudiments of the same, investigators who have lately busied themselves with the study of blood plaques, are inclined to the view that they originate from *parts of protoplasmic masses of red blood cells which have desquamated* and which gradually lose their colour.

ANÆMIA—CHLOROSIS—SEVERE ("PERNICIOUS") ANÆMIA

General Appearance of the Anæmic.—Anæmia may be discerned at first sight from the appearance of the patient; skin and mucous membranes are conspicuous by their *pallor*. There is to be specially noted the loss of colour of the conjunctivæ and of the lips, and the colour of the concha auris, in which areas, according to my experience, the pallor may be observed earliest and most intensely. The colour of the cheeks is not a standard to judge by; they may, according to the development of the cutaneous capillaries, which are more or less filled with blood, be pale, in spite of

the absence of anæmia, and, *vice versa,* in unquestioned cases of anæmia they may appear red.

Metabolism in Anæmics.—In a certain proportion of cases *œdema* develops, rarely being distributed over the entire body, more often usually present only at the ankles and the eyelids. Its development may be due to a *hypalbuminosis* which usually goes hand in hand with low states of the blood, and to the secondary poor nourishment of the vascular walls, due to insufficiency of the cardiac action in anæmics. Some patients appear to have lost flesh, others, on the contrary, are fat, so that they have a fatty-spongy appearance.

The reason for the last-named, conspicuous condition is to be looked for in various directions. Primarily we might expect, from a theoretical standpoint, that in anæmics a lesser degree of oxidation occurs than in the normal person. But estimations of the respiratory change of gases lately made have shown that the consumption of O_2 in anæmics is *not decreased,* but rather increased; also the estimation of the conversion of albumin in chlorotics has shown that this may be quite normal. If, now, in these cases, nevertheless, the accumulation of plentiful amounts of fat is observed, it must be due to the fact that in such anæmics the quantity of nourishment ingested is too great in proportion to the metabolism, and that, besides, certain conditions, increasing the calorie conversion, such as muscular labour, etc., are possibly less manifest in anæmics. In fact, it is not rarely observed that chlorotics take in more than the necessary amount of calories, and, on account of the continuous sensation of lassitude, do not perform any muscular labour, clothe themselves warmly, etc., i. e., systematically fattening themselves in that the superfluous food material, not being combusted, is accumulated as fat. A poorly chosen diet may, as will be thoroughly explained in the chapter on Obesity, also result in that, besides a loss of body albumin, on the one hand, fat will be accumulated if in the food but very little albumin is partaken of, besides much fat and carbohydrates. In such cases the organism sustains a loss in albumin; metabolism in general becomes impaired, and this gives rise to the fact that but little fat is decomposed and eventually is even accumulated (see Obesity). In other, *severer* cases of anæmia, however, a more marked decomposition of albumin appears to occur. At least, according to the experience of others and my own, *a conspicuously high nitrogen and urea excretion* was found in such cases. It is to be hoped that this question will be investigated further by new, exact examinations in metabolism in various stages of severe anæmias. If, then, as is likely after what has been mentioned, a marked decomposition of albumin occurs, the accumulation of fat in such cases could be readily explained. Owing to the marked decomposition of albumin, split products derived from substances rich in carbon would become free in large amounts and, as these are more readily combustible than the fat of food, the latter, if it is ingested in sufficient quantity in the nourishment, may be saved, and accumulate as fat.

Composition of the Blood.—The pale appearance of the patient suggests to the physician the examination of the blood of the anæmic. We may hope, by the microscopic demonstration of morphological changes in the blood corpuscles and by the estimation of the amount of hæmoglobin, i. e., of the material which furnishes the blood with its red colour, to obtain an opinion *intra vitam* regarding the nature and intensity of the disturbance of the blood in the individual case. We have all the more reason at once to describe the diagnostically important facts which have been developed lately in the examination of the blood, as the results of investigation are suitable to give us important points regarding the functional, diagnostically essential disturbances which arise in anæmia.

Amount of Hæmoglobin—Number of Erythrocytes.—The estimation of the *amount of hæmoglobin* may be easily determined by the use of various instruments (v. Fleischl's hæmometer—Gowers's instrument, [Dare's instrument,] etc.). The hæmoglobin is absolutely diminished in the various forms of anæmia; it varies, however, in its proportion to the amount of the red blood corpuscles. The latter may remain normal in amount in comparison, but may show great diminution of their hæmoglobin contents, or they may be diminished numerically, without producing a marked change in the hæmoglobin contents of the individual erythrocyte; in fact, in severe forms of anæmia there is even usually a relatively increased amount of hæmoglobin of the erythrocyte, with a simultaneous, enormous decrease in the number of the same.

In general, a *one-sided decrease of hæmoglobin with retention of, or but slight decrease in, the number of red blood corpuscles* is a sign of a *mild* character of anæmia; it is especially the character of the blood alterations in *chlorosis* and *anæmia following blood loss*, in which at a certain period of blood generation it is found that the number of red blood cells has increased more rapidly than the amount of hæmoglobin.

In the so-called *secondary anæmias*, which are due to other conditions occurring in affections characterized by more marked loss of substance and blood, *the number of red blood cells is diminished, the amount of hæmoglobin of the individual erythrocyte is*, however, slightly, if at all, diminished.

In the *severest* form of anæmia, finally, the so-called *progressive, pernicious anæmia*, with an *enormous diminution of the number of erythrocytes* (to a half, even a quarter, of a million, instead of four to five millions per cmm., as is normal), *and with a minimal total hæmoglobin contents, the latter is relatively high as compared with the number of red blood cells* (to which fact Laache especially called attention), i. e., therefore, the hæmoglobin contents of the individual red blood corpuscle does not appear as diminished but, on the contrary, increased.

But the diagnostic differentiation of the individual forms of anæmia upon the basis of the contents of hæmoglobin of the erythrocytes is only characteristic in general; exceptions to this rule occur in all forms, so that, therefore, chlorosis is noted with a marked diminution in the number of red blood corpuscles, exquisite secondary anæmias with unaltered numbers of erythrocytes and a relatively very marked diminution of the hæmoglobin contents (in a case of hepatic carcinoma in a man, aged sixty, I found no diminution in the number of red blood cells, but a hæmoglobin amount of 35 per cent), and also pernicious anæmias with a more marked decrease in the amount of hæmoglobin than is in keeping with the number of erythrocytes.

Changes in the Blood Corpuscles in Anæmia.—The *microscopical examination of the blood* shows variations in the size of the individual red blood cells (megalocytes and microcytes, see Fig. 67, 3), and various forms of the same (spindle, pear-shaped, etc.—*poikilocytes;* see Fig. 67, 2). The microcytes, which for the most part are extremely small and even may be pin-point in size, according to the very usual assumption of to-day, represent *fragments of the complete red blood cells* ("*schistocytes*," Ehrlich), in that the latter scatter into small plaques (similar to drops

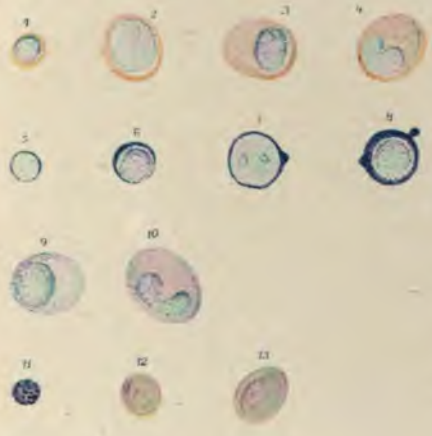
Fig. 67.

Red Blood Corpuscles.

1—8 b: Triacid stain.
9–12: Eosin-methylen blue "
13–15: Eosin-hæmatoxylin "

1, 9, 13. Normal red blood corpuscles.
2. Poikilocytosis.
3. Microcyte and macrocyte.
4, 14. Polychromatophile red blood corcles.
5, 10, 15. Normoblasts.
6, 7, and 11. Normoblasts with polychromatophile cell body.
8 a, 8 b, 12. Megaloblasts (large nuclei, polychromatophile protoplasm).

Fig. 68.



White Blood Corpuscles.

A. Mononuclears.

1–4: Triacid stain.
5–10: Eosin-methylen blue "
11–13: Eosin-hæmatoxylin "

1, 5, 11. Small lymphocytes.
6, 12. Medium-sized lymphocytes.
2, 7, 8, 13. Large lymphocytes, partly with protoplasmic segmentations.
3, 9. Large mononuclear leucocytes.
4, 10. Transition forms.

Fig. 69.

B. Polynuclear Forms and Myelocytes.

1–4: Triacid stain.
5–8: Eosin-methylen blue "
9 and 10: Eosin-hæmatoxylin "

1, 5, 9. Ordinary neurophile polynuclear leucocytes.
2, 6. Polynuclear eosinophile leucocytes.
8. Mast cell.
3, 7, 10. Myelocyte of a neutrophile variety.
4. Eosinophile myelocyte.

of fat forming into small balls). The process may perhaps be looked upon as one of compensation, in that from the deficiency of erythrocytes large corpuscles decompose into many small ones which in comparison to the former have a larger surface-complex, thus rendering them more favourable for combination with oxygen. They occur in all varieties of anæmia and are, therefore, not characteristic of any special form, especially not of pernicious anæmia, as was supposed at first. The same statement is true of *poikilocystosis*. Although the poikilocytes, which may be looked upon as degenerative forms of the red blood cells, are usually met with in severe anæmias, yet, exceptionally, they occur also in simple chlorosis, and even then in amounts which are greater than the normally formed red blood cells. Of more importance is the property of *the red blood cells in anæmia*, which was discovered by Ehrlich, to stain with *various* colouring materials (polychromatophylia), differing from the normal erythrocyte which stains with but *one*. The blood corpuscles in this case stain with eosinhæmatoxylin, not as is usual a pure red, but take on a mixed colour, at one time a more violet, at another time a blue-red tint. Ehrlich has proven that this behaviour is in connection with degenerative conditions of the erythrocytes, perhaps dependent upon an impregnation of the same with albumin substances, by which they obtain the property to combine also with nuclear-staining materials (see Fig. 67, 4, 11, 12, 14). Polychromatophylia of the red blood corpuscles is found especially in severe—pernicious—anæmia, also in secondary anæmias due to the chronic loss of blood.

Especially important, however, is the determination of *nuclear-containing blood corpuscles* in the blood of anæmics; we differentiate here, according to Ehrlich, *normoblasts* and *megaloblasts*. The *normoblasts* (Fig. 67, 5, 6, 7, 10, 11, 15) contain a nucleus surrounded by a ring of hæmoglobin (rarely more than one nucleus) and change into the usual non-nucleated blood corpuscles in that the nucleus is dissolved, according to others (Rindfleisch) in that it is desquamated. The authors accepting the last-named opinion assume that a nucleus which in this manner passes into the circulating blood, is again surrounded by protoplasm, and this gradually takes up hæmoglobin, thus causing new nucleus-containing blood cells to arise. The megaloblasts (Fig. 67, 8a, 8b, 12), (twice as large or even still larger than the red blood cell), on the other hand, do not desquamate their nucleus, but gradually resorb it. They are always found in small amounts in the blood of anæmics, especially in the severest (pernicious) form of anæmia, whereas the normoblasts are found in the milder forms of anæmia, as in chlorosis, and, on account of their property to transform into non-nucleated blood corpuscles, represent a favourable prognostic sign of the beginning regeneration of blood formation. The latter is to be especially assumed when, as occurs in cases of pernicious and secondary anæmias (not in chlorosis), it is observed that suddenly *large amounts of normoblasts appear simultaneously with numerous white blood corpuscles* (and especially the usual polynuclear neutrophylic) (von Noorden "blood crises").

As has been mentioned previously (compare p. 773), we observe in the adult organism, in the red marrow of the sternum, of the vertebral bodies,

etc., besides white cells and their preliminary stages, nucleus-containing, reddish-yellow stained cells, known as *hæmatoblasts*, being specially designated normoblasts. These latter lose their nucleus in the bone marrow by caryolysis and with this change into the usual red blood disks which under normal circumstances, loose, enter the circulating blood. In anæmia, however, besides non-nuclear, also nucleus-containing red blood cells, which usually remain in the bone marrow, enter the blood, and only then, by caryolysis or, perhaps, partly by desquamation, lose their nucleus. Further, however, also the production of normoblasts appears to suffer in that, in the severer cases of anæmia, they are not re-formed in sufficient amounts, or that mostly only the production of *megaloblasts* occurs and the hæmatopoiesis is limited to this process. As the normoblasts, so also do the megaloblasts enter the blood under these pathological conditions, becoming megalocytes if they there lose their nucleus by resorption, i. e., they become large hæmoglobin-containing blood disks. If megaloblasts and megalocytes are found in the blood, this is always a sign of severe anæmia, especially when they are met with in large amounts and are constantly present in the blood. In such a case we are dealing with a "*pernicious anæmia.*" The normoblasts and especially the megaloblasts are only inconstantly met with in the other anæmias, and then are but few in number.

In a sense of compensation, i. e., for the purpose of increasing the area of blood formation in the course of anæmia, the yellow bone marrow changes into red in various portions of the bony framework of the body. This process, however, is insufficient in pernicious forms of anæmia; in fact, in some cases this change into red marrow is absent entirely, and the regenerative power of the bone marrow is completely exhausted. Such cases of severe pernicious anæmia have been lately described by the name of the "*aplastic*" (Ehrlich) form. As the non-nucleated erythrocytes which are destroyed are not replaced by new ones, their number must diminish greatly, but also nucleus-containing reds must be absent in the blood, as new ones are not formed in sufficient amounts. Also the other derivatives of the bone marrow, the leucocytes, must be diminished in amount and their primary stages be absent in the blood; but the lymphocytes, which originate in the spleen and the lymph glands, may not be diminished, which gives rise to a change in the proportions of the leucocytes (see farther on).

In general, the *white corpuscles* show various conditions, according to the form and severity of the anæmia. Their *number* is at times increased, at others diminished; in the severest form, in pernicious anæmia, as a rule, they have been found *diminished* in number; in contrast to this, they were mostly *increased* in secondary anæmia; and almost constant in anæmia which was the result of septic affections and carcinomatous infection; but in simple chlorosis the number of leucocytes is about normal. Of more importance is the fact that the *mixture of leucocytes* is a specifically altered one in some forms of anæmia. Whereas in most cases of anæmia, most pronouncedly in carcinoma, a relative diminution of the polynuclears can generally be determined compared with the lymphocytes, *there is found.*

in contrast to this, a decided *percentile increase of the leucocytes in pernicious anæmia,* compared with the polynuclears, so that the proportion of both varieties of leucocytes (instead of the normal, three to one) may be one to one, or even may be still further altered in disparagement of the polynuclears. This fact is of great importance in a *diagnostic* respect, as in doubtful cases it facilitates the recognition of pernicious anæmia in general, and especially the differential diagnosis between pernicious anæmia which is accompanied with atony of the mucous membrane of the stomach and insufficiency of the gastric-juice secretion, and the anæmia dependent upon gastric carcinoma (Strauss and Rhonstein).

As a rule, the change of white blood cells in the blood of anæmics is not important compared with the severe damage of the hæmatopoietic process, in so far as the erythrocytes are concerned. In my opinion it is very largely a question in anæmia of the action of deleterious substances which in the main only influence the production of red blood cells.

Behaviour of the Blood Plaques.—Regarding the *conduct of the blood plaques* in the various forms of anæmia, nothing of importance has as yet been determined for the diagnosis. It is certain that in the blood of some anæmics, especially chlorotics, numerous large groups of granules are found, in which the fibrinous net of coagulation may be seen to originate in the preparation, and which, in my opinion, may be looked upon as blood plaques.

Coagulability of the Blood.—The *coagulability of the blood* varies greatly in the different forms of anæmia. In the mild and in the medium severe forms, such as chlorosis and secondary anæmia, a tendency to rapid coagulation and thrombus formation is undoubted (in the veins of the extremities and in the cerebral sinuses), whereas, in contrast to this, in the severest form of anæmia, progressive pernicious anæmia, the blood is characterized by an absence of the tendency to coagulation, so that patients affected with this form of anæmia are liable to bleed very strongly from slight wounds.

The estimation of the *specific gravity and dry residue of the blood* shows in anæmics, in keeping with the loss of hæmoglobin, a uniform diminution of the same; i. e., we find, instead of the normal figures 10.55 to 10.60 (specific gravity) and 21 to 22 per cent (dry residue), only 10.30 to 10.50 and 12 to 18 per cent; besides, the blood of anæmics shows a diminished *quantity of iron,* of, for example, 0.35 per cent instead of 0.53 per cent medium value of the normal blood. Great as the scientific value of the last-named investigations of the blood may be in physical and chemical respects, they can, at present, be very little utilized in the practical diagnosis of anæmia.

Of the greatest diagnostic importance are the *consequences* of the anatomical and physiological changes in the blood in anæmia, i. e., the *functional disturbances* which become more or less prominently manifest in the course of this affection.

Dyspnœa.—One of the most common symptoms is *dyspnœa* caused by the diminution in the number, and the loss of hæmoglobin, of the blood corpuscles, producing an impairment of the respiratory interchange of gases in the blood. As a rule, however, air hunger during rest is but scarcely indicated.

The occurrence of dyspnœa is evidently prevented by compensatory arrangements which become active in anæmia. But it is very difficult to decide what they consist in; relatively greater O_2 combination by the diminished hæmoglobin, (arterial blood contains normally only as much O_2 as its hæmoglobin can combine) may be a factor of

compensation, and the complete O_2 combination may be favoured by the decomposition of the blood disks into microcytes. Besides, the diminution in the formation of biliary colouring matter and urinary colouring matter prevents the utilization of hæmoglobin. Examinations of the urine of chlorotics have actually shown a marked diminution of urobilin contents, up to the merest traces.

As soon, however, as a more marked consumption of O_2 occurs, which is certain to take place in muscular labour, especially in marked body movements, the difficulty in taking up oxygen and the more pronounced irritation of the respiratory centre by the blood which has become poor in O_2 and rich in CO_2 is noted in the form of deeper, and especially of increased, respiratory movements, the excursus.

Muscular Weakness—Lassitude.—A further important symptom of anæmia is *insufficiency of the property of function of the musculature*; anæmics *tire readily*, even upon insignificant bodily exertions, and almost without exception complain of general *lassitude and atony.*

To explain these symptoms in anæmics we must consider certain physiological facts, viz., that the blood-containing muscle in general is able to perform a greater amount of work than the bloodless muscle; further that the restitution of the irritability of tired muscles is especially assisted by the injection of blood-containing O_2.

Anæmic Disturbances on the Part of the Nervous System.—We now come to a subsequent symptom of anæmia, which explains the various morbid phenomena of the condition, the disturbance of nervous irritability. The irritability of the nerves depends under normal circumstances upon a uniform nutrition and a sufficient supply of blood. As soon as these fundamental conditions for the normal performance of nerve function are interfered with, the nerve irritability declines. *This diminution in nerve energy is preceded, as a sign of disturbed nutrition and blood supply, by a stage of increased irritability of the nervous system.* In a *clinical* respect this influence of disturbed nutrition is shown, on the one hand, by *increased irritability of the nervous system,* and, on the other hand, *by a rapid tiring of the overexerted, stimulated nerves.* This condition has, therefore, been given the suitable name of "*irritable weakness*"; it is a characteristic attribute of the nerve activity in anæmics and the source of various disturbances in the nervous organism of the same. To this are due the *neuralgias,* the cephalalgia, the pain in the back, the gastralgia, the nervous dyspepsia, the neurasthenia and hysteria, the *abnormal irritability in the region of the combined sensations* (which see) (manifesting itself by disgust and bulimia, satiation, perverse desires, etc.) and certain disturbances in *the psychical sphere* (conditions of exalted moods, of crying, apathy, etc.). Further, also, the *phenomena of abnormal reaction of the vaso-motor nervous system* belong here (morbid blushing and pallor of the skin, trophic disturbances in the hair and nails, polyuria), of the *nerves of special senses* (tinnitus aurium, being sensitive to bright light, noises, etc.). *The increase of reflex irritability* which is combined with anæmia, the tendency to spasms, which in women and especially in children ("hydrocephaloid") may increase to eclampsia, are also the results of the increased nervous irritability, whereas the decrease of nervous energy, which is very apt to

occur, may, on the other hand, show itself in the form of *pareses*, etc. In some cases, in the course of severe anæmias, tabetiform symptoms and, later, paralytic phenomena were observed, to which degenerative processes in various column systems were found to correspond post mortem. Among the most frequent phenomena depending upon anæmia are the symptoms of anæmia of the brain (see p. 700), especially the *attacks of fainting*. These are the result of the insufficient cerebral nutrition and flow of blood, and the function of the brain may be suddenly and momentarily suspended by the weakness of the cardiac activity. This latter condition also manifests itself in other changes in the circulatory apparatus, which may be partly physically demonstrable.

Anæmic Symptoms on the Part of the Circulation.—The *size of the heart*, as a rule, is *not altered*. Dilatation may be determined in some few cases, which is due to the poor nourishment and the fatigue of the heart muscles; in other, but very rare, cases—I have never seen such cases in my experience—hypertrophy of the left ventricle may be found, which is said to be due to a congenital hypoplasia of the arterial vascular apparatus. Very commonly *accidental* "anæmic" murmurs are heard on *auscultation*; they are (compare Anæmic Murmurs) less intense, as a rule, than those which are due to valvular lesions, they are soft, blowing, and sufficiently plainly heard during the systole and especially loud over the punctum maximum of the pulmonary artery. They are especially distinguished from endocardial, respectively myocardial, murmurs in that their intensity varies at certain times, and, further, hypertrophy of the heart as well as an accentuation of the second pulmonic sound are absent. The murmur is heard only at the pulmonary artery or, at least, is most intense there.

Anæmic Cardiac Murmurs—Explanation of their Origin.—To account for the production of *accidental, respectively anæmic, murmurs* in a satisfactory manner, gives rise to certain difficulties. All explanations, which, as is usually the case, ascribe them to a weakness of the valvular apparatus of the mitral valve, are, in my opinion, not sustainable. In this case a cause of the production of murmur would have to depend upon a relative insufficiency of the mitral valve, but this itself would necessarily have to appear, with other physical consequences, in the clinical picture, which in fact does *not* occur, as a rule at least. I therefore place the area of production of the murmur at the base of the heart, at which the murmur is most distinctly heard, i. e., in the beginning of the large vessels, especially of the pulmonary artery. In that the wall of the latter, in anæmic conditions, is in an atonic condition it dilates more markedly in the systolic pressing in of the blood than under normal circumstances; by this dilatation, isochronous with the systole, of the beginning of the pulmonary artery, the conditions for the production of the murmur are given. Another explanation of the occurrence of this murmur is given by R. Geigel, who believes the cause of the production of the murmur to consist in the slighter pressure in the aorta and pulmonary artery (compared with the *relatively* strong ventricular pressure). This is to prevent the closure of the semilunar valves in the first part of the systole, in the so-called closure period, giving rise to the production of a murmur at the base of the heart, the area of "physiological stenosis."

Not always do anæmic murmurs exist without simultaneous changes in the size of the heart being demonstrable. On the contrary, it is not so rare in anæmics to find an increase of cardiac dulness (to the right up to the right sternal border and even beyond). This need not, however, be dependent upon an increase in the size of the heart, as has lately been emphasized by various authorities, but may be due to a more marked exposure of the heart, in that, in consequence of the superficial respi-

ration in the anæmic patient, the borders of the lungs retract. The high position of the diaphragm, respectively of the lower border of the lungs, and the high position of the impulse of the heart in the fourth intercostal space, favour this view. Apart from this more marked exposure of the heart, which may simulate a dilatation, there will remain cases in which an *actual* enlargement of the heart in anæmics occurs and an *accentuation of the second pulmonary sound* can be determined besides the systolic murmur. The murmur in general is weak, varies in intensity according to the temporarily stronger contraction of the valve muscles, and may even *be absent entirely at times.* In such cases we are dealing with a *relative insufficiency of the mitral valve* with its consequences, which is diagnosticated according to the signs previously described, i. e., can be differentiated without difficulty from those cases in which pure functional anæmic murmurs are present.

Venous Murmurs.—The *anæmic cardiac murmurs are almost exclusively systolic in time*; if in very rare cases a *diastolic murmur* is found, this is a transmitted venous murmur (compare Venous Murmurs), therefore has actually nothing to do with a genuine anæmic murmur. The auscultation of the *veins* of anæmics may show murmurs, as a rule, which are audible most constantly and most intensely as uninterruptedly persisting hums over the bulb of the jugular vein (*nun's murmur*). A satisfactory explanation for the origin of the same I cannot give; they are not diagnostically important, because they also occur in healthy persons, although much more rarely.

Condition of the Pulse.—As already remarked, the energy of the heart is weakened in anæmia; in severe cases the heart muscle becomes fatty (at autopsy the musculature appears yellow, speckled or "tiger-spotted"). Accordingly, in such cases the impulse is weak, the heart sounds feeble and the *pulse small*; in other cases, on the contrary, the pulse wave is *large,* simultaneously the pulse is exquisitely *dicrotic.* These are alterations in the condition of the pulse which point to the fact that, in consequence of the diminished tone of the vascular wall, the resistance to the blood wave which streams into the arteries is slighter than normal; this also is in connection with the *capillary pulse* and the *crural double sound* in anæmics.

The occurrence of the *crural double sound* is explained in the manner that, in spite of the diminution of arterial blood pressure which is found in anæmics, the pulse wave is still sufficiently great to change the abnormally weak tension of the arterial wall into a *relatively* strong, abrupt one, giving rise to the production of tone; during the systole this occurs by the primary wave, during the diastole as the so-called recoil wave.

The *pulse in anæmics is usually accelerated* (80 to 100 and more per minute); why this is so is not easy of explanation. From the diminution in blood pressure we should expect a diminished frequency of the pulse. However, it must not be forgotten that the pulse frequency is dependent upon various factors, and that the slowing of the same, which might be supposed in one direction, is compensated for by the action of other factors, and may even be changed into the contrary condition. Such a factor, which would prevent the slowing of the pulse in the present instance, increasing the rapidity of it, is the influence of the vagus centre upon the pulse frequency. In that this centre is poorly nourished in anæmia and, by the diminution in the blood pressure occurring therein, is subject to a lessened mechanical stimulation, a diminution of the tonus within the centre occurs, giving rise to a curbing of the cardiac activity by the vagus fibres, thus accelerating the rapidity of the pulse. The latter may even be increased in case, by an abrupt rising of the patient, the gravity interferes with the flow of the arterial blood to the brain—a fact which can be proved at any time. But there may also be a reason in the heart itself for the increase in the pulse frequency, in that the increased irritability of the nerves in general also reaches the cardiac nerves in anæmia.

Cardiac Palpitation.—In favour of this would be the sensation of *cardiac palpitation* which is frequently combined with increased pulse frequency; both may be referred to some marked irritability *of the cardiac ganglia* (see Cardiac Neuroses).

Temperature Conditions.—*Chilliness* in the anæmic is due to the deficient circulation in the skin; rarely do actual chills occur, i. e., involuntary muscular movements, which may possibly result in an increase of the production of heat in the sense of a regulation of the latter. Especially frequent are the complaints of the patient regarding cold feet and hands, slight "numbness" of the fingers. The *temperature,* taken in the rectum, in the majority of cases shows *normal* conditions, between 98° and 99.5° F.; on the other hand, it is certain that occasionally "febrile" temperatures, for which only the anæmia itself may be held accountable, are observed. It it almost universally assumed that this "anæmic fever" only occurs in the severe forms of anæmia. According to my experience, this is not quite correct, as slight rises of temperature, between 100° and 101° F., also occur in chlorosis, although very rarely, and the most minute examination reveals no other cause for the fever than the chlorosis itself. With the assumption that the source of the fever in anæmia is due to the poor regulation of heat by the exhausted nervous system, not much is gained for the understanding of the conspicuous facts.

Disturbances of Secretion.—On account of the faulty supply of the blood, the activity of the secretory organs, above all of the gastric glands and of the kidneys, suffers.

Gastric Disturbances.—In a certain number of cases, as might *a priori* be expected, it is a question in anæmia of a *diminution of gastric-juice secretion,* especially of the acid, subacidity of the gastric juice being present. In other cases, and this appears to be the majority, the contrary, *superacidity,* is found.

This apparent contradiction, that an organ poorly supplied with blood develops an excessive activity, in regard to its secretion, can easily be explained in that the greater irritability of the nervous system produced by anæmia also shows itself in the sphere of the gastric nerves as a neurosis of secretion, in the form of a morbid, excessive secretion of gastric juice. At all events, dyspeptic phenomena are among the most usual symptoms of anæmia, and, after the determination of the condition of secretion of the affected stomach, an explanation of the prevailing disturbance of function of the stomach should be attempted in this sense. It need scarcely be mentioned that, as a result of acidity, chronic gastric catarrh develops, and, on the other hand, on account of the existence of superacidity, gastric ulcer may develop; this complication may often give rise to difficulties in the diagnosis in respect to the question which affection is primary in the individual case.

A pathological yielding of the gastric walls may be in connection with the early gastrectasis which occurs in anæmia, viz., *gastric atony.* However, according to the existing reports of investigations, no certain opinion is as yet possible regarding this sequel of anæmia. The former assumption, according to which gastric atony, respectively gastroptosis, could be determined in 80 and more per cent of chlorotics, has turned out to be too sweeping; especially was it but rarely possible to determine a diminution of the motor power of the stomach after test meals.

Urinary Changes.—The function of the *kidneys*, under the influence of anæmia, is altered in various ways. The colour of the urine is conspicuously light, which is *not* to be wondered at on account of the derivation of the colouring matter of the urine from hæmoglobin. The amount of urine varies; marked polyuria is rare. The specific gravity also varies; usually it is diminished; in rare cases, especially in the severe forms, it is relatively high on account of the previously mentioned, increased excretion of urea. The *nitrogen* respectively *urea excretion* in the mild forms of anæmia remains within normal bounds; only in the severe (the secondary and pernicious) forms is it occasionally increased. Admixtures of *albumin* in the urine in mild cases of anæmia are rare, frequent, however, in the severe anæmias; also *albuminosuria* is said to occur in cases of severe anæmia.

Albuminuria.—The cause of albuminuria is principally due, in my opinion, to the poor nutrition of the renal epithelium, especially of the glomerular epithelium. If albuminuria, in spite of this, is absent in most cases of anæmia, this may be explained in that this damage of the epithelium by the absence of oxygen, as a rule, does *not* take on such marked dimensions that the property to retain albumin is lost thereby, but rather that the activity has only transitorily become weakened and occasionally permits albumin to be present in the urine, if at the same time the factors that favour the presence of albumin, such as bodily exertion, etc., are active. *Casts*, apart from hyaline casts, are not present in the albuminous urine unless a nephritis is the cause of the condition; blood, however, has been demonstrated in the urine, probably as the result of the hæmorrhages in the urinary passages. That the last-named condition may occur in the course of anæmia is easily understood, as anæmia in general predisposes to hæmorrhages in the various organs of the body, a fact to which we shall refer in detail later on.

Œdema, Hæmorrhages.—The insufficiency of nutrition (eventually fatty degeneration) of the vascular walls, resulting from the deteriorated condition of the blood, gives rise either to *œdema* or to *spontaneous hæmorrhages* which may occur in the tissues or also in the surfaces of the body. Such hæmorrhages are met with, besides in the retina, especially in the gums, in the skin (as purpura), in the muscles, in the meninges; the tendency to hæmorrhages is further shown in nosebleed, profuse menstruation, etc. *The hæmorrhages are in general a sign of severe anæmia, but, according to my experience, they may even occur, and be very profuse, also in the mildest forms of anæmia, i. e., chlorosis*, therefore, neither in a diagnostic nor in a prognostic respect dare their importance be exaggerated. Although, as has been mentioned, menstruation occasionally occurs profusely, much more frequently the flow is sparse or may be absent altogether; with this very usually leucorrhœa is present.

Enlargement of the Spleen.—In a small number of cases of anæmia moderate *enlargement of the spleen* may be demonstrated; in the cases observed by me clinically the spleen was palpable in about 16 per cent (71 in 434 cases). It appears certain that the enlargement of the spleen in anæmia is not accidental but is in close relation with the malady, even if I am not yet in a position to explain it. In a practical connection I have made the observation that the cases of anæmia, and chlorosis, in which the enlargement of the spleen could be recognised, offer especially favourable therapeutic chances by the simultaneous administration of quinine and iron, more favourable, as I believe, than cases of chlorosis that are not accompanied with enlargement of the spleen and which were treated with iron alone.

Condition of the Eyes in Anæmia.—Characteristic changes in a diagnostic respect are given finally, by the *visual changes in anæmia*: If *anæmia has occurred suddenly* by a more or less plentiful blood loss, a marked diminution in the sense of sight occurs, as a rule, blindness. Ophthalmoscopically the vessels, especially the arteries, appear poorly filled, the optic-nerve papilla is pale, besides slightly turbid, as well as the adjoining retina; more and more an atrophy of the optic-nerve papilla

develops. In *chronic anæmia* most frequently a tortuous and diffused condition of the venous vessels of the retina appears, here and there also a more marked dilatation of the arterial as well as of the venous retinal vessels, combined with a less dark colour of the venous blood (deficiency of hæmoglobin). Rarely are small retinal hæmorrhages noted. In *secondary anæmia* the arterial vessels are poorly filled and narrowed; the column of blood is light red in colour. The veins are less filled than normally, frequently showing in individual portions a coarser branching, the blood column is engorged and of a deep dark-red colour. Corresponding to these areas the retina is to a great extent dimmed to a yellowish or whitish-gray colour, hæmorrhages being present throughout; without doubt this is due to a thrombus formation. A very pale, turbid appearance of the optic-nerve papilla is conspicuous at the same time. In *pernicious anæmia* hæmorrhages are found in the immediate neighbourhood of the optic-nerve papilla, being plentiful, as a rule, though not of marked extent. Frequently a lighter, reddish-green area is noted in the midst of a hæmorrhage. The arteries are narrow, the veins markedly tortuous, the blood column in the latter is of a deep dark-red colour, and in some areas it appears as being thrombosed. The optic-nerve papilla and the retina appear to a greater or less extent turbid, also the macula, which may be permeated by hæmorrhages, even giving rise to the pathological picture of so-called retinitis albuminurica.

Diagnosis of Primary and Secondary Anæmias.—After the diagnosis of anæmia has been determined upon the basis of the phenomena just described, the question suggests itself, which is usually more difficult to solve, as to the causation of the affection; there is to be decided, further, whether in the individual case a primary, an "idiopathic" or a secondary anæmia is present. Practically the latter may be excluded at first, i. e., we determine by minute investigation of the individual organs and of the secretions whether changes are present anywhere which are acknowledgedly in connection with a marked disturbance of metabolism, especially with a greater destruction of the blood. To be considered, above all, are malignant disease, diabetes mellitus, Bright's disease, exhaustive diarrhœas, a prolonged and weakening puerperal period, phthisis pulmonum, in general long-lasting febrile conditions and diseases which run their course with severe blood loss. Especially is there also to be considered the presence of chronic intoxications and infections (mercury, lead and arsenic poisoning; of long-lasting malarial and syphilitic infections, etc.), and it should be observed whether or not milder affections of the gastro-intestinal tract, which, on account of their long duration, produce deleterious affections of the digestive organs (gastro-intestinal catarrh, atrophy of the mucous membrane), which impair the blood formation by a faulty assimilation and by disturbance of the nourishment. The fæcal evacuations are to be especially investigated in doubtful cases, for the presence of the anchylostomum or for eggs of this parasite (a relatively frequent cause of severe anæmia). Why the presence of a tapeworm in one case gives rise to severe anæmic conditions and in other cases all such symptoms are absent, has not yet been made clear. I do not regard it as necessary to enter more minutely into the ætiology of secondary, symptomatic anæmia; only it shall be especially emphasized that the most important rule for the diagnosis of anæmia is, only then to assume a primary anæmia if none of the causes which give rise to an impoverished condition of the body regarding the constituents of the blood, is present as an underlying condition—a rule to which it is

also well to adhere in the interest of the therapy. The blood findings as such, as we have seen, give *no absolutely decisive* explanation regarding the secondary nature of the anæmia; *in general,* there is a diminution in the number of the erythrocytes in secondary anæmias, with slight reduction of their hæmoglobin contents, and an increase of the white blood corpuscles. As we have seen, only the *positive* finding that a well-developed leucocytosis is present, *besides a diminution of the erythrocytes,* may be utilized in the diagnosis of secondary anæmia. The absence of leucocytosis in the individual case does not exclude the presence of secondary anæmia, as, although relatively rarely, cases occur in which no increase of the leucocytes can be noted.

Diagnosis of Individual Forms of Primary Anæmia.—If the anæmia, after excluding the secondary nature of the affection, has been determined as *primary,* the question must now be decided which variety of primary anæmia is present, a question which is especially important in a *prognostic* respect. To separate the mild form from the severe by the symptoms, is very frequently, at least in certain stages of the disease, quite impossible. It is true, retinal hæmorrhages, continued fever, and dropsy occur to a marked extent only in the severe, so-called "pernicious" form of anæmia. But none of these symptoms, which are considered partly to be almost pathognomonic, is exclusively peculiar to pernicious anæmia, even if they (as well as the waxlike, pale appearance, in short an increase of one or the other subsequent phenomena of anæmia *ad maximum*) give rise to the suspicion that we are dealing with the pernicious form. Only the results of careful examinations give a firm support to the diagnosis: The presence of poikilocytes and polychromatophilic red blood corpuscles, but, still more, the presence of megalocytes and of numerous nucleated red blood corpuscles, especially of *megaloblasts.*

None of these abnormal blood constituents is *absolutely* pathognomonic of *pernicious anæmia.* Only the amount and constancy with which they are found in the individual case, decide the pernicious character of the anæmia. The severe nature of the special case may under some circumstances be deduced also from the *number* of the erythrocytes; the number of the erythrocytes is occasionally so enormously diminished in pernicious anæmia, that the diagnosis of chlorosis may be excluded without difficulty. Whereas counts of less than one half million per cmm. were found in the former, in chlorosis the number of the red blood corpuscles is either entirely normal or rarely falls, not even in severe cases, below three millions. As the number of red blood corpuscles, so also are the leucocytes quite normal, as a rule, in *chlorosis.* The polynuclear leucocytes are by no means diminished, and the lymphocytes, on the other hand, not increased. If a conspicuous *relative increase of lymphocytes* is present, this may be *utilized in diagnosis* in favour of the presence of pernicious anæmia.

[Colour Index.]—Of great importance is, further, the observation of the *amount of hæmoglobin* of the blood in proportion to the number of erythrocytes. *This, in chlorosis, is diminished without exception, usually to a conspicuous degree, compared to the number of erythrocytes*; in *pernicious*

anæmia, however, the hæmoglobin contents of the erythrocytes, although absolutely much reduced, yet relatively, i. e., in comparison to the number of erythrocytes, it is often high, so that individual erythrocytes are abnormally *rich in hæmoglobin.*

Finally, the observation of the *ætiology* of the special case of primary anæmia occasionally gives the key to the diagnosis.

Ætiology.—*Chlorosis* is *a disease of development especially of the female sex,* in connection with a weakness of the constitution and especially of the hæmatopoietic organs. The disease may even attain its full development in childhood, usually, however, at the time of puberty, from the twelfth year on (up to the first half of the third decade). Why in this period of life only the formation of blood, particularly of hæmoglobin, suffers, has not been determined. Probably it is due to a condition of *exhaustion* in connection with excessively increased growth and formative processes which only occur during the time of puberty. That the production of chlorosis may be favoured by deleterious auxiliary causes acting upon metabolism, such as loss of sleep, bodily overexertion, early pregnancies, bad nutrition, faulty hygiene, etc., is self-evident.

The anæmias which occur in the second half of the second decade, provided they are not secondary, are as a rule of a severe character. The therapeutic use of iron, in contrast to the cases of chlorosis, has rarely a favourable influence on the course of the affection, which, on the other hand, often shows an exquisitely progressive character and then almost always terminates unfavourably (*"pernicious" anæmia*). Whereas chlorosis almost exclusively occurs in the female (rarely also in cases of weakly, effeminate young men), severe pernicious anæmia is not at all rare in men (my own experience regarding this disease, for example, has been more with men than with women), although it is certain, on the other hand, that it shows a special preference to follow upon pregnancies and the puerperium. It has been determined as certain, further, that the appearance of the disease geographically is unequally distributed, i. e., in certain cities and countries (for instance, Switzerland, France, Scandinavia), it is unquestionably more frequent than in others. The secondary causes which have been described as causing chlorosis are also active in progressive anæmia; they are, however, by no means the original causes of primary severe anæmia, as little as severe digestive disturbances, toxine-producing (?) helminthes or certain diseases which go hand in hand with disturbances of metabolism, etc. For some cases of pernicious anæmia it is impossible to find an ætiological support; in others we note the appearance of deleterious causes producing the disease, of which we know that they give rise to secondary anæmia. Why in one case they should be of a harmless character and in another give rise to a pernicious disease which progressively leads to a fatal termination, is undetermined.

In my opinion it is principally a question in diseases of the blood, *which kind of noxa* influences the blood-forming process, and upon this it depends which changes appear in the clinical picture of the blood.

1. If the damaging cause *in the main affects the red blood corpuscles,* there results the usual *"anæmia,"* in which the erythrocytes, in regard to number, form, maturity and amount of hæmoglobin, more or less deviate markedly from normal conditions; whereas the white blood corpuscles are only secondarily, and especially numerically, altered.

2. If the noxa in the main affects the *white blood cells,* and if, above all, their number is increased, the disease of the blood shows itself in the form of *leucæmia.*

3. If the damaging agent affects *all* constituents of the blood, there arise diseases of the blood with complete suspension of blood formation, with changes in the number and form of the white *and* red blood corpus-

cles; blood affections arise which may be looked upon as transitional and mixed forms of pernicious anæmia and leucæmia. If we adhere to the usual classification of anæmia or leucæmia, it is difficult to place individual cases of this third category into the framework of anæmia or leucæmia. I regard it more proper, therefore, to describe them especially and designate them by a particular name, such as "leucanæmia." Before we proceed to the description of these cases, which, as yet, are not well known, we must first discuss the second variety of blood affections, *leucæmia.*

LEUCÆMIA

The symptom which primarily determines the diagnosis of leucæmia is the *increase of leucocytes in the blood.*

Composition of the Blood.—The microscopic examination of a drop of blood obtained by puncture or incision in the tip of the finger, reveals, in cases of leucæmia of high grade, without more exact examination, the abnormal increase in the white blood corpuscles. A count of the latter in comparison to the number of erythrocytes is absolutely necessary in less markedly developed cases. Whereas normally an average of 8,000 leucocytes (in children somewhat more, 9,000 to 10,000) is contained in a cubic millimetre of the blood taken from the veins, i. e., for 600 erythrocytes one white cell is present, the number of leucocytes may increase so in leucæmia that the proportion is 1 to 50, 1 to 10, yes, even 1 to 2, or the red and white blood corpuscles may even be present in similar amounts in the blood.

Erythrocytes.—Besides the increase of the white cells in some forms of leucæmia, no noteworthy change in the *erythrocytes*, either in number or appearance, occurs: *as a rule, however, they are decreased in numbers* to about one half of the normal and below, similar to conditions in severe anæmias. In general the number of erythrocytes decreases in proportion to the increase in the number of white blood corpuscles. However, the numerical condition of the erythrocytes in leucæmia is more of a secondary consideration, and the determination of the affection finally depends primarily upon the white blood corpuscles. If, in cases of anæmia, the number of red blood corpuscles has become markedly diminished, but the production of white has remained normal, it may occur that the proportion of the white cells to the red ones is markedly changed, 1 to 25, etc., without leucæmia being present at all; for example, in cases of pernicious anæmia with a diminution of the erythrocytes to 250,000 to the cubic millimetre, in a normal condition of the white blood cells (8,000) the proportion would be about 1 to 30, and still no leucæmia would be present. Under all circumstances, however, leucæmia is to be assumed if the proportion falls below 1 to 20, in that these figures *can only occur* if, besides the decrease in the number of the red blood corpuscles as a result of anæmia (even in the most excessive diminution of the same, observed until to-day in cases of pernicious anæmia), the number of leucocytes is absolutely increased at the same time. Besides the diminution of the number of red blood corpuscles, the blood also contains, as a rule, *nucleated red blood corpuscles* in the form of normoblasts, more rarely megaloblasts or transitional forms between both of these. The *amount of hæmoglobin in the blood* in leucæmia is also diminished, but the colouring of the *individual* (abnormally decreased in numbers) corpuscles need not be diminished. An increase in the blood plaques has also been observed upon several occasions.

Whereas in cases of extraordinary increase of the white blood corpuscles the diagnosis of leucæmia can be made without further consideration,

in questionable cases the appearance of the latter affection can only be determined by an exact *microscopical examination of the morphological composition of the leucocytes.*

As has been mentioned previously (see p. 774), the normal blood contains partly mononuclear white blood cells (lymphocytes, large mononuclears), partly polynuclear leucocytes (neutrophile, eosinophile and basophilic polynuclear cells). The mononuclear cells may be further differentiated from the polynuclear in that they contain a *non-granular*, basophilically reacting, protoplasm mass, whereas the protoplasm of polynuclear cells is almost always neutrophilic, eventually eosinophilic, in reaction, but, above all, with finer or coarser *granules*. Besides, the polynuclear cells are further characterized by their great *amœboid movability*, which gives them the property of emigrating from the bone marrow and from the vessels to a marked extent.

Regarding the formation of the white blood corpuscles, we know that the *polynuclears in the bone marrow* originate from mononuclear cells, *the myelocytes* (see Fig. 69, 3, 4, 7, 10), that their protoplasm is granular and reacts neutrophilically or eosinophilically. In that the originally round nucleus becomes sinuate and segments, the respective neutrophilic or eosinophilic myelocyte becomes the neutrophilic or eosinophilic polymorphonuclear or polynuclear leucocyte. These matured cell forms are capable of entering the circulating blood, whereas the primary stages of the same, the *myelocytes* (like the primary stages of the erythrocytes, the nucleated hæmatoblasts), remain in the bone marrow until they are matured and on this account *are normally absent in the blood.*

Leucocytes are seldom formed in the *spleen* and *lymph glands* in normal conditions, these organs are rather the *regions of formation for the lymphocytes. Besides, the assumption that the leucocytes are only produced in the bone marrow, the lymphocytes for the most part solely in the lymph glands, is only correct in general.* Because, on the one hand, typical myelocytes which were introduced with the blood current and which are capable of further formation into polynuclear leucocytes, are now and then found in the spleen and lymph glands, and, on the other hand, small quantities of lymphocytes are met with in the bone marrow, which may partly be looked upon as having wandered there, but which partly, as has lately been assumed to be certain, may have been formed in the bone marrow. But, at all events, the production of the polynuclear cells in the spleen and in the lymph glands, that of the lymphocytes in the bone marrow, occurs under normal circumstances in a very secondary measure, compared to the formation of the polynuclear cells in the bone marrow—and of the lymphocytes in the spleen and lymph glands.

What is particularly conspicuous in the microscopic examination of leucæmic blood, besides the increase in the colourless cells, is that in a number of cases, and these are by far the most frequent, the polynuclear cells are in great majority; in other cases, however, the lymphocytes form the greater number, and that this type of blood alteration persists during the entire course of the illness. We therefore quite properly differentiate *two* different varieties of leucæmia.[1]

1. *Lymphocyte leucæmia.*
2. *Leucocyte leucæmia.*

As the source of the lymphocytes, as has been mentioned, is found in the spleen and the lymph glands, and that of the leucocytes in the bone

[1] In some cases the leucocytes and lymphocytes are absolutely increased, but in the manner that neither of the two forms of blood cells are specially dominating in the microscopic picture; such cases may be designated *mixed forms* of leucocyte and lymphocyte leucæmia.

marrow, the usually chosen designations of *lymphatic leucæmia* (lymphæmia), *myelogenous leucæmia* (myelæmia) could be selected for these two basic forms of leucæmia. However, it must not be forgotten that in leucocyte leucæmia, the myelogenous origin of which no one doubts, not only in the bone marrow, but also in the lymph glands, myelocytes are found, which have not wandered in at all, but which are produced in excessive amounts; and, *vice versa,* as the bone marrow normally produces typical lymphocytes, the lymphæmia of myelogenous origin may arise by these proliferative changes. In fact, there are rare cases of *lymphocyte leucæmia without enlargement of the spleen and lymph glands with lymphadenoid changes of the bone marrow,* so that the cells of the latter are prominently lymphocytes. Almost exclusively lymphocytes were found in the blood in such cases (90 per cent and over), but few polynuclears and, besides, nucleus-containing red blood cells, showing their origin from lymphocytes of the bone marrow, and also normoblasts and megaloblasts in small numbers. The old opinion of Neumann, the reformer of our knowledge regarding blood formation, that for the origin of every leucæmia, of the leucocyte as well as of the lymphocyte forms, a disease of the bone marrow is necessary, corresponds with this condition. However, it seems to me to be going too far if we look upon the markedly developed hyperplasias of the spleen and lymph glands as insignificant, which are so generally noted in lymphanæmia, as these organs are unquestionably, under normal conditions, the main points of formation of the lymphocytes. I believe, to prevent misunderstanding in designating the two chief varieties, i. e., not exclusively to connect the name of the respective leucæmia variety with the affection of a distinct organ, it would be well to adhere to the diagnosis of *lymphocyte leucæmia* or of *leucocyte leucæmia.*

In the blood at the autopsy, or upon long standing of the blood of leucæmics, *Charcot crystals* are found which usually occur within the leucocyte and particularly in the eosinophilic polynuclear cells. Their relation to the last-named cells has lately been determined beyond doubt, so that we may say: Wherever eosinophile cells are present in large amounts, Charcot crystals are also found. This at once makes it clear that they are absent in lymphocyte leucæmia, and, on the other hand, that the crystals may be found, besides in lymphocyte leucæmia, in all kinds of diseases in which eosinophiles occur in large amounts, such as nasal polypi, bronchial asthma, etc. The demonstration of Charcot crystals is, therefore, by no means pathognomonic of leucæmia, all the less so as they are also found in *normal* bone marrow, which is not to be wondered at.

In comparison to the results of the microscopic examination of the blood, all the other morbid phenomena of leucæmia, even if in the individual case they greatly assist the diagnosis, are of decidedly subordinate importance. There are cases, as has already been mentioned, of leucæmia in which, besides the alteration in the blood and the symptoms in connection with it, of anæmia, all other objectively demonstrable morbid phenomena are absent, and the diagnosis must be made almost entirely from the blood findings! These are, however, under all circumstances, as will be shown farther on, rare, exceptional cases; the rule is that the most varied organs, especially those in connection with blood formation, suffer marked changes which may be demonstrated by physical examination.

The Spleen in Leucæmia.—In this connection particularly the *enlargement of the spleen* is to be mentioned, which is present in the majority of cases of leucæmia. The size of the splenic tumour is usually a very considerable one; it reaches to the median line and beyond, downward to the hypogastric region, and, on account of the *sharp indented margin, the direction of the growth of the tumour,* especially its plain origin over the left hypochondrium, there is rarely cause to doubt that we are dealing with a tumour of the spleen. The diagnosis may become difficult if, as in one of my cases, the large spleen turns so that the hilus appears at the top. The consistence of the splenic tumour is hard; its circumference, after it has attained a certain size, is usually constant. Rarely are transitional shrinkages noted, or, on the other hand, in an excessive swelling of the spleen, rupture of the organ. The swollen spleen is not sensitive to palpation, as a rule; the subjective difficulties which the splenic tumour causes are also very slight in general. At most the patients complain of the feeling of fulness in the abdomen and of slight difficulty in respiration; less frequently do pains occur in the splenic region or even inflammatory phenomena, manifesting themselves in peritoneal friction sounds in the region of the spleen.

Enlargement of the Lymph Glands.—In a part of the cases, especially most frequently in lymphocyte leucæmia, *lymph-gland enlargement* occurs. As a rule, those of the neck and axillary region, more rarely the glands in other areas of the periphery or the internal lymph glands, are affected. The consistence of the glandular tumours is moderately hard, the skin over them is movable and not reddened. If the mesenteric and retroperitoneal glands are hyperplastic, they may be occasionally felt through the abdominal walls. If the enlargement affects the tracheal and bronchial lymph glands, a probable diagnosis of this result of leucæmia may at least be made, if the symptoms of tracheo- or bronchiostenosis, of paralysis of the vocal cords, the result of pressure upon the recurrent laryngeal nerve, or if difficulty in deglutition arise. In more decided swelling of the tracheobronchial lymph glands or of the persisting thymus gland, the note upon percussion over the manubrium sterni appears dull and the bone is arched outward. The adenoid tissue of the tonsils and also the adenoid follicles at the root of the tongue may likewise be noted to be in a condition of hyperplasia and swelling.

Changes in the Bone Marrow.—The third organ, besides the spleen and the various lymph glands and the other adenoid organs, which is almost always affected by anatomical changes, is the *bone marrow.* According to the experiences gathered up to this time, *changes in the bone marrow are never absent in leucæmia.*

Especially do we also find in *lymphocyte leucæmia,* in the lymph glands, in the spleen and eventually in the liver, but regularly also in the bone marrow, proliferations of the lymphoid tissue, which in these cases may become so plentiful that the production of the polynuclear cells in the bone marrow and the passage of the same into the blood may be prevented by the overgrowth of the lymphocytes in the marrow. As the lymphocytes passively enter the blood, i. e., float in, from the bone marrow and from the, eventually hyperplastic, lymph glands and the spleen—as we

must assume according to Ehrlich's convincing deductions, the picture of lymphocyte leucæmia develops.

The implication of the bone marrow in the leucæmic processes can never be determined with certainty, *intra vitam*, by the presence of painfulness of the bone to pressure, but from the microscopic condition of the blood this may be accomplished when mononuclear, granular marrow cells, the origin of which from the bone marrow is certain, and, besides, also large quantities of nucleated red blood cells or even myeloblasts, are found in the circulating blood.

Besides the above-named three organs which are particularly affected in leucæmia, other portions of the body, although less frequently, yet in quite a number of cases, are involved in changes which are in direct connection with leucæmia. *The liver*, in consequence of leucæmic-cell infiltration between the acini, is *enlarged in the majority of cases*; the surface is smooth, its consistence moderately hard. More marked grades of ascites and jaundice may only be expected if the periportal lymph glands are enlarged, pressing upon the portal vein and biliary passages. Ascites, respectively the transudation of a larger amount of fluid into the peritoneal cavity, may also occur by a leucæmic nodular infiltration of the peritoneal leaves and of the omentum. This is, however, not frequent.

Leucæmic infiltrations occur also in the *stomach and intestine*; they arise from adenoid tissue areas of the wall (i. e., the solitary follicles). Dyspeptic phenomena and diarrhœa point to this complication, they do not, however, permit of a certain diagnosis. In some patients there develops a leucæmic stomatitis or pharyngitis.

It is noteworthy that *dyspnœa* or even *orthopnœa* does not occur in leucæmia, at least not according to my experience (provided there are no complications on the part of the respiratory organs), in spite of the diminution of the erythrocytes; Pettenkofer and Voit were the first to show in one of their patients, who was suffering from leucæmia, that he was able, during *rest*, regardless of the diminution of the erythrocytes, to assimilate as much oxygen as a healthy person with the same nourishment. Neither do marked disturbances of the metabolism occur in the course of leucæmia, or, if they are now and then noted, they are at least not dependent upon the leucæmic processes as such. The tendency of the leucæmic to *catarrhal affections of the respiratory passages* may favour the production of pulmonary inflammation in some cases; in other cases lymphatic infiltrations form in the lungs. The development of lymphatic nodules has been noted in the epiglottis, in the larynx and in the trachea, also upon the pleura; not rarely do transudations appear in the pleural cavity.

Painful non-ulcerating nodes occasionally develop in the *skin*, and must be looked upon as leucæmic lymphoid formations. A tendency to profuse *sweating* is not rare, aiding in exhausting the patients.

The murmurs noted upon auscultation of the heart are of an accidental nature, the result of the markedly developed *anæmia* which occurs in leucæmia. This gives rise to a large variety of symptoms which may be noted more or less well developed in the picture of leucæmia: Cardiac palpitation, weakness and lassitude, vertigo, headache, and attacks of syncope, œdema, perhaps also a *fever* which is irregular and which at times may be quite high (about 102° F. to 104° F.).

Long-continued *priapism* occurs in rare cases in the course of leucæmia the production of which perhaps is due to the thrombosis in the corpora cavernosa. This symptom has been observed upon various occasions and occurs so rarely in other conditions that its presence should be considered suggestive of leucæmia.

Changes in the Eye-Ground.—Of special interest in the diagnosis of leucæmia are the *changes in the eye-ground* which occur in the course of the affection. In keeping with the increased number of white corpuscles, the fundus of the eye shows an unusual pale, orange-yellow appearance, particularly of the blood column in the vessels of the retina and chorioid. However, this discoloration is not conspicuous

in all cases; it is especially absent if the amount of hæmoglobin has not been materially decreased. The so-called *retinitis leucæmica* is characterized by a high-grade tortuous condition of the veins, band-like opacity of the retina, and hæmorrhages into the same, as well as by an opacity, indistinct contours and bluish-yellow colour of the optic-nerve papilla. Not rarely are the veins accompanied with white streaks, and white areas intermixed with hæmorrhages are visible in the macula; the hæmorrhages appear round in shape, with a prominent yellowish-white centre. Occasionally there is a tendency to a high-grade development of numerous and quite large hæmorrhages which may also occur in the vitreous body, so that the ophthalmoscopic picture may resemble that of thromboses of the central vein of the retina. Such a thrombosis may occur simultaneously with the hæmorrhages around the optic nerve and in the latter itself, and may also develop in the orbital course of the optic nerve. Functional disturbances are only present to a slight extent.

Urinary Changes.—*The changes in the condition of the urine* observed in leucæmia are of great importance in a practico-diagnostic, as well as in a theoretical, respect.

Apart from the, occasionally observed, *albuminuria,* which is partially the result of the anæmia, partially the consequence of lymphomatous infiltration of the renal substance (which fact is said to be indicated by the presence of casts and of a large number of leucocytes in the sediment of the urine), a change in the excretion of the solid constituents of the urine is more or less frequently found in leucæmia. Most constantly an *absolute and relative increase of the excretion of uric acid* (up to 8 grammes per diem) has been found.

This increase of uric acid in leucæmia is certainly not the result of insufficient oxidation in the organism, as was formerly supposed. This opinion is contradicted not only by the results of the (previously mentioned) investigations of Pettenkofer and Voit, but also by the fact determined by Stadthagen, that the patient suffering from leucæmia is capable of further oxidizing in the body the sodium urate which has been administered *per os.* Neither may the superproduction of uric acid be brought in connection with the enlargement of the spleen which is commonly present in leucæmia, as patients with chronic splenic tumour excrete uric acid in normal amounts, in contrast to leucæmia with enlargement of the spleen, in which the excretion of uric acid appears to be markedly increased. As we know now that uric acid originates from the nuclein which is liberated in the decomposition of the cellular nuclei, especially by oxidation of alloxur bases which are contained in nucleinic acid, the conclusion is obvious that the increase of uric-acid excretion in the course of leucæmia may be referred to the destruction of a relatively greater number of leucocytes compared with normal conditions. However, the increased excretion of uric acid may also be an expression of the increased functions, respectively of the metabolism, of the superfluously present leucocytes, so that the observation of the increase of uric-acid excretion in the course of leucæmia is by no means a certain proof of the massive decomposition of leucocytes and their nuclei.

The excretion of *urea* usually does not differ from the normal; in two cases of severe leucæmic cachexia in my clinic Fleischer and Penzoldt noted an increase of urea excretion, respectively nitrogen excretion, i. e., in the late stages of leucæmia, similar to the condition in carcinomatous cachexia, a more marked decomposition of organic albumin with increase of the excretion of nitrogen occurs. *Hæmaturia* may also occur in the course of leucæmia.

Hæmorrhagic Diathesis.—The last-named function is in connection with the general *hæmorrhagic diathesis* of the leucæmic, one of the most

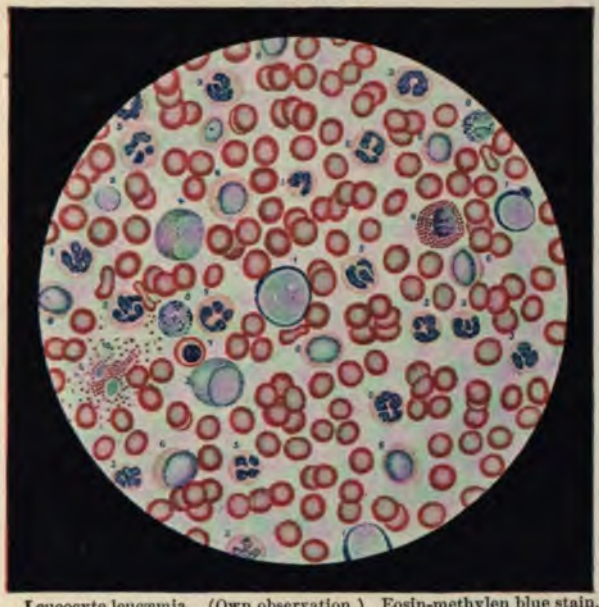
important symptoms of leucæmia in a diagnostic, but, above all, in a prognostic, respect. Hæmorrhages may also occur, besides in the urinary passages, at the most varied parts of the body: In the respiratory passages, in the digestive tract, in the skin (purpura), in the subcutaneous connective tissue (in the form of sometimes colossal hæmorrhagic tumours), in the muscles, in the internal parts of the ear, etc.; hæmorrhages are also noted in the central nervous system in the course of leucæmia, and may give rise to apoplectiform attacks with paralysis, or they may result in sudden death; these intercurrent hæmorrhages are very usually the cause of the unfortunate outcome of leucæmia, or, at least, they predispose to it. In the peripheral nervous system occasionally multiple effusions occur in the sheaths of the nerves or in the nerves themselves, which result in fatty degeneration of the nerves and muscles supplied by them. In some cases of leucæmia the hæmorrhagic diathesis may so dominate the clinical picture that the cases appear as purpura hæmorrhagica until the examination of the blood clears up the error.

The symptoms of leucæmia—the increase in the number of leucocytes in the blood cells as well as the enlargement of the spleen and lymph glands—may transitorily be greatly diminished in the course of intercurrent infectious diseases.

Genesis of Leucæmia.—The classification of individual forms of leucæmia, which has been generally in vogue until lately, according to whether the spleen or the lymph glands were enlarged in "*lienal*" and "*lymphatic*" leucæmias, can no longer be maintained from a *hæmatological* standpoint, primarily from the important labours of Neumann and Ehrlich. For some time we were obliged to differentiate not only between lienal and lymphatic leucæmias, but also between the *lieno-medullary* variety, or even between a pure medullary form as *myelogeneous* leucæmia. But even this diagnosis of the individual varieties of leucæmia, according to the changes prominent in the clinical picture, of organs which are more or less implicated in the formation of the blood, is not justified either from a hæmatological or from a clinical standpoint. For, in both forms, the lienal and the lymphatic, the bone marrow is primarily implicated in the changes of the hæmatopoietic process and, on the other hand, the characteristic blood picture, which in one case may show a marked preponderance of lymphocytes, in another a great majority of leucocytes, and their primary stages are usually not at all altered, whether the lymph glands or the spleen are enlarged or not. We therefore recur to the differentiation chosen by me of only two varieties in regard to the point of origin of the cell forms which are markedly increased in the blood—*lymphocyte leucæmia* and *leucocyte leucæmia*. The differentio-diagnostic points of these two varieties of leucæmia are the following:

1. **Lymphocyte Leucæmia** (see Fig. 71).—The blood picture is characterized by the conspicuous preponderance of large and small *lymphocytes* in comparison to the leucocytes, whereas nucleated red blood cells and megaloblasts, although they are found in the blood in lymphocyte leucæmia, yet, compared with their usually numerous occurrence in leucocyte leucæmia, they are by no means prominent. According to the course

FIG. 70.



Leucocyte leucæmia. (Own observation.) Eosin-methylen blue stain.

Unnumbered: Red Blood Corpuscles.

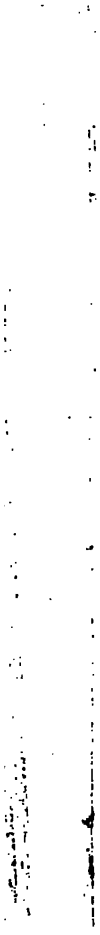
1. Large lymphocytes [1] with protoplasmic segmentation.
2. Small lymphocyte.
3. Large mononuclear leucocyte.
4. Transition cell.
5. Neutrophile (not granulated here, as stained with eosin-methylen blue) polynuclear leucocytes.
6. Neutrophile myelocytes.
7. Nucleated red blood corpuscle (normoblast).
8. Mast cells.
9. Eosinophile myelocyte.
10. Ruptured eosinophile polynuclear leucocyte.

FIG. 71.

Chronic form of lymphocyte leucæmia; in the upper half eosin-methylene blue stain, in the lower half triacid stain. (Own observation.)

Unnumbered: Red Blood Corpuscles.

1. Small lymphocytes.
2. Obliterated small lymphocytes (artifacts).
3. Large lymphocytes.
4. Neutrophile polynuclear leucocytes.
5. Eosinophile polynuclear leucocyte.
6. Nucleated red blood corpuscle = normoblast.



of the affection, two varieties must be differentiated, the acute and the chronic.

Acute leucocyte leucæmia (*"acute lymphæmia"*), the recognition of which we owe, above all, to Ebstein and A. Fraenkel, is characterized by its rapid, often febrile, course, resembling a severe infectious disease. Besides the usually quite insignificant enlargement of the spleen and lymph glands, the other clinical phenomena of leucæmia are present: Stomatitis, retinitis, etc. Most prominent in the clinical picture is the hæmorrhagic diathesis, so that the affection gives the impression of purpura hæmorrhagica and may be confused with this, all the more as the hæmorrhages occasionally occur earlier than the characteristic blood picture of acute lymphæmia. The blood picture itself is highly characteristic: Preponderance of the lymphocytes, especially of the large ones, very rarely of the small form, with a vesicle-like, occasionally deeply sinuated nucleus; erythrocytes are present, but few in numbers, the polynuclear cells are not only relatively but eventually also absolutely decreased. The number of white blood cells compared to the red ones is extraordinarily great (up to 1:1!). The affection may terminate fatally in a few days or a few weeks.

Chronic lymphocyte leucæmia is especially differentiated from the above-described form by its greatly protracted course: Gradual enlargement of the lymph glands, especially those of the neck, also enlargement of the spleen to a greater or less extent. In rare cases, even in a very chronic course (in one case of my observation the affection lasted at least two years), only insignificant enlargements of the glands appear to develop. The lymphoid hyperplasia of the bone marrow is not absent here, as little as in the acute form. The hæmorrhagic diathesis is also quite marked in the chronic form with the clinical phenomena of leucæmia, and the consequences of anæmia may show themselves in a great variety of symptoms, threatening life, such as dilatation and insufficiency of the heart, etc. The blood picture in the main resembles that of the acute form, i. e., mononuclear lymphocytes are most prominent, but usually the ***small*** forms, whereas the other colourless blood cells are almost entirely absent. ***Large*** lymphocytes, however, are also found in chronic lymphocyte leucæmia during the entire course of the affection, and in rare cases, as in the acute form, they may even dominate the blood picture.

2. **Leucocyte leucæmia** (Fig. 71), the *by far more frequent* form of leucæmia, may be easily differentiated from both last-mentioned forms of leucæmia by its entirely different blood picture. The increase of the white blood cells is usually a very marked one; here, however, the polymorphonuclear *leucocytes* present themselves as greatly increased in the microscopic picture: *Neutrophiles* and, above all, also *eosinophilic* polynuclear cells, which, as Ehrlich has found, are always *absolutely increased.* Besides, there is constantly an *absolute increase* of the *Mast Zellen,* which occasionally are twice as numerous as the eosinophiles, and their determination on this account is of great importance in a diagnostic respect, because a marked increase of the mast cells is only observed in leucæmia. The phenomenon, however, which especially characterizes leucocyte leucæmia, showing its origin as due to changes in the bone marrow (*" myelogeneous*

leucæmia"), is that, besides the polynuclear cells, also their primary stages, the *mononuclear* granular leucocytes, i. e., the neutrophilic and eosinophilic myelocytes, are regularly found in the blood and occasionally are present in such great numbers (up to 100,000 per cmm. and above), that at first sight the blood picture of acute lymphæmia with its large mononuclear cells may be simulated; some of the myelocytes show, as has already been mentioned, coarse eosinophilic granules (eosinophilic myelocytes). The cells which have been described, the polynuclear cells as well as the mononuclear, are occasionally large, at other times conspicuously small ("dwarf forms"). Besides these immature dwarf leucocytes, the myelocytes which normally are not met with in the circulating blood, other immature forms of erythrocytes which also originate in the bone marrow are found in the circulating blood stream of patients with leucocyte leucæmia, such as *normoblasts,* more rarely *megaloblasts* in varying amounts, at times even in great numbers.

The enlargement of the spleen in this variety is especially well marked, the enlargement of the lymph glands is quite prominent sometimes, at other times not so. It is remarkable that we rarely have an opportunity to observe the development of the tumor step by step. Usually the physician sees the disease in its full intensity, so we must assume that leucocyte leucæmia rapidly reaches the acme of its development. The clinical phenomena which appear, besides the splenic tumour and the enlargement of the glands, have been fully described previously: The hæmorrhagic diathesis, the retinitis, the increase of uric acid in the urine, etc.

Ætiology of Leucæmia.—From an ætiological standpoint there is scarcely anything which is of value in the diagnosis of leucæmia. In some cases leucæmia appears to follow the infectious diseases (malaria, diphtheria, the puerperium, influenza, etc.), or it occurs in connection with trauma, concussions (perhaps especially of the bones). All of these "causes" of leucæmia, as direct agents producing the disease, are of a very questionable nature. The sensation was justified, therefore, which followed Löwit's discovery, the investigator who greatly distinguished himself also in other respects regarding the explanation of the leucæmic process, that the development of the disease was due to the presence of *amœbæ* in the blood and in the organs producing the blood cells. In leucocytes leucæmia Löwit found a form of amœba that probably increases in the blood by sporulation (hæmamœba leucæmiæ magna), in lymphococyte leucæmia another form of parasite differing from the former by its active motility (hæmamœba C. parva vivax). There is no doubt that Löwit's discovery may clear the nature of leucæmia; unfortunately, however, we cannot yet reckon with this factor, as the investigations have by no means been concluded and have given rise to many contradictions.

Nature of Leucæmia.—As previously indicated, we may probably assume that in some diseases of the blood a specifically different causative factor, which impairs the blood-forming process, is active, so that, in the main, at one time the red, at another the white, corpuscles are more affected, sometimes both simultaneously are altered in their development and in their further utilization in the economy. In *leucæmia* the specific cause concentrates its action upon the white blood corpuscles, according to the nature or the irritant at one time upon the lymphocytes, at other times upon the leucocytes. The process is probably the following: By the action of the specific cause, which is not transitory but permanent, a more marked production and emigration of the white blood cells is incited and with this not only matured forms, but also, in one case less in another case more, *immature* elements enter the blood: in leucocyte leucæmia the myelocytes, in lymphocyte leucæmia large lymphoid cells which must probably be looked upon as less mature elements not capable of

entering the blood under normal conditions. The emigration of the complete polynuclear cells (and partly perhaps also of the myelocytes) may be easily explained as a consequence of their amœboid contractility, whereas the enormous emigration of the leucocytes which are not capable of amœboid movability in lymphocyte leucæmia is apparently difficult to understand. However, there is nothing against the assumption, as I believe, that the lymphocytes, because in such cases they are produced in excessively great amounts, may also in the same manner, in large numbers, as under normal conditions, i. e., by " passive exudation," by a stronger lymph circulation, etc., enter the circulating blood, provided the processes which cause the entrance of the lymphocytes are more active, which is the case at one time to a great, at other times to a less, extent. In a number of cases of hyperplasia of the spleen and lymph glands, in spite of marked formation of leucocytes, no marked migration occurs into the blood.

This of itself leads to the discussion of the question regarding the behaviour in the leucæmic process of the various areas in which blood-cell formation takes place. It may be regarded as certain to-day that the *bone marrow* is particularly implicated in the occurrence of leucæmia, not only in leucocyte leucæmia but also in *lymphocyte leucæmia.*

We may assume that normally the lymphocytes are for the greatest part produced in the spleen and lymph glands and only to a slight extent in the bone marrow. As, however, hyperplasias of the spleen and lymph glands occur partly without (" pseudoleucæmia "), partly with, an increase of the lymphocytes in the blood (lymphocyte leucæmia) and, further on, cases of leucæmia with lymphadenoid changes of the bone marrow without enlargement of the spleen or lymph glands, we may conclude therefrom that the *implication of the bone marrow, respectively the supervention of the same to simple hyperplasia of the spleen and lymph glands, represents the most important element in the development of leucæmia* (Neumann). However, this does not explain, in my opinion, why, in hyperplasia of the spleen and lymph glands without an affection of the bone marrow, an increase of the lymphocytes in the blood may not occur. As under normal conditions the small lymphocytes which are formed in the lymph glands permanently enter the circulating blood, it is not readily seen why, with an increased production in the lymph glands and with a marked migration into the blood, they should not appear in greater numbers, and why this is always the case, on the other hand, in an implication of the bone marrow. The explanation that in hyperplasia of the tissue of the spleen and lymph glands their capsule also dilates and so, in contrast to the conditions in hyperplasia of the bone marrow, no *mechanical* migration of the lymph cells into the blood occurs, is contradicted by the reflection that in such an instance every disturbance in the expansile power of the capsule should result in a marked migration of lymph cells into the blood. In my opinion, it is better for the present not to bring pseudoleucæmia (see below) into connection with leucæmia at all, and also not to look for the nature of leucæmia *only* in the *increase* of the white blood cells.

We cannot omit the question, as I believe, what becomes of the white blood cells which are so enormously increased in the organism in leucæmia. That, under normal circumstances, they are partly excreted, partly perish in the internal parts of the body, is not doubted. If, now, with the enormous increase in the white cells in the blood of leucæmics, the consumption does not go hand in hand with the increased production, a surplus of the white cells in the blood must continue. But there is no cogent proof as yet for the assumption of such an increase in consumption, which in this instance must be enormous.

My conception of the pathogenesis of leucæmia is, therefore, the following: *The action of a specific cause incites a pathological, increased irritative growth in the hæmatopoietic tissues of the body, and especially in the production of white blood corpuscles. As a result of this, a flooding of the blood, at one time with lymphocytes, at another time with leucocytes, with mature and immature forms of the same, occurs. This change*

of the blood is prominently continued, partly by the continuous action of this pathologic irritation upon the blood-forming organs, partly by an insufficient use of the superfluously produced white blood cells in the economy of the body.

Two diseases, respectively morbid conditions, are to be described in connection with leucæmia, which are in a certain external connection with leucæmia, *leucocytosis* and *pseudoleucæmia.*

LEUCOCYTOSIS

In contrast to leucæmia, which is a genuine affection of the blood, with a severe, progressive course, we understand by leucocytosis a transitory, certainly not in itself pernicious, alteration of the blood, which has in common with leucæmia an increase of the white blood cells, in which, however, we are not dealing with a ***morbis sui generis,*** but only with a *symptomatic* phenomenon, occurring in the course of various affections.

In some physiological processes, viz., during digestion, pregnancy, after bodily exertion, etc., we find, upon examination of the blood, an increase of the white cells. In this "*physiological*" leucocytosis the proportions of the various forms of leucocytes to one another do not vary greatly from the normal (whereas it is characteristic of *pathological* leucocytosis that, according to the nature of the disease in question, in the course of which a leucocytosis appears, marked alterations in the proportionate numbers of the various leucocyte forms are noted. According to whether the lymphocytes or the leucocytes are individually increased, we speak of "*lymphocytosis*" or "leucocytosis" in a restricted sense, and in the latter we may still, according to the prominence of one or the other leucocyte form, especially differentiate in the blood picture between a polynuclear *neutrophilic,* and an *eosinophilic,* leucocytosis.

Lymphocytosis.—It has been emphasized at various times that the ***lymphocytes*** have no substantive active movability; if, therefore, in the course of pathological conditions a (symptomatic) one-sided increase of lymphocytes occurs in the blood, it must be estimated as a result of this that large numbers of them have reached the circulating blood by a marked cell production in the cytogenic tissue of the lymph glands, of the spleen or of the bone marrow (here, however, in a very subordinate measure) and by a more active lymph circulation ("passive leucocytosis"). Ehrlich. This form of leucocytosis, lymphocytosis, is quite rare, occurring especially in catarrhal affections of the stomach and intestines of nurslings, in pertussis, after injections of pilocarpine and after extirpation of the spleen.

Polynuclear Leucocytosis.—All the more frequent is the second form of leucocytosis, *polynuclear leucocytosis.* Various irritations (infectious agents, chemical poisons, etc.) cause these leucocytes, which are always present in great amounts in the bone marrow and which, on account of their amœboid movability, are always ready to pass into the blood, to emigrate, and they enter the blood by "chemotaxis" ("*active*" leucocytosis). As a rule, the neutrophilic polynuclear leucocytes are met with circulating in large numbers in the blood in leucocytosis; i. e., almost all *intoxications and infections* lead to this "usual" form of leucocytosis. These are: Poisoning by potassium chlorate, phenacetine, oil of turpentine, albumoses, arsenic, etc.; further, cachectic conditions, as a result of malignant tumours, phthisis, severe blood loss, and, finally and above all, the various infectious diseases—pneumonia, sepsis, diphtheria, erysipelas, etc. The stronger or the milder degree of the leucocytosis in the course of the infectious diseases depends upon the virulence of the infective substance, the property of resistance of the individual, above all, however, also upon the specific nature of the originator of the disease. Whereas, for example, croupous pneumonia gives rise to a specially uniform and marked leucocytosis, this condition is entirely absent in enteric fever and in the eruptive stage of measles, in which affections, on the contrary, we note a diminution in the number of white blood cells (at the expense of the polynuclear), a hypo-leucocytosis, a so-called "leucopenia."

In the usual form of polynuclear leucocytosis the number of eosinophiles in the blood is so greatly diminished that they may not be observed at all; we may assume, therefore, that the irritative substances previously mentioned have no affinity for the eosinophiles but, on the contrary, have a repelling action upon them. On the other hand, in certain pathological conditions we note a one-sided increase of the polynuclear eosinophilic cells (*eosinophilic leucocytosis*). This is the case in bronchial asthma, in various diseases of the skin, in malignant tumours and especially also in helminthiasis (due to the presence of teniæ, ascarides, ankylostoma, etc., in the intestine). We will not go wrong if we assume that in these pathological conditions materials develop which chemotactically preferably act upon the eosinophiles, giving rise to a more active emigration of the same into the blood and, if the irritation be continued longer, cause their more copious production in the bone marrow.

In contrast to leucæmia, the nature of which, as we have seen, depends upon a pathological *blood formation*, i. e., not only upon a successive increase of the white blood cells but also upon a *qualitative* change of the same (the passage of immature forms into the blood), in leucocytosis the condition is only a functional alteration in the blood-forming organs, and in the majority of cases (leucocytosis in the restricted sense) exclusively of the *bone marrow*, in that the latter reacts to pathological irritations with a more marked formation and exit of normal and mature colourless blood corpuscles. This explanation of leucocytosis, given in its strict conception by Ehrlich, as an increased function of the bone marrow, has lately been fully confirmed experimentally by Rubinstein. He was able, after the injection of an agent causing leucocytosis, to follow step by step the changes in the bone marrow. As soon as the leucocytes appeared in the blood in large amounts, the granular mature elements in the bone marrow decreased almost to complete absence, and, instead of these, in a few days, the immature forms, especially the myelocytes, increased. The latter, therefore, covered the deficit due to the enormous emigration of the polynuclears into the blood as a result of the leucocytosis. The spleen and the lymph glands, however, in Rubinstein's experiments, did not take part in the substitution of the white corpuscles in polynuclear leucocytosis, which is not to be wondered at, according to our present opinions regarding the separate development of leucocytes and lymphocytes.

Differential Diagnosis.—*Regarding the clinical differentio-diagnostic division of leucocytosis from leucæmia*, this is not difficult if the points are observed which were emphasized in describing leucæmia, and also those which were given in connection with the abnormal production of leucocytes. The number of white blood corpuscles in the blood picture usually allows no doubt as to whether the one or the other pathological condition is present, as in leucæmia, almost always, an enormous increase of the white cells compared with the erythrocytes is present (1: 10; 1:2, etc.), which is never the case in simple leucocytosis. However, upon a mere count of the white and red blood corpuscles a differential diagnosis cannot be made with certainty, as especially in anæmic conditions, particularly in secondary anæmia (p. 780), a leucocytosis develops, and then, with diminution in the number of reds and a simultaneous increase of the white blood corpuscles, a proportion of numbers is found (for example, in a case of carcinoma ventriculi by Strauss, 1: 20), which would erroneously place the diagnosis into the frame of leucæmia. More important and alone conclusive for the diagnosis of polynuclear leucocytosis is the microscopic condition of the white blood corpuscles circulating in the blood in the individual case. Whereas, in polynuclear leucocytosis, only normal elements circulate in the blood and myelocytes are found only in exceptional cases (in severe processes, going hand in hand with a too rapid new formation of blood cells), in the blood of leucæmics we note regularly, besides the neutrophilic polynuclear cells, large and small myelocytes, also eosinophile and mast cells, both in absolutely increased numbers, and, furthermore, normoblasts and occasionally even megaloblasts; i. e., the blood picture is characterized by a certain polymorphic condition of the blood elements which is not so prominent in leucocytosis. More difficult, even impossible, is it from the blood picture alone to differentiate a *lymphocytosis* from a lymphocyte leucæmia. Here, the circumstance is of value in the diagnosis that leucæmia, as a rule, is a chronic, pernicious affection, whereas leucocytosis is usually an acute,

transitory, non-progressive condition and in all instances is only a symptom of another affection, the determination of which at once decides the diagnosis of leucocytosis.

PSEUDOLEUCÆMIA—HODGKIN'S DISEASE—LYMPHADENIA

This designation has been chosen for cases of multiple lymph-gland enlargement, which on microscopic examination have proven to be hyperplasia of the adenoid tissue, the process not infrequently being combined with enlargement of the spleen; therefore, similar to leucæmia, a hyperplasia of both these blood-producing organs is present, without an increase of the white cells occurring in the blood during the entire course of the affection. At first, usually the lymphatic glands of the neck enlarge, to which there is soon added intumescence of the axillary and inguinal glands and of the internal lymph glands also. Besides, enlargement of the spleen may also open the scene and, with the anæmia which goes hand in hand with it, may be the only demonstrable morbid phenomenon (*"anæmia splenica"*); the splenic tumour is later supervened, eventually, by enlargement of the lymph glands and frequently also by increase in the size of the liver. Hyperplastic changes which are more or less marked may occur in the course of the affection in the entire lymphatic apparatus, and heteroplastic formation of lymphomata may appear in various parts of the body. The pressure phenomena due to this condition naturally vary in each individual case, at one time it is a question of œdema or ascites, at other times of jaundice, bronchostenosis, paralysis of the recurrent laryngeal nerve, etc.

Common to all cases of "pseudoleucæmia" is the early developing, *steadily progressive anæmia* with its resulting severe consequences; there may arise, particularly, a *hæmorrhagic diathesis* (purpura, etc.) in the course of pseudoleucæmia, even if this condition is not so frequent nor develops to the same extent as in the case of leucæmia. The microscopic examination of the blood shows a diminution in the number of the red blood corpuscles (one half of the normal number and less), in the later stages occasionally poikilocytosis, a diminution in the hæmoglobin corresponding to the decrease in the number of erythrocytes, and the other characteristics usual to the blood picture of severe anæmia. *The white blood corpuscles, on the other hand, are not increased,* and if now and then a greater number of leucocytes, especially, recently, a *relative* increase of the lymphocytes, has been found, the blood picture, in my opinion, cannot be counted as belonging to leucæmia; the excretion of uric acid in the urine is also *not* increased. In some of the cases the disease runs its course with *fever*, which shows a peculiar type in which febrile periods alternate with afebrile ones ("chronic relapsing fever"). The cause of this fever is unknown, the separation of such cases from those that run their course without fever does not appear wise to me, especially as in anæmia, and also in leucæmia, fever occurs intercurrently without our being able to find the cause of it and to separate these cases fundamentally from those running an afebrile course.

The relation of pseudoleucæmia to leucocæmia has not been cleared up as yet. It is a fact that, in some cases of pseudoleucæmia, shortly before death, the affected patients show a rapid increase of the white blood cells in the blood; pseudoleucæmia has even, very exceptionally, merged into a chronic lymphocyte leucæmia. But in by far the great majority of cases of "pseudoleucæmia" the most marked hyperplasia of the adenoid tissue is present with enormous enlargement of the lymph glands and of the spleen, without an increase in the white blood cells occurring even in a very protracted course of the affection.

Why this is the case, and why, on the other hand, a number of the cases of leucæmia, with relatively slight alterations of the blood-forming organs, show the most pronounced type of the leucæmic blood condition, cannot be satisfactorily explained at present. The most plausible conception is that of Neumann, *that the predisposing condition for the occurrence of the leucæmic state of the blood is a hyperplastic proliferation of the bone, and that, so long as the spleen and the lymph glands are affected by the pathological conditions alone, merely pseudoleucæmia occurs.* I have previously explained that there are certain objections even to this explanation, and, as a transformation of pseudoleucæmia into leucæmia is extraordinarily rare, it appears to me, for the present at least, to be better not to bring pseudoleucæmia and leucæmia into genetic connection, and also, in making a diagnosis, not to use the term pseudoleucæmia, i. e., rather to choose, instead, "multiple lymphomata," "hyperplasia of the spleen with severe anæmia," and similar non-prejudicing designations. This is all the more to be advised as then the difficulty is removed to designate which grade of splenic or glandular hyperplasia is to be looked upon as pseudoleucæmia, and we are not forced, as lately occurred, to bring other processes, such as multiple lymphosarcoma or tuberculosis of the glands, in connection with pseudoleucæmia.

LEUCANÆMIA

Severe blood affections with disturbances in the production of the white, as well as of the red, blood corpuscles. Mixed forms of pernicious anæmia and leucæmia.

I understand by this: Severe affections of the blood in which the production of white and red blood corpuscles is simultaneously damaged. In the blood picture marked alterations then arise in the leucocytes and erythrocytes, similar to those we have noted in pernicious anæmia and in leucæmia. The blood formation may cease entirely in such cases, and this defect may be noted in all formations so that the organism in the briefest period may succumb to the pernicious blood affection. The following case which was noted in my clinic shall be described as an example:

Illustrative Case of Leucanæmia.—A boy, aged ten, entered the hospital May 6, died May 9, 1900. Patient is said to have been always weak, but never seriously ill, and attended school regularly up to April 22. Mild symptoms, vomiting twice so that the patient was compelled to remain at home from April 22 to April 29. On April 29 he still sang as a chorus boy in church, and on May 1 again attended school, but on May 5, on account of his pallor, which the teacher noticed, he was sent home. From May 3 to May 6 he still partook of his food with appetite; then, four days before his death, epistaxis occurred, with yellow discoloration of the

skin; pain in the nape of the neck and toothache appeared, as well as pain in the epigastrium. In the course of the day severe apathy became more and more noticeable, increasing to complete loss of consciousness, so that the patient was brought to the Julius Hospital by his relatives.

Status præsens: The appearance of the patient denoted serious illness; skin and mucous membranes deathly pale; eyelids and hands markedly œdematous; the skin over the rest of the body slightly bloated, showing yellow discoloration but not jaundice; bulbar conjunctivæ snow-white. Coma. No opisthotonus, no rigidity of the neck and no spasms. Pupils dilated, reacting normally. Liver and spleen moderately enlarged and hard, sensitive to pressure. The musculature of the calves of the legs not especially sensitive to pressure, but the bones of the thigh as well as the sternum extraordinarily painful. Lungs normal, the cardiac dulness extended to the right sternal border. Heart sounds clear. Gallop rhythm, undulation of the veins of the neck. Urine contained traces of albumin; no albumoses, no casts. The ophthalmoscopic examination showed feeble filling of the vessels, especially of the arteries, which were partly thready—"marantic thrombosis of the central artery with numerous disseminated hæmorrhages." Temperature 102° F. to 104° F.

As the symptoms of extraordinary impoverishment of the blood were present, besides a slight enlargement of the liver and spleen, the diagnosis was restricted to a "severe anæmia." Regarding the cause of the same, on account of the œdema of the skin and presence of albumin in the urine, a nephritis was first to be thought of. This assumption, however, was soon dropped, when it was found that only traces of albumin, but neither blood cells nor epithelium, respectively casts, were present. Much more likely was an acute infection, in favour of which was the rapid course of the disease, the severe disturbances of the general condition, the high fever, enlargement of liver and spleen and the albuminuria. However, the pathological picture did not correspond to any of the usual infectious diseases. It was obvious that the exact examination of the blood would point the way diagnostically to clear this difficult case.

The blood count showed an exorbitant diminution in the number of erythrocytes: 256,000 in a cmm.! The blood corpuscles varied in size (also megalocytes), and the triacid stain showed corpuscles some of which also contained nuclei: Normoblasts 76, megaloblasts 152 in a cmm. The amount of hæmoglobin of the entire blood was not more than 10 per cent, the hæmoglobin contents of the individual blood corpuscles [colour index] was therefore relatively increased.

The absolute number of white blood cells was scarcely increased: 10,600; however, compared to the total number of the erythrocytes it was markedly higher, 1:24. The form of the leucocytes and proportion of the individual leucocyte varieties among each other varied greatly from normal conditions—the blood picture resembled that of leucæmic blood: Besides the usual, neutrophilic, polynuclear leucocytes, which were present in diminished amounts (4,680), many myelocytes were present (1,380 neutrophiles and 76 eosinophiles); mast cells were absent; however, there were large mononuclear leucocytes (228) and conspicuously numerous lymphocytes (4,260), large and small—almost as many as polynuclear cells. Expressed in percentages the blood contained: Lymphocytes 40.2 per cent (large 35.3 per cent, small 4.9 per cent); polynuclears (neutrophiles) 44.1 per cent; myelocytes 13.6 per cent (neutrophiles 13 per cent, eosinophiles 0.6 per cent); large mononuclears 2.1 per cent; and but very few mast cells.

From these findings a severe damage to the blood-forming process was present without doubt, and the diagnosis was made of severe pernicious anæmia with the usual disturbances in the erythrocyte production and a simultaneous, conspicuously severe, alteration in the production of white corpuscles.

The course of the affection during the four days in which the patient remained in the hospital, was an extraordinarily rapid one. The temperature, which varied between 102° and 104° F. from the first to the second day, dropped on the third day to 100° F., falling upon the fourth day, up to the time when death occurred, to 97.8° F.; consciousness, which had been lost prior to his admission to the hospital, returned on the second day and remained up to the time of death. The energy of the heart, however, weakened from hour to hour. The cardiac boundaries which already

were diffused at the onset, increased more and more without murmurs appearing in the heart. With increasing weakness, death occurred on May 9.

The clinical diagnosis, which was entered at the pathological institute, was: "Progressive pernicious anæmia, enlargement of the liver and spleen, infectious degenerative processes in the bone marrow, with disturbances in the formation of red and white blood corpuscles; dilatation of the heart, myocarditis (?)."

The anatomical diagnosis (v. Rindfleisch) was as follows: *Anæmia maxima corporis totius; Hyperæmia ossium cum infiltratione cellulari partim leukocytica partim erythrocytica. Lien hyperplasticus ex intumescentia leukocytica corpusculorum Malpighii. Hepar modice auctum degeneratio hepatis amyloidea. Degeneratio cordis adiposa præsertim ventriculi sinistri, dilatatio ventric. sinistri. Myocarditis. Œdema pulmonum, atelectasis.*

The marrow of the femur in its lower half was markedly red, that of the sternum reddish; spleen slightly enlarged, (13.8; 5.0; 3.25) no enlargement of the lymph glands. The microscopic examination revealed amyloid of the liver cells; no *siderosis* of the liver, fatty degeneration of the heart muscle, with hæmorrhages the size of a millimetre; kidneys only anæmic.

Epicrisis.—If the clinical diagnosis is compared with the anatomical findings, in the main it agrees with the autopsy, but the explanation of the case in its individual features was difficult. The blood findings, in so far as the red blood corpuscles were concerned (76 normoblasts, 152 megaloblasts in a cmm.), would place the affection in the category of pernicious anæmia. Numerous normoblasts and megaloblasts were circulating in the blood, whereas the non-nucleated disks were diminished to an extraordinarily slight amount, to about 250,000 in a cmm. Also, that the erythrocytes showed themselves relatively rich in hæmoglobin, is in keeping with the usual findings in pernicious anæmia.

More important than the condition of the red, is that of the white, corpuscles in the case in question. We note point for point the findings to correspond to leucæmic blood, without there being any absolute increase worth mentioning in the white blood cells.

Primarily the great number of myelocytes in the blood (and also in the bone marrow) is conspicuous. They form one seventh of all the leucocytes! It can hardly be disputed that in our case the transportation of the immature leucocytes, the mononuclear neutrophilic and eosinophilic myelocytes, represents an analogous process in the blood as the emigration of the immature erythrocytes, the normoblasts and the megaloblasts, into the blood. This analogy is so natural that the question may well arise whether this condition—emigration of immature leucocytes with the simultaneous emigration of the immature erythrocytes from the bone marrow into the blood—does not occur more frequently in pernicious anæmia, even whether or not the condition occurs regularly. To decide this question, only the latest investigations regarding this point, which are based upon the methods of staining that are in use to-day, should be utilized. From the investigations which are determining in this respect, those of Lazarus, it is shown that the appearance of myelocytes in the blood of patients suffering from pernicious anæmia is not usual, rather that none is found, or apparently but few (at the utmost up to 0.5 per cent), of these immature leucocytes. In our case, however, the blood was rich in myelocytes—they represented the seventh part (!) of the white blood cells and the one-hundred-and-eighty-sixth part of all the blood cells. We must therefore assume that in this rare case *we were dealing with an insufficiency in the function of the bone marrow, affecting simultaneously the formation of the red as well as of the white cells,* so that the maturing of the embryonic forms of the red and white cells did not occur, but rather the emigration of the same, i. e., of the (nucleated) normoblasts and megaloblasts as well as of the myelocytes into the blood, took place before their transformation into normal, finished blood cells could be completed.

It is surprising that, besides the—neutrophilic or eosinophilic—as is well known, granular myelocytes, also non-granular myelocytes and even, what is more conspicuous, non-granular polynuclear cells could also with certainty be demonstrated in the circulating blood—a fact that perhaps may be explained in the manner that in this rapidly running reduction of the hæmatopoietic process not even the granulation

could take place, much less the complete maturing of the leucocyte forms which were produced in the bone marrow.

Noteworthy also is the relative *increase of the lymphocytes*, the number of which closely approached the polynuclear leucocytes (similar to the conditions occurring in pernicious anæmia, Strauss). It would be plain to suppose the increase of lymphocytes in the blood to be due to a disturbance of the formative process of the blood cells in the bone marrow; this, however, is not possible in our case, in my opinion, because there were decidedly too few lymphocytes in the bone marrow, whereas, in contrast to this, fluid taken from the tissue of the spleen, one hour after death, showed great numbers of small (10.8 per cent) and large (9.4 per cent) lymph cells, so that any other assumption than to ascribe the lymph-cell increase in the blood to some marked production of these cells in the spleen, appears to me to be a forced one, all the more so as the lymph glands in this case proved to be unchanged all over the body.

It would be quite obvious to represent the case as one of pernicious anæmia, on account of the changes in the erythrocytes in the blood. Then, however, we should assume at once that this is an unusual case of pernicious anæmia, without siderosis of the liver, that an important constituent of this affection, therefore, is absent; just as conspicuous would be the sparse presence of megaloblasts in the bone marrow, and that myelocyte production and transportation into the blood should have taken on such unusually great dimensions. The same is also true of the assumption of an incipient myelogenous leucæmia. We should then be dealing with a leucæmia in which an absolute increase of the white blood cells would be absent and mast cells would scarcely be demonstrated at all, and, further, that the eosinophile cells not even reached the number of 100 in a cmm. and that Charcot crystals were absent in the bone marrow, facts which are very difficult to reconcile with an assumption of a leucæmia which in this case certainly originated in the bone marrow.

We may therefore only say that in our case we were dealing with *a severe, perhaps infectious, disturbance of the process of formation of the blood cells in the bone marrow, which affected the red, as well as the white, blood corpuscles, having the result that both remained in an immature condition, the white cells even not being entirely granular, and that this reduction of the function of the bone marrow in a few days led to the complete destruction of blood formation, this leading to the cessation of the vitality of the organism.*

This minutely described case accordingly belongs to the category of severe blood diseases which we have designated "leucanæmia," in which a severe alteration of the hæmatopoietic process affects uniformly the red and white blood corpuscles, in the sense of the blood alterations noted in pernicious anæmia and leucæmia, showing transitory forms of both of these blood affections. Such mixed forms have been noted now and then, and it will require a further collection of such cases to determine and designate a distinct clinical picture for this severe form of blood affections. The classification of the individual case will even then, under some circumstances, be difficult and should occur *"a potiori."* We must not forget that in ordinary leucæmia not only normoblasts, but even, although rarer, megaloblasts were found in the blood, and that in severe anæmia, apart from the changes in the forms of leucocytes, even isolated myelocytes were occasionally met with in the blood. But in both cases these findings are of an unessential nature, only then of importance for a nosological conception of the individual case as leucanæmia when the pathological changes of the cells of the blood simultaneously affect the leucocytes and erythrocytes in a marked manner.

The category of leucanæmia embraces probably, besides the transitional

forms between pernicious anæmia and leucæmia, also certain cases of so-called "*anæmia pseudoleucæmica infantum*," in which a marked increase of the white cells, myelocytes, megalocytes, and normoblasts, was noted, and conjointly always a diminution of the red and white cells and of the hæmoglobin, much more marked than is ever the case in leucæmia.

HÆMORRHAGIC DIATHESIS—PURPURA HÆMORRHAGICA, PELIOSIS RHEUMATICA, MORBUS MACULOSUS WERLHOFII, SCURVY, HÆMOPHILIA

The affections to be considered in this chapter form, in a diagnostico-pathogenetic connection, *one* morbid group. Hæmophilia is exceptionally placed in this category. This affection, in contrast to the other diseases belonging to it, represents a congenital, permanent malady, *transmitted* by women from families of bleeders, and, as a rule, only affecting the male descendants. We shall first consider the diagnosis of this affection.

Hæmophilia.—The pathognomonic signs of *hæmophilia* are hæmorrhages which are exceedingly difficult to control, which occur from the slightest external causes (rarely spontaneously) at the most varied parts of the body: In the skin, the mucous membranes (especially as epistaxis), and in the internal organs (stomach, intestines, kidneys, etc.). Hæmorrhages also occur often into the joints, with swelling and immovability of the same. Hæmophilia may manifest itself in women in profuse menstrual periods which last a remarkably long time. As a result of the hæmorrhages, the symptoms of anæmia develop secondarily: Pallor, cardiac palpitation, anæmic murmurs in the heart, etc.; nervous phenomena have also been noted in particular (headache, insomnia, psychical disturbances, etc.), as well as *arthritic affections*, which are perhaps the result of repeated hæmorrhages which have occurred in the deeper tissues of the joints, and which, later, may take on the appearance of arthritis deformans. *Urinary analyses* which are of value, do not exist; albuminuria was noted in a number of the cases, whereas it was absent in other cases of most intense hæmophilia. The *renal hæmorrhages* which occur upon the basis of hæmophilia, may in some cases assume a certain individuality ("renal hæmophilia," Senator).

The examination of the blood has as yet not shown any constant results. It is certain that there is no diminution of the erythrocytes; on the contrary, several observers have shown an unusual abundance of erythrocytes, compared to the few white blood cells present. Competent investigators have further shown a *diminution in the coagulability of the blood;* this manifests itself especially, in contrast to normal conditions, if large amounts of blood were lost.

All other theories proposed to explain the origin of hæmophilia are of but slight value, such as the assumption of superficial positions and thinness, and insufficient elasticity of the cutaneous vessels, or of a relative plethora and blood hyperplasia, in comparison to the extraordinary narrowness of the arteries; further, the supposition of an abnormal vascular innervation or, finally, an increased production of red blood cells ("erythrocythæmia"). None of these opinions has been ascertained by anatomical findings; and the pathogenesis offers absolutely nothing that is of value for the diagnosis of hæmophilia.

Common to those morbid conditions which have been subsumed under the name of hæmorrhagic diathesis, is the *tendency to spontaneous exit of blood into the tissues and to the free surfaces of the skin and mucous membranes.* In the mildest forms only *hæmorrhagic areas become manifest upon the skin, which do not disappear upon pressure*; they are more fre-

quently found upon the extensor than upon the flexor surfaces, with special preference for the lower extremities (so-called *purpura simplex*); rarely are these hæmorrhagic areas combined with an œdematous prominence of the skin (*purpura urticans*). Associated with these are very commonly *pains and swellings of the joints* (*purpura or peliosis rheumatica*); in the severe forms *hæmorrhages of the mucous membranes,* of the nose, mouth, respiratory and intestinal tracts, etc., occur (so-called *purpura hæmorrhagica,* morbus maculosus Wehrlhofii). Hæmorrhages from the gums may also complicate the picture; this is, however, not the rule. On the other hand, frequently retinal hæmorrhages, hæmorrhages from the kidneys and brain, bleeding and inflammation in the serous cavities, and also endocarditis may often associate themselves with the harmless external and mucous-membrane hæmorrhages, so that the clinical picture may become manifold and the course of the affection dangerous. In some of the cases we note the affection accompanied with high fever, up to 104° F.; in other cases fever is entirely absent. The course of the disease is *chronic* in the majority of cases; in some cases, however, an actually fulminant one, resembling an acute infectious disease, so that death may occur in a few days; this form is noted especially in children and is designated by a special name, *purpura fulminans* (Henoch).

Purpura Fulminans.—The characteristic of this very acute purpura form, according to the cases that have been observed up to now, consists in the fact that the hæmorrhages are limited exclusively to the skin, forming confluent ecchymoses and hard infiltrations of an enormous extent; eventually they may cause the formation of hæmorrhagic vesicles, whereas the internal organs have always shown themselves to be normal at the autopsies, as well as the condition of the urine and of the fæces. In some cases there is no rise in temperature, in others high fever is noted. The ætiology of purpura fulminans has not been cleared up as yet; it has occurred in connection with pneumonia and scarlet fever, but has also occurred in children who were entirely healthy.

Scurvy.—The symptoms of the cases of the hæmorrhagic diathesis that have been specially designated as scurvy (*scorbut*) and which were usually diagnosticated as such, do *not* differ in their nature, in my opinion and experience, from purpura, as all phenomena which were considered characteristic of scurvy, are occasionally also noted in purpura, such as the hæmorrhages into the deeper soft tissues, into the subcutaneous tissues and the muscles (most frequently into the popliteal space, into the muscles of the calves of the legs, the glutei and abdominal muscles, eventually with perforation of the skin, inflammations, suppurations, and ulcerations), as well as an affection of the gums which is regarded as especially characteristic. The latter is also noted by livid swellings which bleed readily, by looseness and ulceration of the gums adjacent to the teeth (especially those parts lying *between* the teeth), which, under some circumstances, may become necrotic and decompose into a dark, decaying mass. Rarely does the scorbutic process develop more posteriorly in the mouth, upon the mucous membrane of the lips, cheeks and pharynx, forming proliferations and hæmorrhagic ulcers. The secondary (hæmorrhagic) inflammations (pleurisy, pericarditis, endocarditis, peritonitis, nephritis), which are

frequently noted in scurvy, are also more or less often found in all the varieties of the hæmorrhagic diathesis, as well as occasional rises of temperature, joint swellings (due to serous or hæmorrhagic effusions) and internal hæmorrhages. I believe it is at present not permissible nor imperative to consider the swelling and loosening of the gums or the inflammatorily hæmorrhagic infiltrations of the muscles, etc., to be *so* specific that, in spite of the congruence of the other phenomena, an artificial separation of scurvy from the other phenomena of the hæmorrhagic diathesis would be justified. Because even physicians who distinctly differentiate scurvy diagnostically from other affections, must admit that we cannot decide in certain cases whether they belong to the category of scurvy or to chronic purpura hæmorrhagica. If we desire to retain the designation scurvy, *solely to denote the prevalence of certain symptoms in individual cases*—the affections of the gingivæ, the hæmorrhages in the deeper tissues (in the muscles and the periosteum, in the epiphyses, between the bones and cartilages, etc.)—there is nothing to prevent this, in my opinion.

Barlow's Disease.—Lately an affection resembling scurvy has been noted in children between the first and second years of life, which was designated by a special name, *Barlow's disease*. It may be that, with or without slight fever, at various portions of the body swelling and painfulness of the bones, especially of the epiphyses, occur. *Periosteal hæmorrhages* were found at the autopsy or during surgical operations in such cases, the periosteum loosened from the bones, the latter saturated with blood, and certain lamella over the bones desquamated. Besides, the clinical picture of the hæmorrhagic diathesis may also be noted (purpura, hæmaturia, etc.), and the connection between Barlow's disease and this group of affections thus becomes plainer.

It is self-evident that the various *subsequent phenomena of anæmia* may complicate the symptom-complex of the hæmorrhagic diathesis. The patients suffer from lassitude, fainting, vertigo, cardiac palpitation, anæmic heart murmurs, etc. Also albuminuria arising upon an anæmic basis is noted, more rarely as a result of hæmorrhagic nephritis.

There is no unanimity of reports regarding the number and the alterations of form of the blood corpuscles in the hæmorrhagic diathesis; nor is anything certain regarding the amount of fibrin contained in the blood, and the theory which was generally accepted, formerly, that the development of scurvy depends upon a *diminished amount of potassium in the blood*, has been proven to be incorrect upon close investigation.

Ætiological Diagnosis.—In general we are still very far from an insight into the pathogenesis and nature of the hæmorrhagic diathesis; and, still, a decision which cause of purpura is present in the individual case, is of greatest importance for the diagnosis, prognosis and therapy. The diagnosis of the existence of hæmorrhages is, of course, so very simple and easy that we may not lose a word in reference to it; all the more difficult, however, is usually the second, more important side of the diagnosis—the ætiology.

It is best in this respect to start from the following points of view: Primarily, traumatic ecchymoses, especially the stings of insects (fleas) with very dark points at which the sting has taken place, suffusions as the

result of contusion or the action of sucking are to be differentiated from purpura spots, which on sufficient observation of the anamnesis presents no difficulties, as a rule. A frequent cause of the appearance of the blood in the skin (due to diapedesis) is venous *stasis* which, if markedly developed, is sufficient to cause ecchymoses, even in an entirely normal skin; in this category belong the hæmorrhagic effusions in the lower leg, especially in the surrounding of varices, and those occurring towards the end of life due to relaxing cardiac power. The development of stasis hæmorrhages is favoured by a simultaneous morbid condition of the cutaneous vessels, as in eczema and urticaria, further in erysipelas, scarlatina and, especially, in variola. The ecchymoses which arise in the last-mentioned *infectious diseases*, and also in others (such as rheumatism, enteric fever, malaria, and, especially, in sepsis), owe their development probably partly to bacterial thrombi, partly (as hæmorrhages also surely occur without any signs of accumulation of bacteria) to a damage in resistance of the vascular wall (a necrosis) due to intoxications by the poisons produced by the bacteria demonstrated or supposed. Such cases may all the more be attributed to a chemical poisonous action, as hæmorrhages also occur in jaundice, Bright's disease and in the usual intoxications, thus in poisoning with iodine, bromine, phosphorus, etc. Why in such a general action of the poison only some portions of the body show hæmorrhages, must be explained in that especial conditions, which give rise to hæmorrhage, must also be present to prepare the soil for an action brought about in this manner, such as stasis due to local exudations or to insufficient cardiac activity (in some cases the affection arose directly after severe fright), to partial spasms, vaso-motor disturbances of innervation, etc. A damage to the nutrition and resistance of the vascular wall must also be looked upon as a cause of purpura in anæmia, leucæmia, multiple sarcomata, etc.

Essential Purpura.—Whereas the ecchymoses which have been mentioned up till now are solely symptoms of well-known affections and as such may be diagnosticated readily, nothing remains for the diagnosis in a great number of purpura affections but the assumption of an *essential* ("idiopathic") purpura, after those symptomatic purpura forms have been excluded. To determine the ætiological basis of these essential purpura forms has not been possible up to now. A great number of hypotheses have been proposed regarding the pathogenesis of the affection, but to enumerate them completely would not be worth while.

Infectiousness of Purpura.—But one of these has a certain basis, the belief of essential purpura to be due to a specific *infection*. To the correctness of this assumption points primarily the *ensemble* of the clinical phenomena, of which we shall name especially the, sometimes frequent, (epidemic) occurrence of the affection, the prodromes which frequently precede the appearance of hæmorrhages, such as lassitude, anorexia, vertigo, etc., the fever, the occasionally demonstrable enlargement of the spleen and the peracute course of individual cases. But the assumption of an infectious nature of the so-called essential purpura forms is based, above all, upon the results of the latest investigations regarding the feasibility of inoculating the disease and in reference to the presence of certain bac-

teria in the organism of purpura patients. Some time ago Petrone and others were able, by inoculating the blood of patients of febrile essential purpura conditions into animals, to produce purpura in the latter. Definite results have been gained by the investigation of the blood for bacteria (Letzerich and others), in which, however, no definite micro-organism has been found in all of the cases, but in some cocci, and in others bacilli. It is possible that various bacteria are really at the bottom of the different forms of essential purpura as the generators of the affection; if this opinion should prove to be correct, there would be more reason than we have at present, strictly to differentiate individual types of peliosis from one another.

Predisposing Factors.—But even if this end should be attained, we would still have to ascribe an important *rôle* which predisposes to the infection, to certain factors which impair the nutrition and the resistance of the general organism: Poor nutriment, or a nourishment consisting of but one kind of food (absence of fresh vegetables, with a relative amount of potassium salts, and meat, etc.), alcoholism, poor hygienic surroundings, overcrowding in tenement houses, in prisons, etc., mental and bodily exertion, etc. That such influences may favour the purpura infection in that the organism impaired thereby is more susceptible to the invasion and development of pathological bacteria, or that the development of the virulence of the latter may in part be increased outside of the organism, is perfectly in accord with our present views in reference to the process of infection in general.

HÆMOGLOBINÆMIA—HÆMOGLOBINURIA

Alterations in the Blood.—The diagnosis of *hæmoglobinæmia* does not give rise to great difficulties. So soon as the adherence of the hæmoglobin to the stroma of the red blood corpuscles becomes lessened, separates from the latter and circulates freely in the plasma, the appearance of the blood changes in such a manner that it becomes lacquer-coloured and transparent, because it now contains its colouring matter as a transparent colour. In clinical cases this well-developed change in colour of the blood does not occur, as this requires such an extensive destruction of the connection between hæmoglobin and stroma of the red blood corpuscles as is never observed in the living human being. However, at least a reddish tinge of the blood plasma is indicated in (microscopical) preparations, and blood that is artificially drawn by cups in cases of hæmoglobinæmia shows a ruby-red colour (Küssner). The examination of the blood under the microscope shows further changes which allow us to recognise the passage of the hæmoglobin into the fluid of the blood: The blood corpuscles appeared abnormally pale to various observers (I was not able personally to ascertain this as being the case, nor did I succeed in recognising entirely colourless blood corpuscles, "shadow corpuscles"). Alterations in shape and faulty rouleaux formation, and, what is diagnostically the most determining sign, smaller and larger yellowish-brown hæmoglobin granules between the blood corpuscles, may more frequently be demonstrated. The number of the red blood corpuscles diminishes markedly during the attacks in which the hæmoglobin becomes free (to a million and less per cmm.); but eventually it returns to its former amount in a few days.

Alterations in the Urine.—Diagnostically more important are the ***alterations in the urine*** which occur as a result of the passage of the hæmoglobin into the blood plasma. The hæmoglobin dissolves in the plasma, as we know from former experience, traverses through the epithelium of the convoluted uriniferous tubules into the urine, and a ***hæmoglobinuria*** arises.[1] The urine shows a dark reddish-brown up to a black colour, and the reactions to blood pigments are noted in the chemical examination, i. e., in combination with caustic potash and boiling, a blood-red sediment, with the Almén test (oil of turpentine and tincture of guaiac), the well-known blue colour, etc. The spectroscopic examination of the filtered and correspondingly diluted urine shows the presence of ***oxyhæmoglobin*** and especially of *methæmoglobin*; sometimes only one of these blood pigments is present, at other times both simultaneously.

The accompanying diagram shows a *spectroscopic* picture which I obtained with the Schmidt-Hänsch instrument in a case of my observation upon examination of freshly voided urine; the line towards C (227–230) corresponds to the marked methæmoglobin line, the other two (243–247; 257–263) to the oxyhæmoglobin lines.

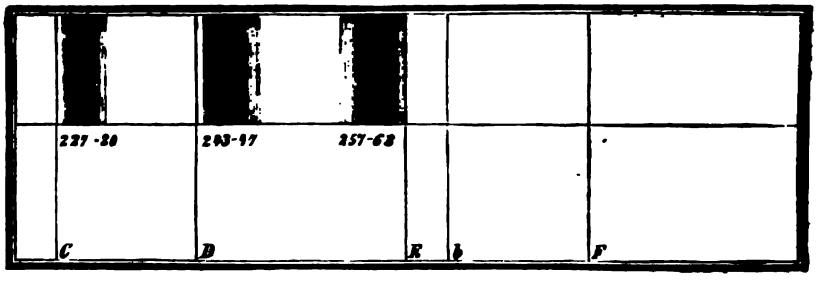


FIG. 72.—OXYHÆMOGLOBIN—METHÆMOGLOBIN LINES IN THE SPECTROSCOPIC PICTURE OF URINE IN HÆMOGLOBINURIA TAKEN WITH A SCHMIDT-HÄNSCH INSTRUMENT.

Besides hæmoglobin, the urine contains serum albumin; in other cases only hæmoglobin is met with. In the latter case, upon boiling of the urine, or upon addition of acid, a brownish coagulation of albumin, which usually floats on top, separates from the decomposing hæmoglobin. Whereas no doubt remains, according to the positive results of all these tests, that hæmoglobin is present in the urine in question, the *microscopic examination* (in contrast to the much more frequent cases of hæmaturia, i. e., the cases of excretion of the entire particles of blood in the urine) does ***not*** reveal *any red blood corpuscles*; however, yellowish-brown, irregular, granular flakes or cylindrical masses, more rarely globular, yellowish-red drops of hæmoglobin, are found in the blood preparations.

[1] This well-chosen name of the disease is due to Popper and has been in general use for twenty-five years. The occurrence of hæmoglobinuria was known for a long time previously; it was probably known to Charles Stewart one hundred years ago. But Pavy was the first who taught how to *diagnosticate* the condition, i. e., first emphasized the absence of the blood corpuscles in the urine containing hæmoglobin as being the most essential characteristic of the affection, and with this separated the latter from hæmaturia.

Ætiological Diagnosis.—The diagnosis of hæmoglobinæmia and hæmoglobinuria may be determined with ease and certainty upon the basis of the above diagnostic criteria. However, such a diagnosis is always an incomplete and a highly insufficient one so long as the origin of the hæmoglobinuria has not been simultaneously determined in the individual case. This part of the diagnosis is the more difficult one; if the diagnostician desires to meet the requirements, he must be familiar with the results of the numerous clinical and experimental investigations which have been undertaken in the last two decades regarding the pathogenesis of hæmoglobinæmia and hæmoglobinuria.

As already stated, the production of hæmoglobinæmia depends upon the separation of hæmoglobin from the stroma of the red blood corpuscles. *The normal close adhesion of the hæmoglobin to the blood cells may cease in that intensely acting agencies which destroy the blood corpuscles liberate the hæmoglobin.* We are familiar with great numbers of such causes of clinical hæmoglobinuria. The action of high temperatures upon the blood, cutaneous burns, various poisons (arsenic, pyrogallic acid, nitrobenzol, antifebrin, quinine, and, above all, *potassium chlorate*), and, furthermore, snake poison and the poison contained in fresh *morchella* [a genus of discomycetous fungi], etc. A similar influence is evidently possessed by various *infectious diseases*, such as scarlatina, diphtheria, sepsis, malaria, influenza, in the course of which hæmoglobinuria has been observed. Hæmoglobinæmia is unquestionably due to the deleterious action of the toxines upon the erythrocytes in such cases; in hæmoglobinuria which occurs in the tropical regions, blackwater fever (compare Malaria), an intoxication due to the quinine which is given for therapeutic effects, is the most important factor, besides the tropical fever itself.

In a second category of cases we should assume, according to my opinion, that *the combination of the hæmoglobin with the stroma of the blood disks has become looser than normal, and that now casual causes which for the most part are of a very harmless nature,* and which are quite without importance for the health of the normal individual, *are sufficient to produce the separation of the hæmoglobin from the blood disks,* and to give rise to hæmoglobinæmia. Such a preparatory loosening of the bound hæmoglobin occurs evidently by some severe diseases which have a far-reaching effect upon the blood life, above all, as the investigations of Murri render plausible, by long-lasting syphilis, so that a slight refrigeration, etc., is sufficient, sometimes, in individuals affected by this disease, to develop a hæmoglobinæmia.

Paroxysmal Hæmoglobinuria.—In a greater number of cases, finally, it cannot be determined how the dissolution of the hæmoglobin from the blood disks occurs. We only know that relatively frequently, apparently without any marked underlying cause, hæmoglobinuria occurs in *paroxysms* in these cases. This so-called *paroxysmal hæmoglobinuria* manifests itself in that, with the usual initial phenomena of a suddenly arising fever (drawing pains in the limbs, headache, etc.), a chill occurs, and the temperature rises to 104° F. or 105° F. In a short time, (usually) after a few hours, the attack is over and the temperature normal again. During the

attack, according to my experience, it may be noted that the *liver* is enlarged and sensitive to pressure, the spleen is palpable and pain is present in the renal region; an eruption of *urticaria* was also often observed. The urine which is voided after the chill—in one of my cases, one hour later—is brownish-red in colour and contains hæmoglobin. The hæmoglobinuria persists for a shorter or longer time, according to the intensity of the decomposition of the blood corpuscles; in the above case, four and one half hours after the rise of temperature, the urine which was voided was again free from hæmoglobin, but contained albumin for one half day longer, whereas the temperature had returned to normal five hours after the beginning of the paroxysm. *Refrigeration* may usually be proven to be the *occasional cause* which produces the attack; in other cases overexertion in *muscular movements* (marching, as was first determined by Fleischer and *myself* in one case), "*excesse in Baccho et Venere*," psychical emotions, etc. A rapid change in the innervation of the vaso-motors with contraction of the small vessels in the circulatory system probably plays the principal *rôle* in all of these ætiological factors. That the previously mentioned, occasional causes actually are the starting-point of the attack and are liable to produce hæmoglobinæmia has been proven with certainty; it is possible, in individuals who are affected with paroxysmal hæmoglobinuria, to bring on a paroxysm by an artificially produced refrigeration (cold foot-bath, etc.) or by forced marches undertaken for that purpose. Among forty-seven attacks which one of my patients had, thirty-two were acknowledgedly the result of refrigeration, six occurred in connection with overexertion in muscular movements, one immediately following a marked display of anger (once probably following fright), and in only eight attacks no marked occasional cause could be proven. Mild jaundice occurs frequently towards the end of the attack; in one attack I observed, instead of the expected appearance of hæmoglobin in the urine, albuminuria which, similar to hæmoglobinuria, could also be artificially produced, running its course with chill and fever. In others I only saw enlargement of the liver and spleen, the urine remaining free from albumin and hæmoglobin.

The paroxysms usually run their course with more or less *fever*. However, there are cases in which the fever is constantly absent, and cases, like the one just mentioned, in which the temperature remains normal during at least some of the attacks of hæmoglobinæmia. The cause of the fever cannot be satisfactorily explained as yet.

Results of Hæmoglobinæmia and their Explanation.—Regarding the explanation of the occurrence of other symptoms in the course of hæmoglobinæmia: Jaundice, albuminuria and enlargement of liver and spleen, etc., decided differences arise; still we are justified in explaining, at least with a great degree of probability, the subsequent conditions which are associated with the appearance of hæmoglobin in the blood plasma, in the following manner:

If for any cause the combination of hæmoglobin with the stroma of the blood corpuscles is destroyed, hæmoglobinæmia occurs. The hæmoglobin which has become free, does not directly, as we might assume, become excreted through the kidneys with the appearance of hæmoglobinuria, but is *eliminated by the liver*, in that it transforms the hæmoglobin into bile pigment. This assumption is proved partly by the

results of experiments (injection of pure hæmoglobin solutions into the blood of animals produced an enormous increase in the amount of bile pigment, about 25 times), partly by clinical experience. In a case of marked paroxysmal hæmoglobinuria of my observation, the colour of the stool following the attack was much darker than normal. The liver undoubtedly enlarged in this case in some of the attacks, with chill and rise of temperature, the urine, however, remained free from hæmoglobin and albumin; I was able to determine the combination: Enlargement of the liver with normal urine—with high fever (103° F. to 104° F.) in one attack after refrigeration, which up to that time regularly produced hæmoglobinuria. The *jaundice* which is combined with the attacks may be looked upon as polycholic, after it is determined nowadays that icterus may be the result of polycholia (in which case it is naturally presupposed that the liver cells are not anatomically degenerated nor functionally insufficient).

The transformation of hæmoglobin which has separated from the blood can only be managed by the liver if no very great amounts appear of the same. So soon as the latter is the case, the intensity of the hæmoglobinæmia having passed a certain limit, the *presence of hæmoglobin occurs in the urine.* The hæmoglobin may then (but by no means always)—as it appears, according to the individual irritability of the epithelia—act as an *irritant upon the epithelium of the kidneys*, but, as I must assume after my clinical experiences, only in a very transitory manner, and only in cases in which no large masses of hæmoglobin must be overcome. In keeping with this, the ribbon-like hyaline casts and the albumin disappear from the urine either simultaneously with the hæmoglobin or at least a short time after (in a few days), when the urine has become free from hæmoglobin. If it is a case of but little free, circulating hæmoglobin, this is not extracted by the renal epithelium at all; but the irritation of the hæmoglobin which passes the renal vessels is sufficient in rare cases to produce albuminuria (even without hæmoglobinuria). The attack, as the previously mentioned case proves, then takes place with precisely the same symptoms as occur in an attack of hæmoglobinuria, i. e., with chill, enlargement of the liver, etc.; only, in such cases, nothing but albuminuria is produced, or the latter may even precede the hæmoglobinuria (Rosenbach). If in the individual case we are dealing with a high grade of hæmoglobinæmia, as in destruction of the red blood corpuscles by poisons, a marked irritation of the kidneys is almost always brought about, producing nephritis. The result of this is an occlusion of the uriniferous tubules with hæmoglobin—albumin, casts; diuresis, in keeping with this, is diminished, and in the severe cases, unless an increased activity of the heart comes to the rescue, complete anuria and death due to uræmia occur. An example of hæmoglobinuria, the result of poisoning by potassium chlorate, may impress this.

Case of Hæmoglobinæmia in Consequence of Poisoning by Chlorate of Potassium.—P. B., aged eighteen, entered the hospital July 23, 1889, died July 29. On July 21 he accidentally drank a solution of 30 grammes of potassium chlorate in 250 grammes of water. Two hours later severe abdominal pains appeared; then vomiting, headache, and vertigo. Upon admission of the patient to the Julius Hospital the following condition was noted: High degree of cyanosis, slight jaundice, strong, regular pulse, spleen and liver slightly enlarged to percussion; the renal region very sensitive to pressure. The urine, excreted in small amounts (scarcely 4 to 5 cc.) was brownish, of a dark cherry-red colour after being filtered, and showed marked reactions to albumin and hæmoglobin. The urinary sediment did not contain any intact erythrocytes, but cylindrical hæmoglobin flakes; the blood also contained particles of hæmoglobin. Ordered lavage of the stomach.

July 24.—Cyanosis and jaundice increased simultaneously; respiration was accelerated; great lassitude; temperature normal. Amount of urine in twenty-four hours only 15 cc., containing epithelial casts; vomiting, severe abdominal pains, muscular twitchings; ordered caffeine, Wildungen water, strong wine. As it was impossible to produce marked diuresis by this means, subcutaneous injections of 120 cc. of a 0.6 per cent salt solution were made three times daily; the blood was free from hæmoglobin clots.

July 25.—Cyanosis less marked; spleen enlarged, reaching to the costo-clavicular line; the stools were dark-coloured. The urine (about 15 cc. during the day) was

clearer, but contained large amounts of albumin; in the sediment only hæmoglobin flakes were noted, no casts. Ordered salt solution subcutaneously and per rectum.

July 26.—Jaundice and cyanosis still less than on the previous day; urine still very sparse in amount and containing large quantities of albumin, but now very light in colour, almost transparent and free from sediment; less vomiting; the former treatment was continued.

July 27.—Continued lassitude; cyanosis and jaundice had disappeared; urine (about 20 cc. in twenty-four hours) contained large quantities of albumin, without hæmoglobin; and about 2 grammes of urea. Traces of urea were noted in the vomited material.

July 28.—The pulse was quite strong, the temperature normal; palpation of the enlarged liver painful. The patient was restless; respiration somewhat difficult; urine very sparse. The pulse shortly before death became small and irregular. *Exitus letalis* without convulsions or without other well-developed uræmic symptoms, at one A. M., July 29.

The *autopsy* on July 30 (v. Rindfleisch) showed: *Acute nephritis* with gray discoloration of the parenchyma; the colour of the cortex more yellowish, with disseminated, brownish, punctate areas, the papilla contained radiating, chocolate-brown striæ. The *microscopic examination* of the kidneys showed: In the collective canaliculi and in Henle's loops were dark-brown, granular masses which were partly hyaline casts, partly massive deposits of desquamated epithelium. The convoluted tubules were markedly dilated, with opaque epithelia the nuclei of which could no longer be completely stained; the lumen of the canaliculi was filled with net-like fibrin masses. *Spleen lightly enlarged*, of firm consistence; the Malpighian bodies showed an increased amount of leucocytes, whereas the pulp contained an extraordinary number of red blood cells that appeared unchanged and between which, often also combined with the white blood cells, were deposits of finer or coarser pigment flakes. The *liver* was also slightly enlarged, and showed, upon microscopic examination, here and there a deposit of fine brown pigment masses in the capillary vessels, rarely in the liver cells. *Stomach and intestine* presented the signs of inflammation and several erosions.

As in the above case, I was also able, in the case of paroxysmal hæmoglobinuria which has been frequently cited, to determine an *enlargement of the spleen*, besides the enlargement of the liver. The cause of this splenic enlargement is partly due, as Ponfick believes, to an intumescence of the organ by the remains of the red blood cells which have floated in ("spodogenous" splenic tumour), and partly, to conclude from the result of the above-described autopsy, to the immigration of hæmoglobin and a reactive new formation of white blood corpuscles.

Differential Diagnosis.—Paroxysmal hæmoglobinuria cannot be confused with any other affection if proper attention is paid to the symptom-complex. The statements of the patients that they periodically void dark urine, and that this occurs regularly after active body movements, refrigerations, etc., make the diagnosis of hæmoglobinuria probable from the onset. A single examination of such dark urine—the proof of the presence of hæmoglobin or the simultaneous presence of red blood cells in the urine—is sufficient to determine the diagnosis with certainty and at once to differentiate the affection from *periodically arising hæmaturia* (the result of renal calculi, hæmorrhagic diathesis, etc.). If hæmoglobinuria has thus been determined, the finer details of the diagnosis of hæmoglobinæmia should now be demonstrated with certainty: The presence of enlargement of liver and spleen, the onset and course of the fever, the excretion of albumin in the urine, and, finally, the last cause of hæmoglobinuria.

DISEASES OF METABOLISM

PHYSIOLOGICAL INTRODUCTION

By *metabolism* we understand the totality of the changes taking place in the living organism, by which it is enabled to assimilate the substances which have been introduced as food and have become resorbed by digestion, to translate the energy acquired in this manner into living force, and to rid itself of the end-products of the metabolic changes, which are no longer of use in the economy, by the various organs of excretion. The preservation of life and of the functions of the living organism unquestionably demands the conveyance of chemical energy, the transformation of which into heat and force makes the manifestations of life possible. The material containing this power is conveyed from without by the food, and the chemical change of the nourishment occurs by means of *oxygen* which is taken up from the inspired air, so that the substances are oxidized into carbonic acid and water. The immortal credit of having established this fundamental fact belongs to Lavoisier, who was the first correctly to discern the process of oxidation and combustion. Following in his footsteps, J. Liebig, about the middle of the nineteenth century, developed the then celebrated theory of the more minute use of oxygen in metabolism. Accordingly, the two main constituents of animal nourishment: The nitrogenous (albumin) and the non-nitrogenous (fats and carbohydrates) were to be utilized in the economy in totally different ways, the former as blood and oxygen producers, "plastic nourishment," the latter, the non-nitrogenous, for the production of animal heat, and, especially, of the products of respiration ("respiratory substance") and, with this, indirectly for protection of the organs from the effects of oxygen. According to Liebig, the oxygen of the atmosphere constituted the active external cause of the consumption of matter in the animal organism, and, through the mechanical effects produced by the body, a portion of the muscle substance was to lose its "vital properties" and perish. The innumerable investigations of metabolism, undertaken upon the basis of the proclamation of this axiom, showed, however, that, although during muscular activity a plentiful supply of oxygen was taken up, the excretion of nitrogen compared to the state of inactivity (rest) was not materially changed, and that the food material was by no means *directly* affected by the inspired oxygen. More and more the law was developed that nourishment, primarily independently of the supply of oxygen, separates into chemically simpler combinations, and only then, in accordance with the magnitude and direction of these chemical processes of division, sufficient oxygen is secondarily conveyed to the blood further to decompose the substances so formed. The continuous decomposition of matter, which varies greatly in its extent, is a necessary supposition for the life of the organism, as by this means it obtains its energy, and is able again to transform it partly into heat and partly into force.

The latter, the transference of energy into living force, is inseparably combined with the conservation of life. Because certain occurrences, such as the action of the heart, the activity of the glands, the maintenance of the body temperature at a certain height, are necessary processes even in complete rest for the continuance of life, which presuppose a consumption of energy and which require a substitute in the form of *food*, provided the organism shall not be compelled to live upon its own resources and, after the loss in weight has amounted to more than one half of the body weight, to succumb to death by starvation.

The foods are the same chemical materials from which the tissue of the organism is constructed: *Albumin bodies, fats, carbohydrates, water* and *salts*. As constantly living substances succumb during life of man, i. e., nitrogenous substances are given off in the desquamating epidermis, in perspiration, in saliva, in semen, and with the disintegrating cellular material, and as for these losses, for the "diminishing organic albumin," a substitute must be obtained, the nitrogenous albuminous substances (and salts) can never be dispensed with entirely in the food. From this follows that the albuminous bodies take the front rank as necessities for the life

of the organism. They consist of C, H, O, and N, (as a rule also of small quantities of sulphur, phosphorus and iron), whereas fats and carbohydrates only contain C, H and O.

After the food has reached the intestinal tract, it is, partially at least, not absorbed at once. It is first, after water has been taken up, split, "hydrated," by the enzymes which are specially supplied by the digestive organs and made more resorbable, then to be conveyed to the tissues of the body by the blood or by the lymph channels and to be further acted upon by the cells. The process of *fat* and *carbohydrate* absorption is readily understood. They are rendered suitable for absorption in that the fats are first emulsified into the finest drops, and the carbohydrates are changed into sugar. On the other hand, the processes of digestion and absorption of *albumin bodies* in the digestive tract are more complicated and partly difficult of explanation. Similar to the carbohydrates, the albumin bodies are also hydrated in the stomach and intestines by enzymes, viz., by the pepsin of the gastric juice and by the trypsin of the pancreatic secretion, i. e., they are separated hydrolytically by taking up water, and are changed gradually into albumoses and, finally, into peptones, which, in contrast to the native albumin bodies, present more readily soluble albumin modifications. The absorption of these albumoses and peptones is quicker than that of the simply dissolved albumin bodies, and herein lies the key of the explanation why peptonizing is necessary at all in the organism, because the incorporation of peptones in the economy in itself would be unnecessary, even harmful. Not alone is a lesser amount of energy introduced with the latter than with unchanged albumin bodies, but albumoses and peptones, which, without the intercession of the wall of the intestine, are directly injected into the blood, actually behave as foreign bodies, i. e., are promptly excreted as such by the kidneys, unaltered, and even develop a toxic action, provided large amounts circulate in the blood current. The peptones, however, lose this toxic property in their passage through the intestinal wall, in that they here undergo a metamorphosis into albuminoid bodies, coagulable by the action of heat at the temperature of boiling water. This process, which at first sight appears to be a luxury, becomes comprehensible if we reflect that under all circumstances it is of advantage for the organism if albuminous substances can be rapidly absorbed in large quantities, which is only possible in the shape of peptones; otherwise it might be expected that a large portion of the native albumin substances, which are surely very slowly and incompletely resorbable, would pass into the small intestine and, in its further course, putrefy in the large intestine. Furthermore, the reconversion of the peptones into albuminoid substances, taking place in the wall of the intestine, may prevent that the quantity of the juices is temporarily overwhelmed in rapid changes with albumin substances, which it is impossible for the body to utilize rapidly enough. It may be assumed, therefore, that the assimilable metamorphosed products of the peptones, which originate in the intestinal mucous membrane, are retained and only decomposed according to necessity, to serve partly as sources of energy, partly as substitute material for the formation of new cells in place of those which have been consumed in the living organism. The process of the absorption and utilization of *sugar* in metabolism must be regarded in a similar manner. It is also true of sugar that, if injected directly into the blood, it acts in the organism as a non-assimilable foreign body. Under such conditions it is excreted by the kidneys, the same as the peptones, in an unaltered condition, but only when the quantity of grape sugar exceeds a certain limit, 0.2 per cent. As normally there is no sugar excreted by the kidneys, we must assume that an arrangement exists in the body which regulates the amount of sugar in the blood. This actually takes place, as innumerable experiments have shown, in the liver. The sugar which in the digestive tract is formed from the carbohydrates, is carried by the portal vein directly to the liver and here, by the union of a greater number of sugar molecules and with the loss of water, is changed into animal amylum, glycogen, and deposited. The muscles and glands, the same as the liver, are also capable of transforming nutrient sugar into glycogen and of storing it. Glycogen, therefore, in metabolism plays the part of a transitory reserve product, from which there is taken as much as is necessary for use. And this occurs, as may be assumed with certainty to-day, in spite of contradictions, in that the glycogen in

the liver is changed back into sugar by a diastasic ferment (contained in the blood) and passed into the circulation as soon as the amount of sugar in the blood diminishes as a result of use; the hepatic cells may therefore be looked upon as finely adjusted regulators of the normal amount of sugar contained in the blood current. The sources of glycogen are certainly, to the greatest extent, the carbohydrates of the food, but partly also the albumin bodies, as these in their decomposition are changed not only into nitrogenous, but also into non-nitrogenous, atom-complexes, from which, then, sugar, respectively glycogen, originates synthetically. It is most probable that grape sugar, respectively glycogen, may be also produced from fat, but it is quite certain that, inversely, fat may be formed from sugar, respectively carbohydrates.

The very great albumin molecule splits into various atom-complexes in its use in the body, which partly belong to the fat bodies, like leucin and asparaginic acid, partly to the "aromatic" substances, like tyrosin and various products which form from the albumin decomposition in the bowel. Upon the basis of examinations in metabolism it may be further assumed that the N-free atom-complexes which split off from the albumin molecule during decomposition of the food albumin (as has already been mentioned) may change into glycogen and, at least indirectly, into fat. From all this it appears that the albumin products of the food play an omnipotent part in the animal economy, in that they do not only serve as sources of energy, but that they can also be utilized universally in the formation of the constituent parts of cells; but, besides, the carbohydrates and the fats are employed for the production of living energy in the human organism. The fat, emulsified into the minutest particles, is taken from the bowel into the chyle, respectively into the lymph channels, passes into the blood, is dissolved in the plasma (in a manner not quite clear) and then brought to the cells, to be used partly as material for combustion, partly to be deposited in the tissues as fat (adipose tissue). From this point fat is used as required for the development of energy and heat.

The transformation of foods in the economy is accomplished in the main by oxidation, i. e., the excreted products contain more oxygen than the materials taken up in the food; however, the chemical process until the end-product is reached is by no means entirely known as yet in detail. We know, however, that the substances taken into the body (albumin, carbohydrates and fat) undergo changes, such as splitting up, oxidation, reduction and syntheses, and that combustion occurs at a temperature which is decidedly lower than that required for the combustion of the same products outside of the animal organism. The end-products of metabolism which can no longer be utilized leave the body in the form of carbonic acid, water and urea. In these chemical processes, both in oxidation and also, up to a certain extent, in the splitting of the molecule of food stuffs, energy is consumed and transformed into living force, which may become manifest as heat or as demonstrable activity (work). We are accustomed to express the living force which becomes available by the decomposition and oxidation of the materials in the body (therefore the value of the food products as the sources of heat and force), in "*calories.*" By (great) calorie [the heat unit] we understand the amount of heat necessary to heat one kilo of water 1° C. Fats and carbohydrates, combusting into carbonic acid and water, give different caloric figures: Namely 1 g. fat about 9.3 cal., 1 g. carbohydrate, 4.1 cal. The combustion of albumin in the body results in less simple end-products (urea, etc.) which, in leaving the body, represent a certain calorimetric combustion value; we must, therefore, deduct from the combustion value of albumin that of the end-products; the combustion value then of 1 g. albumin is found to be 4.1 cal. If, now, the values of the various food products are compared with each other, in regard to their physiological combustion value, we find that 100 g. albumin = 100 g. carbohydrates = 44.1 g. fat, in that in their combustion in the organism they furnish the same sum, viz., 410 calories. In this proportion, then, the various substances are able to substitute for one another—they are, therefore, used as material for the development of living (active) force, in definite proportional amounts "isodynamic."

The intake of the assimilated food material which is to sustain the body, balances the outlay under normal physiological conditions—there is an equilibrium of metabo-

lism. This may be concluded from a comparison of the total amounts of the material taken in and given out. If just as much *carbon* is taken in as excreted, this denotes that the amount of organic substances taken up by the system has undergone combustion; if the amount of carbon introduced is greater than the amount excreted, an accumulation, and, in the opposite instance, a loss of organic substances, must be assumed. To estimate the *exchange of albumin* in metabolism, the determination of nitrogen both in absorption and in excretion is used, in that the nitrogen used in the system is almost entirely contained in the albumin. If more N is absorbed than excreted, this indicates an accumulation of albumin, the opposite means a loss in the organic albumin. If the quantity of nitrogen contained in the albumin of the food absorbed corresponds to the excreted N, *equilibrium of nitrogen* exists in the body.

The amount of nitrogen exchange under similar conditions is not the same in all persons. In general, the matter utilized by small individuals is greater than that in large ones, as they possess a relatively larger body surface, and for this reason must develop and give off more heat. The material utilized in child and woman is comparatively greater, therefore, than in man, but not absolutely greater, as the body mass, i. e., the number of cells that are working and which must be nourished, is naturally smaller in the child than in a full-grown man.

Metabolism is to a marked extent influenced by various conditions in the *same* individual, which greatly alter the amount of the exchange of substance. This is primarily dependent upon size and work which the body must perform. Whereas in a condition of rest the adult human being of medium weight uses about 2,000 calories during twenty-four hours, and with light work 2,500 calories, i. e., for the kilo body weight about 30 to 35 calories are transformed, the calorie consumption, and, with it, the requirement, is much greater (about 40 per cent and more) as soon as more marked muscular labour is performed, so that the working human being converts 3,200 to 3,500 calories per day (40 to 50 cal. per kilo. and over). The organism, therefore, in keeping with the greater decomposition, consumes more O_2, and excretes more CO_2, by the lungs, whereas the change of albumin is not increased, as a rule. *The muscle, therefore, in spite of its formation from albuminoid substances, supplies its force-producing combustion material from its non-nitrogenous combinations.* If it has become probable, according to new investigations, that also the nitrogenous constituents of the muscle undergo transformation during muscular activity, this changes nothing in the above postulate, in that the products which are here formed from the nitrogenous material unquestionably are reconverted into their original condition. What has been observed in investigations in metabolism as the consumption by muscular labour, has shown itself to be a consumption of glycogen, respectively of fat and carbohydrates, and only when these are not sufficiently present is albumin used to cover the required amount of calories. In an exclusive, or at least predominant, ingestion of albuminous food, we note exceptionally also an increase of N-excretion, as a result of muscular activity. If the working organism is to remain at its normal condition, a larger quantity of food than is required during rest becomes necessary; larger amounts of carbohydrates and fats, and also more albumin, should especially be added to the food when it is intended to produce in the working organism an accumulation of flesh, i. e., an increase of the muscular mass, the substratum of the power of action. In contrast to the working periods, the change of substance during *sleep* is decidedly less, which shows itself by the smaller quantity of O_2 taken up and the smaller excretion of CO_2 during the period of rest.

As the muscular work, so also is the *digestive process* (the necessary activity of the glands, peristalsis and absorption) combined with an increase in metabolism; and also the steady *giving off of heat* of the human body acts in a similar sense, which is founded upon the greater internal heat as compared with the temperature of the external world. To keep the body temperature at a constant height, the organism must produce combustion of a greater quantity of material (non-nitrogenous substances, whereas the transformation of albumin is not influenced by heat or cold), all the more, the lower the external temperature, and *vice versa*. The regulation of heat, however, is by no means dependent alone upon a change of the condition of

metabolism, i. e., of the production of heat; but, to accomplish this, the organism has at its disposal an entire series of mechanisms regulating this condition (dilatation or narrowing of the cutaneous vessels, increase or diminution of respiration, secretion of sweat, etc.) which, by aid of the nervous system, become reflexly active and markedly influence the amount of heat given off.

Besides the conditions which have been mentioned—the giving off of heat, the quantity of work, the size of the body and the age—metabolism in man is markedly dependent upon the *quantity and variety of the nourishment partaken of.* A great number of experiments, undertaken according to a certain plan with hungry individuals or with persons and animals nourished according to a certain method, have been carried out in the last forty years to determine the changes of metabolism by means of the food supplied. The exact calculations of the C and N contents of intake and output, of the dependence of the decomposition of albumin upon the quantity of albumin which is ingested, of the determination of the relations of carbon and nitrogen equilibrium, etc., made it possible to establish the laws according to which metabolism is influenced by nourishment. The main results shall be briefly mentioned in the following:

1. It is best to start from metabolism during *hunger.* If all supply of food ceases, the body must live on its stock of substances on hand, as the processes of combustion, especially the production of heat, occur also during hunger, even though to a slighter degree. The body, accordingly, during this condition of hunger, at the onset uniformly loses in weight; later, often suddenly a relatively larger loss of weight occurs. The fat is consumed to the greatest extent, the albumin to a less degree; the latter is destroyed in a uniform manner during hunger, which has been determined with exactness by experiments in metabolism. The decomposition of albumin is relatively great during the first period, dependent upon the amount of the former nourishment and upon the reserve albumin which has been accumulated in the body (the "circulating" albumin). In the second period of famine, which begins after a few days, the decomposition of albumin is slight; an almost uniform quantity of organic albumin is destroyed daily. This period continues as long as fat is contained in the body; as soon as the fat on hand in the body is exhausted, a rapid increase of the albumin decomposition and N-excretion occurs—therefore fat, in conditions of famine, plays the most important part of limiting the consumption of albumin and allowing the endurance of hunger for a longer period. At least five sixths of the production of heat in the starving individual are defrayed by fat, and only one sixth by albumin. The excretion of water and of salts decreases steadily during hunger, and, as far as sodium chloride is concerned, finally ceases entirely; before the appearance of death by inanition, with an increase of albumin decomposition, a marked increase of excretion of water (combined with albumin) occurs by means of the urine.

2. If only *individual* substances, or *all* necessary constituents of food but *in too small an amount,* are taken, the body is said to be in a condition of *partial hunger.*

In *salt hunger,* i. e., with the absolute absence of salts in the nourishment, the excretion of salts gradually diminishes, and that of sodium chloride eventually ceases entirely. Finally, with nourishment which is free from ash, death occurs obviously as the system does require a certain quantity of salt in proportion to the organic constituents, not only for the growth and development of the body, but also for the maintenance of life of the adult organism. Besides, the salts play an important *rôle* in the absorption of food. The partaking of calcium salts is absolutely necessary for the development and maintenance of the bones, ferruginous food is most essential for the normal composition of the blood (which contains over 80 per cent of the iron of the body).

The *withdrawal of water* leads to death more rapidly than the withdrawal of nourishment. Even after only 11 per cent of water contained in the organs under normal circumstances are given off without being replaced, discomfort becomes manifest, and if 22 per cent are lost, death occurs—obviously due to the fact that the protoplasm is seriously damaged by the withdrawal of water.

It is self-evident that, if water and salts but no organic food constituents are

taken in, the body lives from its substance, and death must occur the same as in absolute hunger, although somewhat later.

But even a one-sided absence of albumin, fat, and carbohydrates in the nourishment is not at all or badly borne by the organism, if long continued, as will be seen from the explanation of the conditions of metabolism in exclusive nourishment of the body with individual food substances.

3. Nourishment consisting exclusively of *fat* and *carbohydrates* cannot prevent loss of albumin, although it is slighter than in the case of absolute hunger, and in keeping with this death by albumin hunger occurs later and without the above-mentioned preagonal increase of N-excretion. By an overabundant administration an accumulation of fat may even be attained in the famishing individual; this is also true in exclusive nourishment by carbohydrates. But the decomposition of albumin is not retarded either with an exclusive carbohydrate food or with an exclusive fat nourishment; in both cases the trial animal finally dies of albumin hunger. Only the resistance of an animal fed on carbohydrates is greater than one fed on fat, as the carbohydrates diminish the albumin loss by about 15 per cent and more, i. e., they have a stronger action in saving albumin than fats.

Exclusive nourishment of *albumin without simultaneous administration of carbohydrates and fat* may, in the *carnivora*, entirely prevent the loss of substance in the body and may finally produce an equilibrium of metabolism in the organism. However, it has not been possible as yet to nourish a *human being* with albumin alone, as meat, if this constitutes the only food administered, owing to the excessive quantities necessary to substitute fat in the nourishment, cannot be sufficiently assimilated, producing disturbances of digestion. Only the albumoses can completely substitute albumin as food, whereas peptones, lime and lime-giving tissues cannot entirely do so. The last-named substances, therefore, cause only a saving in albumin, which, however, may be quite considerable; it is twice as marked, for instance, in the case of lime-feeding as in feeding with carbohydrates.

The human body, being that of an omnivorous one, must have *mixed nourishment*, as the preceding has shown, to be properly nourished. As sufficient medium nourishment for an adult man with light work the following quantities have been determined: 100 gm. albumin, 60 gm. fat, and 400 gm. carbohydrates per day. The calorie requirement of woman is in general smaller, only four fifths of the quantities mentioned above being necessary for her full nutrition. With this the equilibrium of metabolism can be maintained; if lesser quantities of food substances are taken than are necessary for the equilibrium, the body yields of its substance until equilibrium is re-established, i. e., so that the body may sustain itself from the nourishment taken in.

The composition of nourishment in regard to individual food substances is of distinct importance in certain directions in regard to metabolism.

The increase of *albumin* alone in the food produces a more marked consumption of albumin and an accumulation, but only to a slight degree, of the albumins taken in. This causes the body to become somewhat richer in flesh, and it now requires a greater amount of food albumin to attain a new equilibrium of nitrogen. Why the body does not accumulate all the superfluous albumin, but for a greater part soon combusts it again, why, therefore, an apparently superfluous consumption occurs in regard to it, is unknown. For, although albumin represents of the three principal nutritious substances the one easiest to transform, i. e., easier of combustion than carbohydrates and much more so than fat, it remains an enigma for the present why the superfluous amount is used almost exclusively for the production of heat. Because the albumin, which is so extremely valuable as a food product, is so indispensable, on the other hand, that if less than 70 to 80 grammes of albumin per day are partaken of in food, an albumin decomposition occurs in the body. An exception to the above-named, apparently superfluous consumption of albumin is only constituted by those cases in which the cells or tissue elements have become poor in albumin by deficient nourishment, severe diseases, etc., or in which the body of the child is growing. Here a complete utilization of albumin and a marked accumulation actually occurs to replace the loss of albumin or to form new cells.

Clearer and more simple are the conditions in regard to the influence of *fat* and *carbohydrates* upon metabolism. If with mixed nourishment the administration of fats and carbohydrates is increased, the transformation of the N-free substances is increased, on the other hand that of the albumin products is diminished, therefore *albumin is saved.* As the carbohydrates are easier of combustion than the fats, they save albumin to a greater extent. Therefore, by favouring fats and carbohydrates in the composition of the nourishment, the quantity of nutritive albumin may be markedly reduced without endangering the albumin of the body. The minimum of albumin intake necessary in this respect has been put at from 70 to 50 grammes and less, which is of great practical importance. *Vice versa*, if in sufficiently mixed nourishment the amount of albumin is markedly increased, there will be, as may be easily understood, an increase in albumin metabolism; but flesh may be accumulated at the same time and, in so far as fat and carbohydrates are used to a less extent owing to the increased combustion of albumin, fat may also be deposited.

When the physician has the duty to bring the body which has been undernourished by disease and malnutrition into a better condition, he must confine himself to these briefly sketched fundamental principles; and just so must the diagnostician recur to them when he is to decide by what method a one-sided decrease or a greater development of flesh or fat has been produced. To sum up, I will once more emphasize that an *increase in flesh* can only be accomplished by an intake of albumin; but in a diet consisting largely of albumin an increase of flesh will only occur to a *slight* degree, relatively most markedly if plentiful quantities of fat and carbohydrates are taken in besides the albumin, and if the muscles are forced to a more marked development by systematic practice (especially, as we know, to an increase in thickness of individual fibres), whereby albumin will be forced to accumulate. But fattening, on the other hand, which, when the average of fat accumulation has been exceeded materially, represents a disease (see Obesity), is always brought about in that the amount of nourishment is too great in comparison to the decomposition of material. Larger fat accumulations especially are caused in the least part, as we have seen, by an excessive increase of the albumin intake, but to the greatest degree by an intake of overabundant quantities of carbohydrates and fats (besides medium quantities of albumin). Besides carbohydrates and fat, especially the use of alcohol is capable of increasing fat. Of alcohol, C_2H_6O, at least 90 per cent are consumed in the body, as we know for certain, whereby under all circumstances fat is preserved from decomposition. As alcohol furnishes on combustion more calories than do carbohydrates (one gramme = 7.2 cal.), its importance is seen without more ado in regard to the substitution of the latter, more so, as the so common alcoholic drink, beer, contains almost twice as much sugar and dextrin, besides alcohol. Most important, above all, in producing fat is the limitation of muscular activity, which, as a method for the increase of fat after what we have explained, is self-evident; and just as clear for the increase of fat is the favourable action of high temperature of the air surrounding the body, of warm clothing and the thick, fat layers of the surface of the body that has become fat, as all these factors diminish the withdrawal of heat and decrease proportionately the amount of fuel necessary to produce the individual body warmth. There are some other conditions which are of importance in obesity which, however, cannot be entered upon except in the diagnosis of this disease, as well as disturbances of metabolism in certain directions, which will be enlarged upon in greater detail in the discussion of the individual diseases of metabolism in the following chapters.

DIABETES MELLITUS

The diagnosis of diabetes mellitus depends primarily upon *changes of the urine,* and only secondarily are to be regarded the *alterations of metabolism,* the expression of which are, besides the abnormal condition of the urine, the various disturbances in the general organism. We have pri-

marily, therefore, to study the condition of the urine in diabetes mellitus and accurately to describe the constituents of the same.

The change of the urine, which gives the characteristic stamp to the affection, is the presence of *sugar*. The presence of this abnormal constituent may be determined without special preparation of the urine. However, in testing urine for sugar certain precautions must be observed if we mean to be certain that sugar is actually present.

This subject shall here be entered into at length only in so far as is required by practico-diagnostic consistency. As a first reaction in all cases *Trommer's test* (with caustic potash and copper sulphate) may be used to determine, at least rapidly, whether the urine in question contains a *reducing substance* in large amounts. Reducing substances (uric acid, pyrocatechin, glycuronic acid and creatinin) are found to a greater or lesser extent in every normal and pathological urine. However, according to my experience, the result of Trommer's test allows us at once to draw at least the probable conclusion that sugar forms the principal part among the reducing substances present in urine. If, in applying the test, much copper sulphate of a lazulite blue dissolves and if, after the fluid has first been heated to boiling, the copper suboxide appears as a *yellowish, powder-like* precipitate, it is at once probable that the urine which has been examined contains sugar. In the presence of the other reducing substances previously named, copper suboxide, as a rule, remains in solution and only a yellow or yellowish-green discoloration of the fluid occurs. The same is also true, moreover, in a *concentrated* urine which contains but small amounts of sugar; here also the copper sulphate does not form like a powder if the urine contains, besides sugar, relatively many substances which precipitate copper suboxide (uric acid, creatinin, ammonium). It must be remembered, on the other hand, that the various therapeutic agents, such as chloral hydrate, turpentine, salicylic acid, etc., give rise to a precipitation of copper suboxide powder in the urine, simulating a sugar reaction. Lately Trommer's test has been replaced in practice by Nylander's test (potassium and sodium tartrate, caustic soda and bismuth subnitrate), in which, upon the addition of about one tenth of the reagent to the urine and prolonged boiling, the precipitate which is at first white becomes black by the reduced bismuth, this colour becoming more and more prominent. It is quite proper to use this test, as the positive result of the test (provided that the reducing action of certain medicaments: Rhubarb, salicylic acid, tannin, turpentine, antipyrine, etc., can be excluded) can only be referred to the reduction of the bismuth solution by grape sugar.

If the result of one of the two sugar tests is positive, it is advisable still further to ascertain the presence of sugar in the urine by the reaction with *phenylhydrazine* (Emil Fischer), by the *fermentation test* and by the *polariscope*. If the physician has such an instrument, the qualitative and quantitative estimation of sugar can be obtained most rapidly and most certainly by the demonstration of a deviation to the right of the plane of the polarized light. The two other methods require a longer time, that is, the phenylhydrazine sugar reaction and the fermentation test. For the purpose of demonstrating sugar according to the first, into a reagent glass half filled with water phenylhydrazine chlorate (about two tips full of a knife) and sodium acetate (three tips full of a knife) are placed, to which is added the same volume of the urine which is to be examined; then the reagent glass is placed in boiling water, or upon a water-bath, for one half hour. After cooling in cold water, a yellow, crystalline precipitate (phenylglucosazone) appears in the presence of sugar, which, under the microscope, is found to consist of fine yellow crystalline needles. A still longer time is required by the fermentation test, i. e., the demonstration of the property of grape sugar to develop with yeast into carbonic acid and alcohol. For this test from three to twenty-four hours are required, according to the greater or lesser quantity of sugar present in the urine. The carbonic acid which develops in fermentation is collected by a suitable arrangement (most easily in a so-called fermentation tube) and, by the addition of caustic soda, is caused to disappear. It is

necessary, however, to have two control tests; viz., one with yeast and water, to prove that the amount of carbonic acid which has collected is not due to the fermentation of the yeast itself; the other control test, with yeast and sugar solution, to determine the property of fermentation of the yeast itself.

Physiological, Alimentary, Pathological Glycosuria.—If in this manner it has been demonstrated with certainty that the urine contained sugar, a further question must be next decided whether the presence of sugar in the individual case allows of the conclusion that it is due to diabetes or not. It has been demonstrated without question that the urine of a normal person may contain grape sugar and probably also in most cases it does contain it, a fact which is easily understood if we consider that under all circumstances sugar circulates in the blood of a healthy individual, and as soon as it increases to beyond 0.1 to 0.2 per cent, it readily filters through the kidneys. But these amounts of sugar which are excreted normally under the usual conditions of nutrition in healthy individuals are in such minute traces that they can neither be detected by Nylander's reagent nor by the fermentation test, the last resort to decide the question (in which amounts of sugar of 0.1 per cent still give a positive result), as to the presence of sugar. The amount of sugar present in the normally nourished person *cannot* practically be regarded in diagnosis.

It is diagnostically more important that in the normal human being also, under certain conditions, especially after partaking of large amounts of sugar, *transitorily* (*alimentary glycosuria*) *large amounts of sugar may appear in the urine*, which, upon a single examination of the urine or with casual observation of the patient, may resemble the condition of diabetes mellitus (a *permanent* pathological excretion of sugar).

Regarding *physiological alimentary glycosuria*, its occurrence cannot be doubted. However, the accidental taking of even large amounts of sugar in the nutriment does not produce a marked excretion of sugar in the urine in the majority of persons; in some healthy persons, it is true, it may amount to several tenths of 1 per cent (up to 0.3 per cent). Under all circumstances, however, in those cases there is a *rapidly passing* deviation from the normal conditions of the urine, in that the urine contains large amounts of sugar a few hours after a meal rich in sugar, but this sugar regularly disappears after two hours or at most after one half day. It is always justifiable, therefore, in determining the presence of sugar in the urine for the first time, to regard the composition of the meal last partaken, or only to utilize the result of the examination of the first urine passed in the day as determining in the diagnosis.

Lately the property of the normal individual completely to assimilate sugar in the nourishment up to a certain amount (150 to 200 grammes) has been utilized to determine deviations from this normal condition (*pathological alimentary glycosuria*), partly in the diagnosis of certain diseases, especially of the nervous system and of the liver, partly for the recognition of diabetes mellitus in the primary stages of the affection. The numerous investigations which have been undertaken in this connection have shown that in fatty degeneration of the liver due to poisoning by phosphorus, in cirrhosis of the liver, in progressive paralysis, in functional neuroses (hysteria, traumatic neuroses, etc.) and, finally, in some fatty subjects, alcoholic and gouty patients, a tendency exists to excrete sugar. Therefore, the proof of the presence of a pathologic alimentary glycosuria (i. e., even with the administration of about 100 grammes of sugar) may be utilized in the diagnosis of these affections, respectively for the assumption of a diabetic predisposition. But it is not allowable to draw far-reaching diagnostic conclusions from either the positive or the negative

results of this test for the disturbances in the assimilation of sugar, as in the above-mentioned affections alimentary glycosuria is by no means a constant phenomenon and individual fluctuations in regard to the property of assimilation of sugar are noted also in the healthy.

Lactosuria.—From the ordinary form of diabetes mellitus there is to be separated, further, the *lactosuria occurring during pregnancy and lactation.* According to recent investigations this form is in relation to the resorption of sugar of milk in a faulty outflow of the milk which collects; the sugar excreted in the urine is not grape sugar but ***milk sugar.***

Milk sugar reduces alkaline copper solutions in boiling and turns the polarization plane to the right, but, in contrast to grape sugar, it does not ferment with pure yeast nor does it show the Rubner grape-sugar reaction (precipitate with lead acetate, decomposition of the filtrate with ammonium until a precipitate occurs which, upon warming, shows a rose-red colour).

Why the thus absorbed milk sugar is excreted in the urine in an unchanged state has been determined lately, F. Voit having shown that milk sugar, if it reaches the blood as such, is not combusted at all but only subject to destruction if, partaken of with the food, it is separated into galactose and glucose after it has reached the intestine.

Modifications of the Excretion of Sugar.—The variety of sugar excreted in the urine is almost exclusively *grape sugar.* Apart from those cases already mentioned, which are at once sufficiently characterized by the history, in which milk sugar is excreted in the urine, levulose and pentose have also been demonstrated in the urine in some few instances.

Pentosuria.—The presence of 5 C-atomic sugar, of pentose ($C_5H_{10}O_5 = COH - (CHOH)_3 - CH_2OH$) in the urine may be easily demonstrated. They reduce powerfully and also give the phenylhydrazine reaction like the hexoses, but, in contrast to these, they do not ferment with pure yeast and do not turn, or but very little, upon polarization. If phloroglucin and fuming hydrochloric acid are added to the urine, there develops, if the urine contains pentose, an intense red colour (Tollen's reaction) upon warming. Pentose is occasionally found also in the urine of healthy individuals, provided pentose or its preliminary stage, pentosan (contained in fruits, various wines, etc.) was partaken of; but rare cases occur in which pentose is ***permanently*** excreted—we are then dealing with an anomaly of metabolism (*"chronic pentosuria"*). The pentose excretion is not then influenced by the nourishment, i. e., pentosuria persists uniformly, immaterial whether albumin products or carbohydrates are partaken of, or whether the latter are entirely omitted from the food. As the nucleins in their carbohydrate group contain pentose, it may be possible that under normal conditions, in the decomposition and in the construction of cells in the body, pentoses are formed which usually undergo combustion, but in patients affected with pentosuria they are not utilized and are excreted. Why the latter should occur would be difficult to explain, for the most obvious assumption that the oxidizing power of the patient affected with pentosuria is superficially insufficient regarding the pentoses (similar to that of the diabetic regarding grape sugar) has been proved to be incorrect in the investigations directed to this point, and we must admit, whether we like to or not, that the cause of pentosuria has not been cleared up as yet.

The *amount* of grape sugar excreted in the urine varies very markedly in diabetes mellitus: from a few tenths of 1 per cent to 10 per cent and over, and from a few grammes up to several pounds as a daily quantity. ***The amount of sugar depends primarily upon the nourishment,*** in that, as

is well known, starchy and saccharine foods (even a few hours after their reaching the stomach) markedly and rapidly increase the excretion of sugar. With a strict observation of animal diet the sugar, in certain cases, disappears entirely from the urine (see below), this is also true during intercurrent diseases. Above all, *muscular movements* usually diminish the glycosuria, as in contraction of the muscles sugar is utilized which is drawn from the stock of sugar on hand in the blood. On the other hand, marked *irritations of the nervous system* may increase the amount of sugar in the urine. The conditions mostly influencing the amount of sugar excreted should be taken into consideration in deciding as to the degree of diabetes in the individual case. Besides grape sugar, *glycogen* has been found by *me* in the urine of diabetics, a theoretically interesting fact, but irrelevant in a practical respect.

Other Alterations of the Urine.—Besides the principal chemical changes mentioned so far, the urine of diabetics shows further deviations from normal conditions, the knowledge of which, in the estimation of the form and intensity of the individual case, is of importance.

The specific gravity of the urine of diabetics is generally high (10.30 to 10.60), according to the amount of sugar contained in the *urine*; only exceptionally, in very diluted urine and with the excretion of small amounts of sugar, on the contrary it is abnormally low, according to my own experience and that of others, being 10.10 and below. But in a practico-diagnostic respect it is important, at all events, that in diabetes mellitus, in contrast to other diseases, the specific gravity is high in spite of the large amounts of urine.

The *excretion of nitrogen is decidedly increased* in the diabetic, primarily because the patients partake of more albumin in the food than do normal persons. It is not strange, therefore, that the *excretion of urea* under such circumstances should appear *enormously increased* (in one of my cases it amounted to 150 grammes in twenty-four hours). A disintegration of organic albumin, which is possibly due to the affection itself, i. e., caused by toxic substances injuring the protoplasm, becomes manifest in some cases of severe diabetes, and with this an increased N-excretion. However, this questionable source of a more marked loss of albumin occurs but very rarely to a subordinate degree. Similar to the excretion of urea, that of *ammonium* is likewise *greater* in the urine of diabetics (in a case of my observation about three times) than under normal conditions; and this is also true of the excreted amounts of the *sulphates* and *phosphates*. In contrast to this, the *excretion of uric acid* is not markedly increased, as a rule; only in some cases is it actually higher, and then the excretion of sugar is accompanied with that of urinary gravel and uric-acid calculi. Perhaps the function of the liver has become insufficient in two directions in such cases, in that not only the formation of glycogen from the sugar of food and its retention in the liver, but also the oxidation of the uric acid which is brought to the liver (from the spleen and the lymphatic apparatus as the result of the decomposition of nucleins), is impaired.

Acetone, Oxybutyric Acid, etc.—There can be no doubt that, further, a marked *acetone* reaction is often noted in the urine of diabetes. Since it has been shown that acetone occurs, at least in traces, in every normal urine (v. Jaksch), and in various affections (in psychoses, in carcinoma, in conditions of inanition, in fever, etc.) an increased acetonuria develops, the demonstration of acetone in the urine of diabetics has lost much of its diagnostic importance which was greatly ventilated formerly. Nevertheless, the excretion of *large* amounts of acetone in the urine of diabetics is not an indifferent phenomenon, as a decidedly increased acetonuria occurs especially in severe cases of diabetes, and in these cases, as it appears, represents a specific characteristic disturbance of metabolism. Unquestionably the production of acetone in the organism is in close relation to two other substances found in the u
betics, *oxybutyric acid* and *aceto-acetic acid*. The former, the

β—Oxybutyric acid: $\begin{matrix}CH_3\\CHOH\\CH_2\\COOH\end{matrix}$ by oxidation forms aceto-acetic acid $\begin{matrix}CH_3\\CO\\CH_2\\COOH\end{matrix}$

and the latter very readily decomposes into acetone, $CH_3 — CO — CH_3$, and carbonic acid, CO_2. It may be readily assumed, therefore, and no good reason contradicts this assumption, that these acids are really the primary stages in the acetone production in the organism and that, occasionally, the diabetic "acidosis" forms at first oxybutyric acid, and from this, by oxidation, aceto-acetic acid and acetone are formed.

The normal individual oxidizes the acetone bodies which arise in the organism almost completely, so that only traces of acetone are noted uncombusted in the urine. However, all three acetone bodies are found in the urine so soon as abnormal conditions of nutrition arise in the body, as in hunger (in healthy individuals), also in all possible affections that markedly impair nutrition, especially also in infectious febrile diseases; in very large amounts, however, only in diabetes. The more oxybutyric acid develops in the body, the sooner its appearance in the urine is to be expected; if oxybutyric acid is formed in smaller amounts, the acid itself will still be oxidized; its nearest derivatives of oxidation, however, aceto-acetic acid and acetone, are excreted in the urine without undergoing combustion. The *source* of the acetone bodies is acknowledgedly not, as was formerly believed, in the carbohydrates (the administration of carbohydrates, on the contrary, diminishes the excretion of acetone), neither is it likely to be in the albumin bodies, but most probably in the *fatty acids* which become free in the decomposition of fat.

The appearance of aceto-acetic acid and of oxybutyric acid in the urine (also other acids, an æthyliden lactic acid which turns to the left, α-oxypropionic acid, and transitory fatty acids have been found in the urine of diabetics) is the sign of *beginning acedosis* and manifests itself by an *increase of the excretion of ammonium* in the urine, in that the acids which appear in the blood are combined with ammonium.

The demonstration of *aceto-acetic* acid is accomplished by Gerhardt's reaction with iron chloride, which develops a dark-red colour of the urine. The presence of β-oxybutyric acid in the urine may be proved or, at least, made very likely in that the urine, in spite of large amounts of sugar being demonstrated by trituration or fermentation, does not turn sufficiently to the right (in that a portion of the action towards the right is suspended and becomes latent as a result of oxybutyric acid which causes a turning to the left). It is of practical importance, that, as long as, with a strict diet, only acetone is excreted, the diabetic case in question is a mild one. If, however, with a strict diet, excretion of acetone and aceto-acetic acid (eventually also of oxybutyric acid, but at most of small amounts) takes place, the case is a medium severe one; in severe cases, finally, very large amounts of acetone, especially also much oxybutyric acid, are excreted, and this excessive excretion of acetone bodies can no longer be diminished by the addition of carbohydrates to the food.

Albuminuria.—A combination of melituria and *albuminuria* is not rare. The latter may occur in various ways. In certain cases it is only due, apparently, to a functional paresis of the epithelia which are overtaxed by the diabetes; this view is favoured by the fact that even quite a marked excretion of albumin, with a change of diet in the sense of a strict meat diet, may completely disappear in a few days, according to my experience. In other cases, however, as the results of autopsies teach us, albuminuria is the result of a contracted kidney. The latter condition, in those cases in which the development of diabetes is due to gout or arteriosclerosis, may be looked upon as a coeffect of these pathological factors; in other cases the albumin is due to the large amounts of sugar circulating in the blood and to other irritating substances which are produced as a result of a disturbance of metabolism in diabetes and which irritate the parenchyma of the kidneys. The albuminuria may alternate with melituria and, after the permanent disappearance of the latter, albuminuria may continue as the expression of a nephritis.

Polyuria.—An especially important diagnostic sign of diabetes mellitus is the *increase in the* [illegible]ch goes hand in hand with the

excretion of sugar. In by far the greatest majority of cases this symptom of diabetes mellitus is well developed (three thousand to twenty thousand cc. daily quantity). However, the cases in which the amount of urine, in spite of quite a marked amount of sugar in the urine, is not increased, are very much more frequent than is usually supposed, according to the observations of others and my own (*diabetes decipiens*). In such cases we are dealing partly with transitory glycosuria, but partly also with permanent conditions, cases of true diabetes mellitus, the severest forms of which, exceptionally, even from beginning to end, run their course without showing polyuria. The amount of water excreted in the urine in general is parallel with the excretion of sugar and with the large quantities of fluid which the diabetic usually consumes. Still, there are also cases in which the amount of water excreted in the urine occasionally exceeds the quantity of fluid taken in the nourishment. Unquestionably, in these cases a part of the water which is excreted is furnished by the decomposition of tissue which occurs in the affection. In some few cases a simple polyuria may for a time precede the appearance of glycosuria.

In connection with the marked diuresis is the fact that less water is evaporated by the diabetic than by the healthy individual. *He, therefore, sweats less,* complains of dryness in the mouth and pharynx, and is tortured by unquenchable *thirst.*

Cutaneous Changes.—Besides dryness and exfoliation, on the part of the *skin* there is in some diabetic patients a pronounced tendency to *furunculosis.* This furunculosis is occasionally the first well-marked symptom that causes the physician to examine the urine for sugar. Other cases present itching of the skin, urticaria, stubborn eczema, pemphigus, defluvium capilitii and desquamation of the nails. Especially characteristic is the tendency of the skin and deeper tissues to the *formation of gangrene*; this is shown partly by the fact that wounds heal badly, respectively become gangrenous, partly by the appearance of spontaneous gangrene in individual toes or in entire extremities. Occasionally, especially lately, a marked *bronzing* of the skin has been observed ("bronzed" (!) diabetes). This condition has particularly been found in cases of diabetes which were combined with a (hypertrophic) pigment cirrhosis of the liver, and appears to depend upon a siderosis of the skin, to which also corresponds a more marked presence of iron in the internal organs.

Changes in the Respiratory and Circulatory Apparatus, etc.—Quite common in the course of diabetes are changes in the *respiratory organs.* The fruit-like (acetone) smell arising from the mouth of some diabetics is absolutely valueless in the diagnosis; important, however, especially for the determination of the prognosis of the individual case, is the unquestioned greater predisposition of the diabetic to *phthisis* and *pulmonary gangrene.* The severe disturbance of metabolism, which is at the foundation of diabetes, is the cause of the markedly diffused vascular changes which occur in the skin and also in the pulmonary tissue; they manifest themselves in that irritations affecting them are followed by an insufficient reaction and give rise to partial or complete mortification of the tissues, furnishing a favourable soil for the tubercle bacilli or the gangrene

ferment to exert their deleterious action and to disseminate. The unfavourable course of pneumonia in diabetics is well known. "*Diabetic dyspnœa,*" i. e., the paroxysmal increase of the depth and frequency of the respiration, will be referred to in the discussion of diabetic coma.

In connection with the damage to nutrition due to the diabetic process is probably also the *insufficiency of the activity of the* (occasionally hypertrophied) *heart,* showing itself by shortness of breath, syncope, and cardiac asthma. The same cause probably produces *arteriosclerosis* which develops early in such cases; but, on the other hand, it is also possible that arteriosclerosis forms the primary process, and, as has been frequently observed, arteriosclerotic changes of the vessels of the pancreas give rise to a chronic interstitial pancreatitis, causing diabetes as a result of this. Apoplexy, intermittent claudication, myocarditis, chronic nephritis, etc., occur as special sequelæ of arteriosclerosis in the course of diabetes. The *blood* of diabetic patients is concentrated, shows an amount of sugar of from 0.2 to 0.5 per cent and more, and is said to show (especially in cases of diabetic coma) diminished alkalinity.

Symptoms on the Part of the Digestive Tract.—Phenomena on the part of the *digestive tract,* apart from the first portions of the same, are rare in the course of diabetes. *Gastric* digestion is most often normal, in spite of the often enormously increased amount of food taken; gastritis, ulcer of the stomach, gastric atony or permanent dynamic gastrectasis may develop in rare cases. *Constipation* is the rule, on account of the great amount of water which is excreted by the kidneys; intercurrently diarrhœa may occur, in the course of which the excretion of sugar in the urine diminishes, whereas the thin white fæces contain sugar. Diarrhœa is generally a dangerous complication in diabetes, as, according to experiments, it may usher in diabetic coma. Of importance, on account of the possible relation of the pancreas to the origin of the diabetes, is the question regarding the amount of fat in the excrements. In some cases, as I can verify, more fat is present in the diabetic than in a normal individual taking the same quantity of nourishment; but this is by no means regularly the case. Alterations in the *liver,* which can be clinically determined, are not rare. Relatively frequent is the combination of diabetes with *cirrhosis of the liver*; as an expression of the diabetic process we also note marked hyperæmia of the liver which shows itself clinically by an increase in the volume of the organ. The changes in the *first portions of the digestive canal* are rather constant; dry tongue, acid reaction of the saliva, stomatitis, caries of the teeth, the latter being a pathological symptom that occasionally gives rise to the first suspicion of diabetes and is thus particularly of value if a rapidly developed caries of the teeth occurs in persons who previously have had entirely normal teeth.

The *urogenital system* frequently shows symptoms in diabetes: Pyelitis, cystitis, balanitis, vaginitis and pruritus pudendorum, the result of the development of fungi. Contracted kidney and albuminuria, which develop relatively frequently in diabetes, have already been described.

Alterations on the Part of the Nervous System.—The symptoms *on the part of the nervous system* are manifold. Besides the more general phe-

nomena, such as lassitude, headache, vertigo, sleepy sensations, mental depression, or even well-developed melancholia, loss of memory, etc., we find in the diabetic a very frequent tendency to *neuralgias* (occipital, trigeminus neuralgias, etc.). There appears to be a special preference in this affection to produce neuralgia of the sciatic nerve.

Occasionally neuralgias are the result of the changed nutrition which occurs in diabetes, and of the abnormal reaction of the nerves dependent upon this, or, also, as the observations of v. Ziemssen teach, of chronic neuritis. In rare cases (I have seen only one) sciatica may be the primary and melituria the secondary condition, and this may perhaps be explained in the meaning of the physiologico-experimental fact that a lesion of the sciatic nerve in animals gives rise to melituria.

Symptoms of *neuritis* (circumscribed and multiple) arise especially often in the course of diabetes, such as sensory disturbances (paræsthesia, anæsthesia, hyperæsthesia, pains in the calves of the legs, etc.), motor disturbances (paralysis, especially of the lower extremities, with muscular atrophy and DeR and ataxia), vasomotor and trophic disturbances (desquamation of the nails, herpes, pemphigus, local œdema, etc.). Occasionally these neuritic phenomena are combined, forming a symptom-complex which resembles tabes (*"pseudotabes diabetica"*).

More constant than all the phenomena just described, occasionally betraying the presence of diabetes at the onset of the affection, is the *diminution in the sexual desire, impotence* and *loss* of the *patellar reflex*. The latter symptom occurs in about one third of the cases; the patellar reflexes are more often obliterated in the severe than in the mild cases; occasionally it is retained particularly in the severest cases, so that the presence or absence of the patellar reflexes is without importance in a diagnostico-prognostic respect. As a cause of the diminution of the patella tendon reflex we may assume either neuritic changes in the course of the crural nerves (lately determined by Eichhorst), or functional disturbances in the reaction of the affected nerve tracts due to toxæmic causes. Even an increase of the tendon reflexes has been observed in rare cases of diabetes.

The following symptoms which sometimes occur in the course of diabetes point to focal affection in the brain: Aphasia, monoplegia, hemiplegia, spasms, etc. Nevertheless, anatomically demonstrable disturbances in the nervous system are but rarely found at autopsies of diabetics. A well-marked dilatation of the small vessels in the medulla oblongata has been noted comparatively oftenest. In some cases there were tumours and softening in the region of the fourth ventricle, encephalomalacia and hæmorrhages due to syphilis or atheromatous endarteritis, degenerations of the posterior columns of the spinal cord, etc.

Symptoms on the Part of the Visual Apparatus.—The diagnosis of diabetes mellitus is markedly complemented by an examination of the eyes; in fact, it is not so rare that the disturbances of sight are the first symptoms which cause the patient to consult the physician. Most frequently these are due to an *opacity of the lens in the form of cataract*, which, as a rule, readily disseminates to both eyes. Furthermore, a so-called *retinitis diabetica*, most frequently bilateral, may develop, which shows great resemblance to albuminuric retinitis (which may even be present simultaneously with nephritis). On the one hand, we find the region of the macula affected in the form of light or white glistening patches or small rounded hæmorrhages, and, on the other hand, we note hæmorrhages in various parts of the retina. Opacities of the crystalline lens and even copious hæmorrhages in this region are quite frequent. In some cases the optic phenomena of a well-distributed, superficial, so-called *chorioretinitis* are observed; it is then a question of a glycogenic degeneration of the chorio-capillaris, respectively of the coarser vessels of the chorioid; the same degeneration

affects the vessels of the retina in cases of retinitis diabetica. A diminution in sight without ophthalmoscopic findings is also referred to *disease of the optic nerve*; frequently a central scotoma is present. Atrophy of the optic nerve, stasis papilla, the occurrence of hemianopsia, etc., should be regarded as the results of diabetes due to cerebral affections. This is also true of paralysis of the external muscles of the eye, which occurs in the course of diabetes, but it must be decided in each individual case whether or not a cerebral affection is the cause of diabetes.

The *loss of accommodation* in diabetes may be ascribed to general muscular weakness, the result of diabetic cachexia; the appearance of *keratitis parenchymatosa* as well as that of iritis to the previously mentioned glycogenic degeneration of the marginal network of the cornea, respectively of the vessels of the iris. The development of a so-called *keratitis neuroparalytica* and that of so-called *hæmorrhagic retinitis* due to a marantic thrombosis of the *central veins* of the retina, are also consequences of the cachexia which arises in the course of diabetes, and in such cases death, as a rule, respectively diabetic coma, will soon supervene.

Diabetic Coma.—The most important symptom-complex on the part of the nervous system in connection with diabetes, which points to a severe disturbance of the former, in the majority of cases terminating in death, is *diabetic coma*. This is ushered in, after excesses, exhausting bodily muscular movements or very mild acute affections, intestinal catarrh, angina, etc., have preceded, by headache, vertigo, unrest, delirium, fear, sensations resembling intoxication by alcohol, until *insensibility* and *collapse* gradually become prominent. In other cases the coma occurs abruptly, being accompanied with cyanosis, dilatation of the pupils, small pulse, a falling temperature (to away below normal ranges), and, above all, with a peculiar *alteration in respiration—deep, noisy (usually rapid) respiratory excursus*, without stridor, whereas no hindrance to the respiration can be demonstrated. This dyspnœic, deep breathing is usually the first sign of developing coma, but the latter may remain absent in spite of the well-marked presence of dyspnœa; the prognosis of such cases is *more favourable* than that of the cases in which coma and dyspnœa occur simultaneously. In the profound coma, occasionally even after muscular contractions have appeared, death occurs sometimes very rapidly, at other times only several days after the onset of the coma. As the breath of such patients usually reveals a fruit-like smell, and as the urine, almost without exception, shows the iron chloride reaction, for a time the development of diabetic coma was thought to be due to an auto-intoxication with acetone. However, the experimental investigations regarding the relative non-toxic properties of acetone, as well as the clinical observations that marked acetonuria and diaceturia may exist for months without producing the faintest signs of coma, contradict this assumption. It seems more likely that oxybutyric acid is in closer relation to the development of coma. Whether acids which actually occur in conspicuously large amounts in the metabolism of the diabetic (aceto-acetic acid, β-oxybutyric acid, æthyliden lactic acid, volatile fatty acid), may, in the end, give rise to a poisoning of the organism (acid intoxication) and produce coma, or whether other toxic products of the metabolism in diabetes play a more important *rôle*, cannot be determined for the present.

Acid Intoxication.—If animals are poisoned by acids, a peculiar symptom-complex appears which resembles diabetic coma, consisting principally in dyspnœa and

insensibility. With this we observe that the ingestion of acids into the circulation increases excretion of ammonium in the urine (parallel to the acid intake), and this is also the case in diabetics in whom large amounts of oxybutyric acid appear in the urine. We may assume that this occurs because a part of the ammonium, before its change into urea, is utilized by the acids, serving to neutralize them. This prevents the alteration of the alkalinity of the blood and of the tissues, preventing their damage. Such an increase of the excretion of ammonium in the urine undoubtedly occurs in diabetics, as was first proved by Boussingault, lately by *me* and by Hallervorden. But the satiation of the excess of acid by the disposable ammonium has its limits; as soon as the acid production exceeds a certain degree, which may very easily occur in the diabetic, the possibility arises that the acid intoxication may reveal itself in a pathological manner and give rise to coma (diabeticum). In favour of this assumption are the experiences in the laboratory as well as at the bedside, that the coma may eventually be cured by the administration of large amounts of alkalies.

Varieties of Diabetic Coma.—From the ordinary forms of diabetic coma, the origin of which may be referred to the action of toxines, those cases are to be separated diagnostically which arise suddenly with the symptoms of somnolence and collapse, rapidly leading to death, in which, however, the condition resembling alcoholic intoxication and the so-called "deep, accelerated respiration" are absent. The cause of this form of coma is to be looked for in *paralysis of the heart* (Frerichs), which may depend upon a fatty degeneration of the heart muscle, as has been determined anatomically.

In other cases in which, besides the melituria, excretion of albumin and the sequelæ of contracted kidney are present, the diabetic coma, especially its milder forms (headache, vertigo, asthma, etc.), dare not be mistaken for *uræmic intoxication*, which is very apt to occur under such circumstances. It is true, the differential diagnosis between the two conditions cannot be determined with certainty in such cases (which is not to be wondered at, on account of the manifold picture of uræmia). However, certain symptoms in the clinical picture, especially vomiting, diminished excretion of urine and well-developed convulsions, favour the diagnosis of uræmia. The condition which has lately been described as occurring in the prodromal stage of the coma, the excretion of large amounts of casts in the urine (Külz, Sandmeyer) may give rise to the development of uræmia as well as of toxæmic coma. Finally, there are also comatose conditions which arise on account of *cerebral apoplexy* complicating diabetes, which must be diagnostically separated from diabetic coma.

Different Varieties of Diabetes.—It is of great importance strictly to differentiate two varieties of diabetes—the *mild* and the *severe* forms—which has become quite usual, following Seegen's proposal.

Severe Form.—In the *severe* form, in spite of the complete absence of carbohydrates from the food, the sugar does not disappear, or at most but transitorily, from the urine, i. e., the *tolerance for carbohydrates is lost in the severe form*. At the same time, upon prolonged observation, the *progressive* character of the process is readily noted, the tendency to a rapid course, to increasing decomposition and acidosis. The diabetics suffering from the severe forms are, as a rule, young persons, not over forty years of age.

Mild Form.—*In the mild form of diabetes,* by the administration of a diet in which carbohydrates are absent, a complete absence of sugar excretion from the urine can usually be rapidly attained, and, furthermore, quite a considerable tolerance for carbohydrates may even occur, so that the patients, even with the *permanent* ingestion of but *few* carbohydrates, may furnish a urine which is entirely free from sugar, or at most shows but traces of it. Above all, the progressive nature of the affection, the rapid deterioration, the growing disturbances in the consumption of carbohydrates, are absent in the truly mild cases, and acidosis only arises occasionally and transitorily.

The two forms may merge into each other; a glycosuria, which has begun as a mild form, relatively often becomes severe—by intercurrent diseases, psychical emotion, but, above all, by continued neglect of a proper diet. The transition of the severe form into the mild, however, is very rare.

A *strict* separation of the severe from the mild variety of glycosuria as two pathogenically different forms of diabetes is no longer possible, according to our lately acquired knowledge regarding the origin of the disease and the alteration of the carbohydrate metabolism in the same. Since we know that, upon the extirpation of the pancreas, the experimenter may optionally produce a mild or a severe form of diabetes, according to whether the gland is removed for the greatest part or only small portions of it are extirpated (see below), the uniform cause of both forms can no longer be doubted. It depends, above all, upon the severity of the damage to the carbohydrate metabolism in the given case.

Carbohydrate Metabolism.—To decide this question, the carbohydrate economy of the organism must be briefly considered (compare introduction to chapter on Metabolism). The *carbohydrates* which are brought to the liver by the blood of the portal vein, i. e., the grape sugar, levulose (fruit sugar), etc., which are derived from the amylum of the food, are transformed in the liver into glycogen which is stored here as transitory reserve material, to enter the blood as sugar and to be utilized for the production of heat and external work. The muscles derive their necessary sugar from the glycogen supply of the liver, and are capable of transforming this again into glycogen and to store it, so that next to the liver they form the principal glycogen deposit in the body. The *albumin* products, on account of the N-less atom groups which become free on decomposition of the albumin products, serve as glycogen formers; for it is possible at any time to produce the formation of glycogen in animals by feeding them on albumin products. Finally, according to the latest discoveries, it is at least likely that, in case of necessity, *fat* (in the liver) may also be changed into grape sugar and be utilized as for the production of labour and heat.

In what manner the sugar molecule is decomposed in the organism has not been determined with certainty. The end-products are known, CO_2 and H_2O; but, unquestionably, in between these are a great number of intermediate products of sugar oxidation. A part of the sugar appears first to split into *lactic acid*, which is indicated by the appearance of this acid upon the consumption of glycogen in the active muscle; another part of the sugar, as we may assume, according to the newest investigations of P. Mayer, becomes first *glycuronic acid* which, again, becomes oxidized to oxalic acid; forming, by further oxidation, carbonic acid and water:

Grape sugar $C_6H_{12}O_6 = COH(CHOH)4 - CH_2OH$ becomes:

(a) Lactic acid $C_3H_6O_3 = CH_3 - CHOH - COOH$ (α-oxypropionic acid) with decomposition of the carbon nucleus;

(b) Glycuronic acid $C_6H_{10}O_7 = COH - (CHOH)4 - COOH$ becomes by further oxidation:

Oxalic acid $C_2H_2O_4 = COOH - COOH$ becomes by oxidation: carbonic acid and water $= 2CO_2 + H_2O$.

In favour of the correctness of these assumptions is the fact that also in normal urine glycuronic acid combined with phenol is excreted in slight quantities, which have escaped further oxidation, and, further, that this is still more the case in cases of severe disturbances of respiration and circulation, i. e., in cases in which the energy of oxidation is unquestionably diminished, and, finally, that also in the urine of diabetics glycuronic acid occurs in larger amounts than in normal urine. The fact is that in diabetes a part of the sugar is not at all oxidized and in another but very incompletely, i. e., only to its first combustion product, and in the same manner it may be explained if oxalic acid also occurs in large amounts in the urine of diabetics, besides glycuronic acid. This is especially to be expected as soon as the assimilation of sugar improves in the course of the affection, and with it the sugar, although it does not oxidize to CO_2 and H_2O, at least combusts up to the point of both those products of incomplete oxidation, to glycuronic and oxalic acids.

Theory of Diabetes.—In reviewing the above-outlined principles of metabolism of carbohydrates, it becomes clear at once that not only *one* disturbance, but various alterations in the carbohydrate economy give rise to glycosuria. The first step in the method of the utilization of sugar, i. e., an *insufficiency or loss of the glycogenic function of the liver* (eventually also of the muscles) must be taken into consideration. That, as a result of this, a flooding of the blood with sugar may occur, and, with this, glycosuria, is plain. In fact, in the diabetic as well as in animals which were made diabetic by extirpation of the pancreas, the liver is poor in glycogen, and it was not possible, even by feeding such animals on plentiful quantities of amylum, to produce an accumulation of glycogen in the liver.

That insufficiency in the storing of glycogen may give rise to glycosuria has been determined, further, by a great number of experiments and clinical observations. Above all, the celebrated experiment of Cl. Bernard has shown that, by puncturing and wounding the point of the calamus scriptorius in animals, a glycosuria of brief duration may be produced. After its termination the liver is shown to be free from glycogen; if the organ, before the experiment is tried, is made free from glycogen, the urine does not contain sugar. The action of this puncture is explained in that, as a consequence, due to nervous influences, directly or indirectly by suddenly arising circulatory changes, the supply of glycogen is at once emptied into the blood as sugar, giving rise to hyperglycæmia and glycosuria. Similar to the effect of puncture are also, as was shown later on, the lesion of other points in the central and peripheral nervous systems, and also the action of a number of poisons (CO, curare, morphine, amyl nitrite, strychnine, etc.). A glycosuria of brief duration occurred in all of these experiments, but sugar did not appear in the urine if there was no glycogen in the body. Clinical experiences also teach that transitory glycosurias occur in various affections of the nervous system and in intoxications, and that, if the glycogenic function is latently insufficient in some affections, insufficiency may become manifest, and glycosuria may be readily produced if 150 grammes of grape sugar are administered to the patient, which quantity can be surely assimilated by a healthy person, but which, in disease, if the production of glycogen or the accumulation of glycogen has been damaged, will show the appearance of sugar in the urine.

If, therefore, insufficiency or absence of glycogenesis cannot be denied in human diabetics, this does not by any means explain the nature of diabetes. For, although it is obvious that the rapid flooding of the blood

with sugar (provided its change into glycogen in the liver is prevented) causes excretion of the sugar in the kidneys during digestion, yet it would not be plain why glycosuria should cease in the interval between the different administrations of glycogen formers, why glycosuria should be almost always absent in severe affections of the liver, etc. We are rather forced to assume *that in diabetes, besides insufficient glycogenesis,* also *an insufficiency in the tissues exists regarding the combustion of sugar.* It is true, the diabetic has the same property of assimilating oxygen in the same amounts as a normal person, and he can just as readily oxidize various substances which are taken into the organism; but, on the other hand, an increase of CO_2 excretion upon administration of carbohydrates is almost entirely absent in the diabetic, and especially when dextrose is ingested, whereas the administration of levulose, which is notoriously utilized completely in diabetes, produces the normal increase of the CO_2 excretion, as in the normal individual. It follows that the diabetic is incapable of assimilating sugar, in spite of the fact that it is a substance which is very easily oxidized.

The diabetic, then, cannot fully utilize sugar—neither in the sense of glycogen formation or accumulation, nor in a sense of its utilization as fuel, nor, finally, as a fat-forming substance, as this method in utilizing the carbohydrates in the animal economy is likewise not employed by the diabetic. The above-mentioned abnormal condition of the activity of the tissues in the combustion of sugar is due primarily to *the absence of the pancreatic function,* either because an anatomically demonstrable, extensive affection of the pancreas exists or because by a weakening of the nervous function of the pancreatic cells the normal metabolism of sugar is damaged.

Influence of the Pancreas upon the Production of Diabetes.—As is well known, v. Mering and Minkowski, by their masterful experiments, have proved beyond doubt that the complete extirpation of the pancreas in the dog results in the excretion of sugar, and that a permanent glycosuria with all the phenomena of true diabetes, i. e., with a large amount of grape sugar, and the appearance of acetone, β-oxybutyric acid, etc., in the urine, increase of thirst and hunger, emaciation, general loss of power and diabetic coma. Preventing the pancreatic juice from entering the intestine, by ligating the duct of the pancreas, does not give rise to diabetes, neither does the partial removal of the gland. Only after nine tenths of the gland have been extirpated, does diabetes occur, but then only in the mild form, i. e., the excretion of sugar is relatively slight and only appears after the administration of carbohydrates. If, later, the last tenth is also destroyed, or if it is also, subsequently, removed experimentally, then diabetes occurs in the severe form. There can be no doubt, after the results of the experiment, that the pancreas is necessary in the economy for the normal metabolism of carbohydrates. It is certain that glycogen is no longer deposited in the liver and muscles after extirpation of the pancreas, even if large amounts of starch, respectively sugar, are administered (excepting levulose, which is utilized in the normal manner). It has also been proved that it is impossible for the animal that has been rendered diabetic by the extirpation of the pancreas, to oxidize sugar. *How,* after the removal of the pancreas, this damage to sugar metabolism arises, cannot be determined with certainty. Perhaps the pancreas, under normal conditions, by an "internal secretion," furnishes a substance which, carried to the liver, accomplishes the production and accumulation of glycogen in the latter organ, or effects the destruction of the sugar molecule in the blood and in the tissues ("glycolytic ferment," Lépine). Whether this function of

the pancreas, which is necessary for the normal transformation of carbohydrates in the body, occurs in this manner or not, it is, finally, obviously influenced by the *nervous system*, in that the pancreas cells receive their nervous influences probably from the central nervous system over certain peripheral nerve tracts.

Differential Diagnosis.—If we adhere to the facts that only the positive proof of sugar in the urine will determine the diagnosis of diabetes mellitus, and that cases of *latent* diabetes are readily disclosed as such by the administration of food rich in carbohydrates which at once produces considerable amounts of sugar to appear in the urine, differentio-diagnostic points scarcely arise in the individual case. Confusing diabetes mellitus with chronic nephritis, diabetes insipidus, and some other (symptomatic) polyurias, which have in common with diabetes mellitus only the excretion of large amounts of urine, naturally must not occur. Such errors in diagnosis are readily excluded if the necessary caution is taken in examining the urine for sugar (therefore, for example, not being satisfied with the positive reaction of reducing substances in the urine), noting the specific gravity of the urine, testing the tendon reflexes, making an ophthalmoscopic examination, etc. If the presence of sugar in the urine has been determined with certainty, only one question arises, viz., whether we are dealing with actual diabetes mellitus or with a simple glycosuria or, perhaps, a lactosuria, these being differentio-diagnostic questions the decision of which does not give rise to difficulties provided the previously given points of differentiation are taken into consideration, the repetition of which I deem unnecessary. It shall only be briefly mentioned that in certain cases diabetes mellitus may be determined at once by the amount of sugar that is excreted, a large amount, exceeding 2 per cent, being exclusively in favour of true diabetes mellitus. On the other hand, we can readily convince ourselves, if there is a large clinical material at the disposal of the investigator, that very often even smallest percentages (0.3 to 0.5 per cent) are noted in cases which, according to their course and their symptoms, unquestionably are true cases of diabetes.

DIABETES INSIPIDUS

The diagnosis of *diabetes insipidus* is very easy. The affection is characterized by a *permanently increased diuresis*, so that *large amounts* (three to ten litres and more, in a case of Trousseau up to 43 litres per day) *of a pale, clear urine, free from sugar and albumin*, of low specific gravity (10.01 to 10.10), are discharged, and, secondarily, depending upon this, dryness of the skin and polydipsia, very rarely bulimia.

Differential Diagnosis between Diabetes Insipidus and Chronic Nephritis.—It is scarcely worth while to discuss the differential diagnosis between diabetes mellitus or chronic nephritis and diabetes insipidus, the affections only having in common the increased amount of urine voided, as a careful examination of urine at once clears up the situation. Occasionally, it is true, so small amounts of albumin are excreted in the course of *chronic nephritis* that it can be determined only upon exact analyses. Upon a more frequent examination of the urine, however, the albumin

reaction, even in such cases, is determined, apart from the fact that the other symptoms of chronic nephritis: Tension in the pulse, hypertrophy of the heart, retinitis, etc., show the true nature of the affection. On the other hand, albumin is sometimes also excreted in the urine in very small amounts in diabetes insipidus. The albumin in this case might be explained in that (similar to the condition in diabetes mellitus), as a result of the colossal overexertion of the epithelial function, the retention of albumin finally suffers temporarily. However, these cases are very rare; I have seen but one case of diabetes insipidus accompanied with transitory albuminuria. The diagnosis of diabetes insipidus in such cases is only allowable if, after careful, often repeated examinations of the heart, of the eye-ground, of the sphygmographic pulse tracings, and of the urinary sediment, a chronic nephritis may be excluded with certainty.

Diabetes Mellitus.—Diabetes insipidus can be confounded with diabetes mellitus only if an accidental alimentary glycosuria appears in the course of the former. The very transitory character of the excretion of sugar in such cases, however, clears the situation. It occurs occasionally, as has been observed by competent investigators, that diabetes insipidus ushers in diabetes mellitus or follows it, and, further, that both varieties of diabetes alternate.

Symptomatic Polyuria.—If, then, the differential diagnosis between diabetes insipidus and chronic nephritis or diabetes mellitus rarely gives rise to actual difficulties, the question whether diabetes insipidus or a *symptomatic polyuria* is present in the individual case, can often only be decided with the greatest care. In this connection the ætiology of the individual case and the duration of the polyuria, which in the case of diabetes insipidus will always have been a long one, often having existed for several decades, must be primarily regarded.

If large amounts of fluid enter the circulation, the excretion of water in the kidneys is markedly increased, in that the glomerular epithelia are stimulated to greater activity. This explains the transitory polyurias with temporarily increased intake of fluid, and also the permanent (secondary) polyuria in *polydipsia*. *That this latter condition in the case of diabetes insipidus is not the primary one*, but that the great thirst from which the diabetic suffers is *a consequence* of the large amounts of fluid excreted in the kidneys, may be regarded as certain. For the patients suffering from diabetes insipidus void, through their morbidly overexerted kidneys, more water with the urine than a healthy control person during the time with the same amount of fluid intake; further, during thirst the blood of the patient becomes thicker and water is drawn from the blood and the tissues, in severe cases to so great an amount that dangerous symptoms appear: Headache, pains in the limbs, cardiac asthma, collapse, etc.

In rare cases *polydipsia* appears to arise as a *primary affection*. The malady could be recognised as such in that the blood does not become thicker upon withdrawal of fluid intake, as is the case in diabetes insipidus. Further, the secretion of sweat (respectively perspiratio insensibilis) is below par in the case of the diabetic, not so in polydipsia, as there is no reason for the non-use of this road of excretion for the increased amount of fluid that is partaken of.

Symptomatic polyuria occurs, further, in the resorption of large amounts of fluid that have been produced or collected in the organism, as in the resorption of œdema, transudates, pleuritic exudates, etc., and in the convalescence from febrile affections. This latter ("*epicritical*") form of polyuria is due to the excretion, after the cessation of the fever, of large quantities of fluid and of urea and sodium chloride (the

latter two having a specifically diuretic action), which were retained during the fever. It shall be briefly mentioned, in connection with this, that we must always consider in milder grades of polyuria whether the increased excretion of urine may not be due to an abuse of any of the various substances which irritate the secretory elements of the kidney and which thus act diuretically (caffeine, spices, alkaline mineral waters, etc.).

As numerous physiological examinations have shown, the variations in the excretion of urine are, to the greatest extent, dependent upon the influence of the nervous system upon the circulatory apparatus of the kidneys. Whereas a severance as well as an irritation of the cervical cord results in inhibition of the secretion of urine—the latter operation, on account of a simultaneous irritation of the splanchnic nerve—the severance of this (vascular) renal nerve produces polyuria. The centre of the renal vaso-motor nerves is situated in the floor of the fourth ventricle (somewhat below the melituria centre), a lesion (puncture) of which will give rise to marked diuresis. It is self-evident, therefore, that polyuria occurs occasionally in the course of the various nervous diseases, above all in hysteria. If the pathological action upon the nervous system is of a *permanent* nature, as in disease of the medulla oblongata and of the neighbouring parts of the central nervous system, continuous polyuria is the natural result; symptomatic, transitory polyuria has now been transformed into a permanent condition, diabetes insipidus.

Whether such a permanent polyuria which can be ascribed to such lasting changes in the central nervous system, may be identified as a true diabetes insipidus, the ætiology of which, as a rule, is not clear, appears to me not to have been decided as yet. I should think that this would be permissible only if the symptoms of such a "*chronic symptomatic polyuria,*" which depend upon anatomically demonstrable changes of the central nervous system, are fully congruent to the symptoms of diabetes insipidus. The latter are: Abnormally and permanently increased diuresis, i. e., excretion of large quantities of water in the urine, much larger than in the normal individual taking the same amount of fluid, secondary polydipsia, pale colour and low specific gravity of the urine, slight increase or normal condition of the excretion of the solid constituents (urea, sulphuric acid, phosphoric acid, etc.), subnormal temperature. The other symptoms which are occasionally observed in diabetes insipidus, such as salivation and ophthalmoscopic changes, are in the main rare complications of the affection, probably co-effects of the same pathological cause which act from the central nervous system, giving rise to a chronic polyuria.

OBESITY—ADIPOSITAS UNIVERSALIS

Obesity, respectively the tendency to accumulate fat, is obvious at once, so that the recognition of this disease does not present any difficulties and is usually made correctly even by laymen. The gain in weight and the increase in size are unmistakable in well-developed cases; the fat which accumulates in abnormal amounts is especially prominent in the cheeks, the chin, the breasts, the abdomen and the buttocks. The head and the upper part of the chest, on account of the change of the centre of gravity, are thrown back, the gait becomes wabbly, and bodily movements are slow and made with difficulty. The cutaneous surfaces, on account of the increased secretion of the sebaceous glands and the sweat follicles, have a

greasy, shiny appearance and are moist; the obese person always suffers from *shortness of breath.*

Dyspnœa, Cardiac Asthma.—This last condition is due to various causes. The great deposit of fat in the mediastinum prevents the expansion of the lungs; this function is even more limited by the insufficient movement of the diaphragm, which may itself be a result of accumulation of intra-abdominal masses of fat, and of the development of a fatty liver (going hand in hand with an increase in the volume of the liver). Besides, the *activity of the heart* in obesity early shows signs of insufficiency.

As was explicitly discussed in the chapter on Cardiac Asthma, the pressure in the pulmonary vessels is increased on a marked increase, as well as in a pronounced decrease, of blood pressure in the arteries, the filling of the capillaries in the pulmonary alveoli is more marked, this causing their walls to become more tense, giving rise to difficulty in respiration. These conditions occur even in healthy persons during marked muscular activity, thus giving rise to an increase of blood pressure; but they are readily compensated by corresponding, more marked respirations, which in the case of the normal person are only transitory. The condition is different, however, in *fatty* individuals! Even the impaired expansion of the thoracic wall which has become massive, and the deficient movement of the diaphragm downward, prevent compensation. But, now, the expected insufficiency of the heart also becomes active regarding the difficulty in respiration. This insufficiency is the result of the extra great demands which, in the course of the disease, are made upon the activity of the heart in the obese individual. The excessive taking of food, and especially of drink, will cause (quite apart from an increase in blood pressure which arises and which may be compensated by the adaptation of the vessels to their more marked condition of fulness as well as by the increased secretion and diffusion) that the heart must continuously pump greater amounts of blood, and in time *tires*. If we consider, besides, that intemperate persons, especially, habitually partake, with their food, of various kinds of cardiac stimulants, such as tea, coffee, and alcohol, it at once becomes clear that in all directions the heart is forced to stronger labour. For a time the heart supplies the greater demands by a more marked activity, but the condition cannot remain so, as the heart does not have sufficient pauses of rest, and fatty degeneration of the organ (see "Fatty Heart") sooner or later arises. The results of *insufficiency of cardiac activity* which are produced in this manner manifest themselves in disturbances of respiration: In the mildest cases in such a manner that *dyspnœa occurs in an unpleasant manner upon a more marked bodily movement*, as, on account of the previously mentioned mechanical impediments to the expansion of the lungs in fatty individuals, an increase of pressure in the lesser circulation supervenes, which is combined with an unavoidable insufficiency of the cardiac activity. The affected individuals, therefore, are always predisposed to dyspnœa; if, besides, a more or less *sudden increase of the demand upon the cardiac activity* arises, for example as the result of muscular exertion of a high grade, the result will be so rapid an increase in pressure in the pulmonary vascular system that the *cardiac asthma* occurs suddenly. In fact, acute pulmonary œdema may arise in such cases, *sudden death* being the result.

Disturbances of Cardiac Activity.—Besides the action upon the respiration previously mentioned, the results of disturbed cardiac activity in the obese produce other marked symptoms. The pulse is rarely retarded, usually increased, arrhythmic; cardiac palpitation, angina pectoris, and, especially, Cheyne-Stokes respiration, pseudo-apoplexies occur intercurrently in the pathological picture. As the cardiac power sinks more and more, stasis phenomena appear, œdema, gastric and intestinal catarrhs, hæmorrhages, etc.; in short, the entire symptom-complex develops which we have previously described as characteristic of *fatty degeneration of the heart* (see "Fatty Degeneration of the Heart").

Fatty Liver.—The *liver* is relatively often enlarged not only by engorgement but also by a deposit of fat; this latter condition, as long as it occurs without symptoms of stasis, may be recognised by the soft consistence of the liver. For the other symptoms I refer to the previously mentioned diagnostic rules which have been laid down in the discussion of fatty liver.

A certain predisposition to *nervousness* cannot be mistaken in the obese, but, above all, the patients show a marked *absence of resistance of the organism*, so that intercurrent febrile affections, like pneumonia, etc., become very dangerous to them.

The Condition of the Urine.—*The condition of the urine* shows changes from the normal. Apart from an eventual excretion of sugar (the result of a relatively frequent combination of obesity and diabetes mellitus), the *excretion of water* may be changed. *Frequently, in comparison to the amount of fluid taken in, too little fluid is excreted.* The cause of this is either a more marked excretion of sweat, to which the obese patient is predisposed, or to insufficiency, respectively fatty degeneration of the heart. In the latter case a slowing of the blood stream in the capillaries, especially in the glomeruli, arises. The consequence of this is a diminution in the amount of urine and a marked flow of fluid into the lymph channels, the tissues and, eventually, into the cavities of the body. Such a disproportion between the intake and excretion of fluids in favour of the former is, as a rule, in favour of a disturbance of the circulation and a faultiness in the auto-regulation of the watery constituents of the blood and of the tissues combined with obesity, a condition in obesity that carries danger with it, and, on this account, must be well considered in prognosis.

Metabolism in Obesity.—This leads us to speak of metabolism in obesity, which shall be explained, at least in its principal points, as an insight into the same makes the diagnosis of obesity very much easier.

In the physiological introduction to the diseases of metabolism (which see) it was explained that metabolism depends upon the *amount of labour* performed (muscular and digestive labour), and, further, upon the amount of *heat given off*, but, above all, upon the amount and variety of the *food* partaken of. We have seen that, in muscular work, especially material which is free from nitrogen (glycogen, respectively carbohydrates, and fats) are consumed, and the latter is also the case in the function of the human organism in maintaining its individual temperature at a constant height as against the temperature of external conditions. It follows that, upon limitation of muscular work, and in the presence of a warm external temperature, the consumption of fats and carbohydrates, *ceteris paribus* becomes less, which eventually may give rise to an accumulation of fat. However, the latter depends to a much greater extent upon the amount and composition of *food* partaken of.

Food materials consist in the main, as is well known, of albumin bodies, fats and carbohydrates. Of these, the albumin products are most easily attacked by the activity of the cells, then the carbohydrates (especially sugar), and then fat.

Formation and Accumulation of Fat in the Body.—In the decomposition of albumin bodies, the split particles of the same which contain N change into NH_3, urea, etc., whereas those that do not contain N are transformed partly into lactic acid and are further oxidized into its end-products, carbonic acid and water, partly they are changed into fat. Whether this latter process occurs in the body has lately become questionable again, in contrast to the opinions formed previously. However, the *fat of food and the carbohydrates may be regarded as the certain sources of the produc-*

tion of fat, respectively accumulation of fat in the body. We may assume this because fat which is foreign to the composition of the affected organism, may be made to accumulate by feeding and, as Rosenfeld has shown, finds its way into the internal organs. On the other hand, the transformation of carbohydrates into fat has been positively ascertained in that, by the administration of large quantities of carbohydrates and little meat, accumulation of fat can be attained in quantities which, even if the fat actually were due to the N-free atom rests originating in the meat, the quantity would be entirely too large to come from this source. We may presume it as certain that, as soon as the intake of carbohydrates is in excess of the demand, the superfluous carbohydrates (inasmuch as they do not find room in the glycogen deposits) are transformed into fat and that this fat is accumulated.

It is certain that, if only albuminoid substances, even in largest amounts, are given in the nourishment of man, complete nutrition does not occur, and especially is there never an *accumulation* of fat. But this takes place at once if sufficient quantities of fat or carbohydrates, or both simultaneously, are administered in a plentiful albumin diet. It has been established that the fat of food is less easily decomposed than atom groups which are N-free and rich in carbon and which split off from the decomposition of albumin substances. If sufficient amounts are available of the latter, the decomposition of the fat of nourishment which is taken with albumin is diminished, and this then accumulates. This is still more the case if carbohydrates are simultaneously available for decomposition. For these, also, are more readily combustible than fat, and therefore cause in the economy, besides a saving of albumin metabolism (and that a relatively more marked one than by fat), also a saving in the decomposition of fat, so that, if carbohydrates alone are added to the amounts of meat and carbohydrates which maintain the standard of the body in albumin and fat upon a constant height, an accumulation of fat occurs.

There can be no doubt, according to above explanations, that a great number of obese subjects owe their corpulence exclusively to food which is habitually chosen in an excessive and improper manner. It appears to me especially noteworthy in a clinical respect that also quite a different variety of obesity of the body occurs, in that, namely, a *deposit of fat with loss of albumin* occasionally takes place. This may be expected, *when, besides an increase in the intake of fat and carbohydrates, the amount of albumin taken in the food is reduced to a very small quantity.* For, although the metabolism of albumin is reduced by the increased intake of fat and carbohydrates, i. e., albumin is saved and the body thus able, even with a slighter albumin metabolism, to maintain its equilibrium, the organic albumin must decompose in the *course of time* upon a markedly reduced administration of albumin in the food. An organism that has become poor in albumin unquestionably has less energy in metabolism than an organism which is rich in albumin. This causes the decomposition of fat to become less and, in the presence of plentiful fat and carbohydrates in the nutrition, *fat is accumulated. The body of an individual that has become fat in this manner is weaker, therefore; fatty degeneration occurring more and more at the cost of the albumin on hand,* and this type of obesity represents a severe form compared to that variety which arises with a plentiful administration of albumin, besides fat and carbohydrates.

Conditions favouring Obesity.—If, accordingly, excessive ingestion of food, especially of fat producers, is adapted to cause a pathological accumulation of fat, the occurrence of obesity is still further favoured by quite a number of *predisposing causes.* Among these primarily is the habitual use of *alcoholic liquors* in large amounts. Alcoholic liquors supply the organism with an N-free substance rich in calories (100 alcohol furnish 700 calories), which is almost completely burned up in the system. This at once shows that the accumulation of fat is favoured by the administration of alcoholic liquors, and especially is this the case if an alcoholic liquor is taken which, like beer, contains, besides, considerable quantities of sugar and dextrin (5 to 10 per cent besides 3 to 4 per cent of alcohol). Another factor which favours the development of obesity, as has been previously mentioned, is the *absence of bodily movements.* As marked muscular labour, as we know, increases the consumption of calories up to 3,000 to 3,500 cal. (instead of 2,000 to 2,500 cal.), and as the muscle, in spite of its development from albuminoid substances, defrays its fuel

directly or indirectly from N-free combinations, it is obvious that the material necessary to accomplish this may also be taken from the fat; in fact, in marked muscular labour fat decomposes. If muscular movement is absent, fat is accumulated if the amount of nourishment remains the same or is increased. Thus it is obvious that *persons who lead an idle life, taking slight exercise and sleeping much, show a tendency to become fat. Sexual abstinence* also appears to favour obesity, which is shown by the fact that women, in the years of the climacteric period, incline to corpulence, and that castration in animal and man conspicuously favours the accumulation of fat. It is certain, furthermore, *that the action of cold upon the skin increases the decomposition of fat*, residence in a warm climate, accordingly, favours the accumulation of fat. Finally, it has been determined by thousands of clinico-therapeutic investigations, since Oertel has called attention to the condition, that the *withdrawal of water* (in a manner which cannot be explained) *unquestionably increases the destruction of fat.*

After obesity has once occurred, this itself favours an increase of corpulency, partly on account of muscular movements becoming more difficult, partly also because the withdrawal of heat from the surface of the body through the thick deposits of fat (being poor conductors of heat) is diminished, this causing a relatively slighter combustion of material for the maintenance of the body temperature. Finally, in my opinion, the explanation of the occurrence of obesity may also depend upon a *faulty tendency of cell activity*, especially a *pathological diminution of cell energy*. This may be partly hereditary and partly acquired later, and cause that, in spite of not essentially increased ingestion of food, accumulation of fat occurs, as even this amount of nourishment is relatively too great on account of the diminished decomposition energy of the cells. Estimations of the O_2 intake and CO_2 excretion in obese subjects have as yet given no markedly demonstrating results regarding the energy of oxidation, but this much has been noted that the amount of O_2 intake and CO_2 excretion is conspicuously low compared to the normal figures which apply to the body weight in healthy persons. A retardation of metabolism in the sense just described is certainly true in some cases of obesity. This is also favoured by the experience that, in some families more readily than in others, the individual members of the family are more inclined to obesity and that the condition persists in spite of the fact that the affected individuals are properly nourished and do not partake of more mental or bodily rest than persons in other families, which, in spite of taking large quantities of food, etc., remain thin. The fact, also, that in some cases obesity occurs in early youth and can scarcely be controlled and, in spite of abatement of appetite, does not diminish but even increases, should be explained in this manner, and likewise the observation of stock raisers that some races of domestic animals are more readily fattened than others. It appears to me that in obesity there is a similar action of cell activity as in diabetes mellitus, in so far as the cells, after having once shown a faulty action, whether it be due to an inherited disposition or to later influences, can with the greatest difficulty be made to abandon this faulty tendency, again to assume it if predisposing factors present themselves.

Differential Diagnosis.—But very few morbid conditions are to be regarded in the *differential diagnosis* of obesity, which may simulate adipositas, as the clinical picture of general lipomatosis is so characteristic that it can scarcely be confused with other conditions. It is sufficient, therefore, to mention that obesity may be simulated by pseudo-muscular hypertrophy, by general cutaneous emphysema, and by cutaneous œdema. Cutaneous emphysema is characterized by the appearance of crepitation, œdema by a doughy condition of the skin, with the well-known results of palpation, allowing us to recognise these conditions readily. It is important, it is true, always to remember the previously mentioned, relatively frequent combination of adipositas and œdema in the later stages of obesity. But even here it is usually not difficult to determine the presence of obesity besides œdema from the relatively slight pitting impression of the skin upon pressure (compared to the marked grade of general swelling of the surface of the body). It requires but slight attention to differentiate ascites from a belly which is rich in fat, or to recognise a combination of the latter with ascites. The differentio-diagnostic points regarding the difference between fatty liver and fatty heart compared with other affections of the liver and heart have been explicitly described in other places.

ADIPOSIS DOLOROSA—DERCUM'S DISEASE

[This affection, first described by Dercum in 1888, is characterized by lipomatosis which may be either diffuse or in tumours. The masses of fat are not noted in the face, on the hands, or feet. Pain, especially over the nerve trunks, is present, with anæsthesia in the contiguous areas. Psychical disturbances are also present. The affection occurs in both sexes, but it is more frequent in females. At autopsies, lesions have been found in the nerve trunks, the thyreoid gland, and the adipose tissues.]

GOUT—ARTHRITIS

Gout, which, as is known for a century, is due to the deposit of *uric acid* in the tissues, especially in the joints, is a true disease of metabolism, which, similar to obesity just described, depends primarily upon improper, as a rule too large amounts of, food and, above all, upon the use of alcohol; chronic lead-poisoning also plays an important part in the ætiology of gout.

The Attack of Gout.—The most important clinical symptoms, dominating the entire pathological picture, are the "*attacks of gout.*" These are usually preceded by prodromes, such as dyspepsia, jaundice, lassitude, oppression, cardiac palpitation, insomnia, paræsthesia, drawing pains in the limbs, pruritus, spasm in the calves of the legs, œsophagismus—symptoms which have no diagnostic value, as they are of a very vague nature and may even be absent altogether, so that the first attack may appear abruptly. The latter is characterized by an especially painful affection of the joints; almost always (certainly in about two thirds of the cases) *one* joint is attacked first and, as a rule, the *metatarso-phalangeal joint of one big toe* (*podagra*). The affected joint swells (partly from an effusion of a turbid-serous fluid into the joint), the skin that covers it becomes hot, red, tense, and slightly œdematous. With this there is moderate fever that reaches its maximum after a few days, terminating by lysis, with the presence of sweating; a falling temperature rarely occurs by crisis. The attack, which almost always sets in at night, ameliorates during the day to recur the next night. After about a week the attacks disappear and recur only after shorter (lasting for weeks) or longer (lasting for years) pauses, usually in spring or autumn, first in the previously affected joint, later in other joints: the wrist joint, the joints of the fingers, especially of the thumbs (chiragra), the knee joint (gonagra), in the clavicular articulations (cleidagra), etc. In the later stages of gout *one* joint is no longer affected, but several joints are attacked. The swelling disappears in the articular region, with the disappearance of the attacks of pain, with the symptoms of itching and desquamation of the skin. The skin appears to be affected to a very high degree in gout, and frequently seems to be primarily inflamed, sometimes even, without the joint itself being affected, it is primarily attacked by deposits of gout in the region of the joint. The gout tophi, surrounded by dilated veins, are deposited especially in the subcutaneous cellular tissue of the *auricle of the ear* (in about one fourth of the cases); but the soft parts surrounding the joints of the extremities

are more often the primary seat of these gouty nodules than the joints proper.

These gouty nodules usually do not affect the cartilage and capsule of the joint until they have further increased in size. Enlargement of the liver and spleen, cardiac palpitation, and arrhythmia of the pulse usually appear with the attack of gout.

Gouty Finger.—In persons that have suffered from typical attacks of gout as well as in those who are affected by chronic, irregular gout, especially among females, we often find a peculiar deformity of the finger: Thickening of the end phalangeal joints of the fingers (Heberden's nodes), the size of a pea, produced by proliferation of the bone (not deposits of urates), which are brought in genetic connection with gout; whether properly or not has not yet been decided.

Urinary Changes.—Of importance for the diagnosis are the *changes of the urine.* The amount of uric acid excreted in the urine, according to former quantitative estimations, has been found to be reduced during the time of the attack, to increase again later. But as Garrod and various other investigators, by means of the thread experiment, have shown an increased quantity of uric acid in the blood, the conclusion was obvious *that in gout the excretion of uric acid is interfered with in the attack, i. e., that the arthritic attack consists in the periodical engorgement of the blood with uric acid.*

Nature of Gout.—However, later investigations (W. His, Vogel, Magnus-Levy and others) have shown that the conditions are not so simple. It was confirmed, it is true, that *uric acid is permanently present in large quantities in the blood of patients with gout,* whereas the blood of normal individuals contains uric acid in but minimal quantities. But also in other diseases, in leucæmia and lead-poisoning, in diseases of the kidney, etc., greater amounts than normal of uric acid were found in the blood, so that the presence of larger quantities of uric acid in the blood of gouty individuals cannot be looked upon as pathognomonic. Besides, in contrast to the views since Garrod's investigations, *no increase in the amount of uric acid in the blood could be determined during the attack,* and also the old teaching of Garrod regarding the *diminished alkalinity of the blood in gouty patients* and the inability of the blood, in connection with this, to maintain the urate salts in solution, has been shown to be incorrect.

Regarding the condition of excretion of uric acid in the urine in gout, according to the latest investigations it appears that the excretion of uric acid probably diminishes a few days *before* the attack; certainly, however, *during,* respectively immediately *after,* the attack it shows an *increase,* the latter being dependent upon the total albumin metabolism. A *nitrogen retention* occurs in certain periods of the affection, which does not go hand in hand with a corresponding increase in weight; according to the cases investigated up to now, this phenomenon cannot be looked upon as specifically belonging to the gouty process.

Without doubt, metabolism, especially of nitrogen, is disordered in gout, so that periods of nitrogen accumulation alternate with periods of nitrogen loss; combined with this is probably a diminished use of nutrition in the digestive canal. It may

be looked upon as certain, furthermore, that, compared with the normal conditi there is a permanent increase of uric acid in the blood. This fact, although increased amount of uric acid in the blood is also noted in other affections, must considered in the explanation of the nature of gout. As an overproportion of u acid in gouty individuals is at least very unlikely, a closer adhesion of the uric a in the blood must be thought of, or we must bear in mind that the kidneys are o able to excrete it after the "uric-acid tension" in the blood has reached a m marked degree. This may be true of purely functional disturbances of the renal-c activity, but also is probably favoured by the granular atrophy of the kidneys (below) which is so common in gout, belonging to the nature of the affection, a which appears to develop gradually from an affection of the epithelia of the con luted tubules.

A portion of the excess of uric acid circulating in the blood is deposited in t joints, the skin, etc., and produces here as a chemical poison, as the injection expe ments with acid urate of sodium of Freudweiler and His have shown, necros inflammation and connective-tissue encapsulation of the uric-acid deposits in t tissues. Why, however, in gout the urate salts are attracted from the blood to t joints, etc., and deposited here, has not yet been explained. In fact, we must adn in general that the points of support for the origin and nature of gout are more of negative kind and do not yet allow a satisfactory gout theory.

Chronic Gout.—The diagnosis of the characteristic attacks of *arthrit gout* which have just been described, is easy in general, on account of t very typical course of the paroxysms (*typical, regular, acute gout*).

The diagnosis becomes more and more difficult if, in the course time, the acute attacks of gout lose their regularity regarding their a pearance and duration, and take on a more insidious character, or if a thritic gout (as occurs especially in debilitated individuals or as is noted i the course of chronic lead intoxication) runs its course from the ons without well-developed attacks (*chronic, atypical gout*). The more fr quently the paroxysm is repeated in acute gout, the more chronic the malad becomes and the more the nodules consisting of urates are develope Their growth is accompanied with continually more marked function disturbances and deformity of the joints, ankyloses, subluxations, etc mucous follicles and tendons are also affected by the gouty changes. Sin ilar to the cartilage, the skin may also become necrotic as the result urate infiltration; this gives rise to the formation of fissures, fistulæ an ulcers, from which necrotic shreds of tissue and uric acid, respectivel sodium urate, are excreted so that, as in one of my cases, the entire extren ity may appear as if covered with urate crystals.

Eye Affections.—Certain *affections of the eye* are also brought in relation gout, especially a circumscribed *inflammation of the sclera*, which occurs mostly i attacks. This arises between the border of the cornea and the equator; if there is hump-like elevation at the inflamed area of the corium, this may be looked upon a small gout nodule. Occasionally, in the interval between the attacks, inflammator areas are noted around the entire cornea (*scleritis migrans*). In connection with di ease of the sclera, so-called *sclero-keratitis* or an *iritis* with their sequelæ may arise but *iritis* may also occur of itself. The occurrence of *cataract* and *glaucoma* in gou is undoubtedly in connection with circulatory disturbances which may be due to a atheromatous condition of the vessels which is so frequently observed in gout.

Visceral Gout.—The *internal organs* are very commonly affected in the course c gout (*visceral gout*). *Dyspeptic symptoms, cardialgias, vomiting*, etc., are either c a purely nervous nature, i. e., due to a disturbance of nutrition affecting also th

nervous system, or the result of dietetic excesses to which patients suffering from gout are very liable and which may have even previously given rise to production of gout. The same as the cardialgias are partly due to abnormal irritation of the nervous system caused by the gouty process, so are also the neuralgias and neurasthenic conditions which are frequently seen in the course of gout. Pains radiating from the vertebral column as the results of *gouty spondylitis* (especially of the cervical vertebræ) or of a secretion of urates into the membranes of the spinal cord have been noted in some rare cases. Whether the headache, the attacks of syncope, psychical disturbances and epileptiform attacks, which are observed in gout, may be ascribed to disturbances of nutrition of the brain ("*cerebral gout*"), I leave undecided. It appears more certain to me that the gouty process is the cause of the *vascular changes* which occur in the course of gouty, even in youthful individuals. It is simply a question in such cases of *atheromatous* changes which are produced in such instances of general nutritive disturbances. It is true that uric-acid deposits have been chemically determined in the concretions of the arterial walls, but these must certainly be looked upon as very exceptional cases. In a case of classic gout in a young man whose peripheral arteries showed thick lime plates in the walls, I was unable, in spite of the greatest efforts, to find uric acid. Going hand in hand with atheromatosis of the vessels are valvular affections and fatty degeneration of the heart, and atheroma of the coronary arteries, with their consequences, etc. In the veins, in connection with the attacks of gout, *thrombosis* and *phlebitis* have been observed. On the part of the *respiratory organs* there are noted, in connection with gout, asthma, pleurisy and *pneumonia*, so that the latter occurred during the course of the attack of gout or immediately following it, and recurred in later attacks. It is possible that the germs of inflammation are more liable to become lodged and more active in tissues which, under the influence of gout, are abnormally nourished, and that the tendency of gouty individuals to be very subject to inflammation in general is in connection with this fact.

However, a *certain* proof of a specific gouty character has not been determined for any of the previously mentioned symptoms of visceral gout. The diagnosis of visceral gout, therefore, is based almost exclusively upon the history and upon the observation that the onset of the affection in the particular organ coincided with the attack of gout, or it may be supported only by the clinical experience that gouty persons are relatively more often affected by the above-named diseases than other patients. Such diagnoses are not of great value; it is advisable, in general, to make a diagnosis of "gouty dyspepsia," "arthritic neuralgia," etc., only if these pathological phenomena affect patients with unquestionable gout and other causes for their production can be excluded.

Renal Gout.—More important, because belonging specifically to gout, are the changes in the *kidneys*. These are occasionally absent, it is true, even after arthritic gout has been present for years; but this is rare. In other cases, on the contrary, the "*renal gout*" dominates the pathological picture to such an extent that the kidney appears as the *primarily* affected organ (Ebstein), in fact, that even advanced forms of renal gout are found at autopsies before other signs of gout, especially arthritic affections, had appeared. The gouty kidney is anatomically characterized as an organ affected by chronic interstitial inflammation, while all deposits of urates may be absent in the kidney. The occurrence of a nephritis in such cases, similar to that of diffused vascular sclerosis, must be regarded as due to the gouty deterioration of the constitution. Usually, however, the secreted uric acid lodges in the Malpighian bodies and uriniferous tubules or in necrotic areas of the renal tissues of the cortex, but especially in the medullary substance. The results of this *gouty nephritis* are the same as those of the usual form of chronic nephritis: Albuminuria, hypertrophy of

the left ventricle, uræmia, etc. The presence of a nephritis uratica, ther fore, cannot be determined from the symptoms of a renal affection; but may be diagnosticated with a great deal of probability if the symptoms c contracted kidney supervene in the course of well-characterized arthriti gout.

Primary renal gout may be assumed with probability if the histor the demonstration of a hereditary disposition, of an irregular and opulen manner of living or a markedly well-developed lead-intoxication, whic must be added to the causes of gout, favour the development of arthriti and if symptoms of the renal affection are those of interstitial nephriti The probable diagnosis gains in certainty if typical joint affections, etc appear.

Differential Diagnosis.—We now come to the discussion of the differ ential diagnosis, i. e., to the discussion of the diagnostic factors whic enable us to recognise gouty affections from conditions which run thei course with similar symptoms.

Arthritic gout may be especially confused with other acute inflamma tory processes, or, if the gouty arthritic changes have become chronic (i. e when typical attacks no longer take place or if, in such cases, these attack from the onset have occurred in a dragging manner) with chronic articu lar inflammations, especially with *arthritis deformans*—among the lait the latter condition is commonly confounded with gout. Apart from th ætiology, which shows that arthritis deformans, in contrast to true gout, i overwhelmingly more frequent in women, among the poor (arthritis pau perum) and in individuals that are poorly nourished, and that, in th majority of cases, it represents decidedly a disease of the aged, the devel opment and appearance of the arthritic affection differ greatly from gout The pains are less spontaneous in arthritis deformans, occurring princi pally upon movements; the deformities in the joints, as a rule, are mor uniform and symmetrical, and especially at the hands, the point of selec tion of the disease, they are quite typical, manifesting themselves as sub luxations in the metacarpo-phalangeal articulations and by lateral dislo cations of the fingers towards the radial, or, more frequently, towards th ulnar, side, and by atrophy of the interossei: the thickening of the carti lages and of the articular capsules is much more uniform than in gout Obviously, all these differential points only refer to those rare cases ir which the onset of gout is insidious, without the appearance of typica attacks. If the latter are pronounced or, besides the alterations in th joints, if typical attacks preceded the appearance of chronic articular gout at least in its early stages, there can be no question of a confusion of both affections. Gouty nodes in the skin, in the auricles of the ear, etc., whic may be present besides the arthritic affection, will eventually at once give the proper trend to the diagnosis.

The necessary points regarding the differentio-diagnostic discrimina tion between individual expressions of *visceral gout* and other morbid con ditions not dependent upon a gouty basis, have already been described.

In connection with constitutional diseases, the diagnosis of some affec tions shall be considered which occupy a median position between these

and local affections of certain tissues, especially of the bones. These are *chronic arthritis, arthritis deformans, rhachitis, osteomalacia* [and *osteitis deformans*]. It will be sufficient to give the main points regarding the diagnosis of these affections, as lately these diseases have been more and more relegated to the domains of surgery and gynæcology.

ARTHRITIS DEFORMANS; CHRONIC ARTICULAR RHEUMATISM

Chronic Articular Rheumatism.—In some few rare cases chronic arthritic affections follow immediately upon attacks of acute rheumatic fever, so that these cases are designated by a special name "chronic articular rheumatism," as it is assumed that the process depends upon a continued action of the morbid condition giving rise to acute polyarthritis. In other cases *chronic rheumatic arthritis* develops very gradually, is not in connection with acute rheumatic fever, but deserves the epithet rheumatic in that it is in an undoubted genetic connection particularly with the effects of "refrigeration," of living in damp houses, etc. Anatomically the disease is characterized by inflammation of the synovial membranes, proliferation and *connective-tissue changes of the cartilages*, giving rise to a gradually increasing connective-tissue (finally bony) *ankylosis of the joint.*

Arthritis Deformans.—In contrast to this, in "*arthritis deformans*," besides a wasting of cartilage and bone (softening process), hyperplastic proliferation of the cartilage and bone occurs, this giving rise to an increasing limitation in the movability of the joints (deformity pseudo-ankylosis), placing the affected part of the extremity in the most varied abnormal positions: hyperextension, subluxation, etc. (deformity subluxation). Arthritis deformans occurs especially in the *hip joint*, in its multi-articular form most frequently in the *joints of the fingers and toes*; in the main it is a *disease of old age.*

The diagnosis of *arthritis deformans* is based upon the course of the disease and the easily demonstrable changes in the cartilaginous-bony processes of the joints. The course is characterized in that it shows an *exquisitely chronic, progressive character of the articular affection*, a gradually increasing stiffness and painfulness of the joints, so that motion in certain directions becomes impossible in time. If the joints are passively moved, creaky sounds are heard. Numerous proliferations in the ends of the bones and cartilages are readily noted on inspection and palpation of the affected joint; the muscles which are usually active in joint movements gradually atrophy (especially the interossei and the muscles of the shoulders).

Differential Diagnosis.—Arthritis deformans is very difficult to differentiate, during its early stages, from chronic articular rheumatism. The *differential diagnosis becomes very much easier after connective-tissue ankylosis has occurred, which without exception is absent in the case of arthritis deformans*; in such cases, upon passive movements of the joints, grinding sounds are heard due to the tearing of the connective-tissue threads. Later the ankyloses become bony, and then can only with difficulty or not at all (in complete loss of motion of the joint affected in arthritis deformans) be differentiated from pseudo-deformity ankylosis which is formed by the proliferation process in arthritis deformans (in which an *adhesion* of the joint ends does *not* occur). Neither is a differential diagnosis possible when chronic articular rheumatism or arthritis affects the vertebral articulations. The anatomical changes and sequelæ in the vertebral articulations are different, however, in both processes in that, in arthritis deformans, nodular bony clasps develop upon the border between two vertebral articulations, whereas in chronic rheumatism the vertebral articulations gradually show a connective tissue and later a bony coalescence of the vertebral articulations among each other. In both cases, however, the final result is the same, namely, an absolute immovability of the entire vertebral column, and a clinical differentiation of both conditions from each other is impossible.

In fact, lately the strict division of "chronic articular rheumatism" from "arthritis deformans" has been given up, and both processes have been designated

by the uniform name of "multiple destructive chronic arthritic inflammation." The same as micro-organisms are looked upon as the cause of the inflammation in acute rheumatic fever, so it has also been assumed of this chronic destructive arthritic inflammation that an infective virus gives rise to the affection; accordingly, bacilli have been looked for in the affected joints and have actually been found. But whether these bacilli which were demonstrated in the synovial fluid of the joints in arthritis deformans by Schüller and others, are the actual causes of the disease or whether we are dealing with a form of trophoneuroses, the *future* will decide.

OSTEOMALACIA

Bony Changes.—The diagnosis of osteomalacia is based upon the flexibility of the bony framework in consequence of a decalcification of the bones, which arises in such a manner that the halisteresis begins from the medullary cavity at the periphery of the osseous trabeculæ and assumes greater dimensions so that, finally, the basic substance of the bone decalcifies more and more, becoming homogeneous, thready or even mucous, and the medullary substance hyperplastic, lymphoid, showing engorgement and containing much pigment. The external cortex of the bone resists this continuous decomposing process the longest and forms a shell which contains the decalcified portions; if this also decalcifies and if the structure of the lamella disappears more and more, the bone becomes soft and is flexible in all directions. In the early stages of decalcification the bone is at least more brittle than normal and, in keeping with this, is more liable to fracture and infraction. The flexibility of the bony structure is instrumental that, on account of the weight of the body and the traction of the muscles, even externally markedly visible alterations in the form of the skeleton become prominent: Bending and infraction of the extremities, kyphoses, lordoses, and scolioses of the vertebral column, but, above all, a curvature of the pelvis, which is characterized in that the promontory is displaced downward and forward and approximates the point of the sacrum and the symphysis, that the venters of the ilium are bent, the os pubis is kinked, and the symphysis becomes beak-shaped, bending forward. This gives the entrance to the osteomalacic pelvis the popular shape of the heart, Y-form, which may be easily determined by an accurate examination. The ends of the joint are never affected by this process.

On account of the malformation and bending of the bones, the patients gradually sink within themselves as it were, becoming smaller and deformed. These deformities develop gradually with *pains*, which are deep-seated, being felt at the sacrum, back and in the extremities, and which are usually increased by pressure upon the bone. Gradually the gait is altered; it becomes swaying and difficult, small steps are taken with a simultaneous forcing forward of the pelvis, or it is waddling ("duck gait"). In a number of cases the difficulty in gait may be due to alterations in the muscles which have been determined on several occasions (fatty atrophy) but the connection of which with the bone affection is not clear as yet. The curvature of the spinal column and of the thorax cause the intensity of the respiratory movements to diminish; the masses of mucus which collect in the bronchial tubes are expectorated with difficulty, so that in this manner a catarrhal pneumonia and asphyxia may develop, in the course of which the fatal termination of the affection occurs. The deformity of the pelvis is especially deleterious during pregnancy, respectively during parturition, which need not be further explained. Of importance for the diagnosis is the comparison of frequency with which the individual bones are affected by osteomalacia. First in this connection are the pelvis and the vertebral column, then the thorax, the upper extremities, the lower extremities; most rarely the bones of the skull. Apart from the pains and disturbance in movement, the patients complain but little. Fever is rarely present; the functions of the internal organs are normal. Now and then fibrillary muscular contractions are noted. External irritations develop *painful contractions*, the genesis of which is unknown, in the muscles of the lower extremity especially in the *adductors*. As in the pathological picture which is occasionally observed even in the early stages of the affection, also pareses and increased tendon reflexes occur (especially in the muscles of the pelvis and of the thigh), the thought

arises that the central nervous system plays a *rôle* in the development of this peculiar, severe disturbance in metabolism of the bones.

The most frequent predisposing cause for the development of osteomalacia is pregnancy; each succeeding pregnancy increases the difficulty after the process, in the interval, has become quiescent or even may have improved some. Characteristic of this *puerperal form of osteomalacia* is the fact that the osseous changes always begin in the pelvis and only from this point attack the vertebral column upward, whereas in non-puerperal osteomalacia, which occurs in both sexes, the affection usually begins in the lower extremity. The process always runs its course without fever, being an especially chronic one.

Of greatest importance for the diagnosis would be the recognition of the cause of the disease. We possess, however, up to now but few points of support to explain the origin of osteomalacia. Nothing is gained with the assumption of a "general disturbance in metabolism," but neither will the setting up of a "lactic-acid theory" and similar hypotheses help us any. Upon a somewhat more stable basis rests the theory which Fehling has recently proposed to explain the development of puerperal osteomalacia. The observation that castration or the Porro operation, i. e., the amputation of the pregnant uterus with removal of both ovaries, cured the affection in osteomalacic women, simple Cæsarean section, however, not giving such a result, caused him to assume that osteomalacia is a trophoneurosis of the bones, being in a definite relation with the *ovaries*.

Urinary Changes.—Positive *urinary changes*, which would be of value in the diagnosis of the early stages of osteomalacia, are by no means constant. Thus the excretion of calcium in the urine, quite opposed to what might be expected, is by no means always increased, but has even been found diminished; in one of my recent cases, at certain times very little calcium was excreted, at other times, apparently in connection with the increased development of the process, abnormally large amounts of calcium were found. The excretion of phosphoric acid in the first stages of the disease has also been found increased. The excretion of uric acid was at times increased, at other times, as in one of my cases, diminished. Similar conditions prevail regarding the excretion of lactic acid in the urine, which, according to the almost generally accepted view, causes the solution of calcium salts in the bones. This acid has occasionally been found in the urine of patients affected by osteomalacia, and, further, its diminution up to almost complete disappearance during convalescence has been noted; but in other cases lactic acid has been looked for in vain in the urine. Occasionally, albumin, and especially albumose (which has been found in the osteomalacic bone marrow, as well as in the normal bone marrow), occurs in the urine; in other cases albumin is absent. However, the demonstration of albumosuria is without importance in osteomalacia, as albumoses are also found in the urine in quite a number of other affections.

Differential Diagnosis.—In the early stages of the disease, when only deep-seated pains and difficulties in movement are present, the infractions and curvatures of the bones, however, are absent, the (beginning) osteomalacia may be confounded with chronic rheumatism, bone syphilis, etc. A probable diagnosis is only allowable if these vague difficulties occur during pregnancy and continue or arise in connection with a birth which has just taken place. The suspicion of osteomalacia grows if in such cases the examination of the urine occasionally shows decided increase in calcium excretion and lactic acid. There can never be a question, however, of a certain diagnosis in this stage; the affection only becomes recognisable if the skeleton shows previously described characteristic changes. Then, however, osteomalacia can no longer be confused with any other affection, at most with *myelogenic sarcoma and diffuse malignant infiltration* of the bone, which, like osteomalacia, give rise to spontaneous fracture and curvature of the bones; but they run a much more rapid and more pernicious course. Osteomalacia cannot be confounded with *rickets*, as it occurs in later life (especially between the third and fourth decades of life), and only very exceptionally in early childhood, then appearing simultaneously with rickets. Besides, as is obvious from the following description of the diagnostically important points of rickets, the pathological picture varies so much from osteomalacia that a differential diagnosis between the two affections scarcely ever arises.

RHACHITIS; RICKETS

Rickets represents a disease of metabolism, showing itself in a disturbance of the normal growth of the bone, especially in an insufficient calcification, this causing a softening on the growing bone tissues.

Anatomical Changes of the Normal and Rhachitic Bone.—The *normal* growth of the bone lengthwise occurs in such a manner, that, in the area contiguous to the diaphysis of the epiphysis, a proliferation of the cartilage, especially of the cartilaginous cells, occurs with an *enchondral ossification* arising from the diaphysis. Accordingly, we find at the border between the epiphysis and the diaphysis two narrow zones: The *cartilaginous proliferation zone*, with an increase and enlargement of the cartilaginous cells becoming more marked towards the bony diaphysis, and a *calcification zone*, in which calcium salts are deposited into the basic substance of the cartilage and into the capsules of the cartilaginous cells. Into these calcified cartilaginous masses now also the bone marrow and the vascular twigs of the diaphysis shoot, dissolving the calcified cartilaginous ground substance of the cartilage with the exception of but a few small trabeculæ. Now the cartilaginous cavities are opened and the cartilaginous cells become free, which, mixed with the medullary cells of the bone, become bone formers—*osteoblasts*. The latter approximate, similarly to single layer epithelium, the previously mentioned trabeculæ of the calcified basic substance, and form young bones around the latter, giving rise to an increase in length of the diaphysis bone. In *thickness* these (long) bones normally have another kind of ossification process, *periosteal ossification*. In this process new bony tissue is continuously produced from the internal layer of the periosteum which is layered around the finished bone, whereas in the internal part of the same bony substance is absorbed, which causes a dilatation of the medullary cavity. The bone formation in the *connective-tissue bones* (skull and bones of the face), finally, occurs in such a manner that bones are produced from connective tissue, that, especially at the borders of the bones, osteogenous tissue remains during the time of its growth, which permanently forms new bone.

Calcification of the newly formed bony masses is incomplete in rickets, both in the periosteal, bone-producing layer and in the zones of enchondral proliferation, respectively calcification. These latter are *broadened* with irregularly interspersed calcified areas; the medullary spaces do not, as is normal, reach only into the calcified cartilage, but farther. The resorption of bony substance in the medullary cavity, which normally keeps pace with the apposition of bone from periosteum, occurs to an excessive degree in the rhachitic bone. Similar conditions prevail regarding changes in the bony growth of the connective-tissue bones; the osteogenous tissue at the margins of the bones calcifies insufficiently, remains soft, and thus wide spaces develop between the individual bony parts.

Therefore, the rhachitic process is characterized in general, on the one hand, by production of osteoid, insufficiently calcified tissue and a permanence of the same, and, on the other hand, by increased osseous resorption. In consequence of the above-described disturbance in bone formation, very important changes in the skeleton occur in rhachitis, which are of importance in the diagnosis.

Alterations in the Skull.—The first phenomena appear in the skull and in the ribs. *In the skull*, even after the first few months of life, the rhachitic disease occurs as *craniotabes*; most marked are the changes in the *occiput* in the form of the "*soft occiput*" (Elsässer). On feeling the skull we find at the lambdal and sagittal sutures areas which have become soft on account of the disappearance of bony substance and which feel as thin as parchment. Their origin may be referred to the combined action of the rhachitic bone process and of the pressure upon the occiput in the recumbent posture; usually complete baldness designates the extent of the soft areas. The consequences of craniotabes are: Sensitiveness to external pressure, convulsions of all kinds, unrest, etc. The large fontanelle, which under normal conditions closes towards the middle of the second year of life, remains open longer, often

to the fourth year; likewise the various sutures of the skull, the coronal, the sagittal and lambdal, remain open unusually long, frequently for several years. The skull, by flattening of the occipital bone, thickening of the other bones of the cranium and especially by the prominence of the frontal and temporal protuberances, acquires a quadrangular form (tête carrée). The development of the maxillary bones also suffers from the influence of rickets. *The teeth* do not appear at the proper time, i. e., only in the course of the second year of life and then show an insufficient, irregular enamel formation; besides, the position of the teeth is greatly changed by the deformities of the maxilla. The lateral parts of the inferior maxilla from muscular action push inward, the anterior portion becomes flattened inwardly, so that the incisor teeth stand in a straight line and behind the teeth of the superior maxilla, especially as the anterior portion of this bone becomes angularly bent in the course of the affection and is forced forward; if the teeth do not find sufficient room during their eruption for a simultaneous production of a row of teeth, they are formed irregularly, standing especially behind each other.

Changes in the Thorax.—Of still greater importance for the diagnosis than the rhachitic changes in the cranium are the *deformities of the thorax* which develop a little later (in the second half of the first year of life and later): The swellings which occur at the boundary between cartilage and bone (the "*rhachitic rosary*"), the lateral bending of the thorax with beak-like projection of the sternal portion of the same (*pectus carinatum, chicken breast*), the kyphotic or scoliotic *curvature of the spinal column* and the excessive curvature of the clavicles.

Changes in the Extremities and in the Pelvis.—The *rhachitic deformities of the extremities* which occur about the end of the first year of life are characterized by *swelling of the epiphyses* and the *arch-like curvature of the different bones of the extremities*, which is brought about by the traction of the muscles on the soft bones. The deformities are especially frequent and well developed at the legs; the lower legs are bent outward, and in severe cases of rhachitis the femurs are also curved outward and convex dorsally (*bow-legs* or *genu varum*). The arms are also curved; the upper arms anteriorly and exteriorly, the forearm convexly towards the dorsal side. These deformities become still more conspicuous if infractions are added to these curvatures. In the *pelvis* the rhachitic malformation shows itself in that the sacrum sinks farther into the pelvic cavity, the venters of the ilium deviate anteriorly, and the acetabula are directed more forward. The rhachitic pelvis is usually flat, occasionally narrowed all over, as well as asymmetric; these changes may, in later life, act in a pernicious manner in possible labours.

The alterations of the bones are accompanied with *pains* in the bones upon external pressure, so that children, upon movement or when standing, cry out aloud and even in the recumbent posture, especially on account of cranial tabes, complain of pains.

Complementary Symptoms.—The above bony alterations in the skeleton give to the diagnosis of rhachitis its firm support. This is complemented by other pathological phenomena which, however, in comparison to the bony changes, are subordinate in a diagnostic respect. The condition of nutrition in rhachitic children is usually bad; *chronic intestinal catarrh*, which, as a rule, introduces the affection, contributes to the cachexia. The most important complications on the part of the respiratory organs are *bronchial catarrh* with its consequences (atelectasis and catarrhal pneumonia), *laryngospasm* (see Laryngospasm) and *tetany* (see Tetany); general convulsions, sweating, especially of the head, enlargement of the spleen and intermittent fever also belong to the common phenomena in the picture of rickets. The alterations of the urine are not characteristic, an increase in phosphoric acid and calcium excretion, which formerly was regarded as certain, has lately become questioned; however, an abnormally great excretion of calcium in the fæces has been demonstrated (Baginsky).

To formulate a theory regarding the pathogenesis of rickets upon this or another finding in the examination of urine or fæces, has not proved to be practical. The most likely view is the assumption of an *infectious* origin of rickets; in favour of this are: The alterations of the internal organs, which are present besides the disease of the bone, the affections of the digestive and of the respiratory tracts, as well as of

the nervous system, the frequently demonstrable enlargement of the spleen, the febrile attacks and the rheumatic polyarthritic phenomena, and, finally, the frequency of the affection dependent upon geographical and climatic conditions, etc.

Differential Diagnosis.—But very rarely are we in doubt as to whether rickets or another affection which resembles it in certain points is present. As long as the rhachitic bony changes are absent, as long, therefore, as only "premonitory symptoms" of rickets are present: Digestive disturbances, diarrhœa, enlargement of the spleen, intermittent fever, sweating and nutritive disturbances, a diagnosis is absolutely impossible, and the question of differential diagnosis does not arise. In this stage the development of rickets may only be surmised—there can be no question of a certain differentiation of the rhachitic diarrhœa from other varieties of infantile diarrhœa. But even later, after the bony changes are fully developed, rhachitis may be confounded with other morbid conditions. Primarily a *kyphosis* eventually may erroneously be looked upon as rhachitic in cases in which, from the action of a tuberculous vertebral affection, curvature of the vertebral column occurs. Apart from the fact that, besides the rhachitic kyphosis, a malformation of the thorax, the chicken breast, the rhachitic rosary, etc., are to be demonstrated, this variety of kyphosis differs from that produced by a tuberculo-carious vertebral affection in that the *curvature of the vertebral column is never, as in the former, acutely angular.* A further question, which occasionally arises, is whether the changes which have occurred in the skull are due to rickets or to *hydrocephalus.* In rickets which is healed, an unusually large skull is simulated, because the frontal and temporal bones stand out prominently, the bones of the face, on account of the deformity previously described, appear shortened, and the remaining parts of the body are but poorly developed and, on account of the deformity of the bones, appear dwarfed. An exact examination of the conditions of the skull and mensuration of the circumference of the skull, however, rapidly clear the situation. In the stage of rickets in which the fontanelles still remain open, there is greater difficulty in the differential diagnosis. The facts that especially the squamous portion of the occipital bone is thin in the rhachitic skull, and the bone borders which are adjacent to the fontanelles opening and to the sutures that have remained, appear thickened, but, above all, that the circumference of the skull, in comparison with healthy children of the same age, in spite of the apparent increase, in reality, however, is not larger, the absence of flattening of the orbits and the transparency of the skull and, finally, the normal development of the mental faculties, decide in a questionable case against hydrocephalus. It must be emphasized, however, that the latter, according to experience, occurs in combination with rhachitis. The changes *in the epiphyses in hereditarily syphilitic children*, which in an anatomical connection show a certain similarity with the rhachitic process, cannot, in their clinical appearances, be confounded with rhachitic epiphyseal alterations. It leads to a necrotic dissolution of the epiphyses, which shows itself by abnormal movability of the fragments and pain upon passive movements, and is found particularly at the lower end of the femur.

OSTEITIS DEFORMANS, PSEUDO RHACHITIS SENILIS, PAGET'S DISEASE

In 1877 Sir James Paget described a disease of the skeleton which consisted in an alteration (curvature) of the shafts of the long bones and also of those of the skull without implicating the osseous structure of the face. This condition he called osteitis deformans; since that time numerous cases have been observed in all countries. Regarding the ætiology nothing is known.

Clinical Picture.—The typical cases are well developed. The disease only occurs in patients in advanced life, for this reason it is more frequently observed in institutions for the aged than in hospitals. It is characterized by "rheumatoid" pains which occur first in the tibia and fibula, as a rule, and thence insidiously implicate the other bony structures. Decided curvature occurs in the long bones, greatly limiting their use. The knees are bent outward and can no longer be approximated, this giving rise to diminution in the height of the skeleton. Curvature of the vertebral

column is noted especially in the cervical region and in the upper thorax; the circumference of the head increases greatly, but the general health is only influenced after a very long period.

Pathology.—Malachitic and hypertrophic changes are found in the bones with resorption and decalcification. The bones which are particularly affected are those which carry the axis of the body: The skull, vertebral column, the tubular long bones of the lower extremity; less rarely those of the face, fingers and foot (Lunn). Changes have also been noted in the pelvis; malignant tumours have been frequently observed in connection with this process. The central nervous system shows no constant changes.]

INFECTIOUS DISEASES

PRELIMINARY REMARKS; GENERAL CONSIDERATIONS

Under the name of *infectious diseases* a large number of maladies are grouped, the nature of which consists in the action of peculiar toxic agents which may be differentiated from chemical poisons in that they possess the property of increasing within the morbid organism and outside of it, and, finding their way into the human body, they produce a pathological condition with a specific, typical course, which is in keeping with the action of the infectious material that is present. The effect of these infectious products can only be explained in a satisfactory manner by the assumption that it depends upon an invasion of micro-organisms. In favour of this view are, apart from the non-autochthonous origin of these infectious diseases which renders this assumption at least probable, the appearance of individual infectious diseases in endemics and epidemics, the usually long period of incubation necessary for the development of the toxic agents, but, above all, the circumstance that *a certain number of infectious diseases has been proven to depend upon specifically acting micro-organisms as the cause of their existence.*

R. Koch, to whom we owe a great deal in the latter respect, has set up certain requirements which are essential to enable us to explain that certain bacteria are the specific generators of an infectious disease, viz., *The finding of the same micro-organism in all cases of the respective disease, the limitation of its occurrence to the latter, the possibility of explaining the symptom-complex of the affection from the mode of distribution and the method of action of the species of bacteria which is supposed to be specifically pathogenic, and, finally, from inoculation of the pure culture of the bacterium into other organisms, to produce in the latter an affection similar to the original disease.* The action of the individual pathogenic bacteria by no means always conforms to all of these requirements; however, this is due partly to the undeveloped condition of our methods, but, above all, to the important fact that many of the infectious diseases of man are not transmissible to animals, and the specific action of the pathogenic bacteria for this reason cannot be furnished by animal experiments. On the other hand, the proof can be furnished for at least some few human infectious diseases that they are actually and solely due to certain pathogenic bacteria. This may be shown with exactness according to the fundamental laws laid down by Koch (especially of anthrax and tuberculosis), so that to-day the doubt of the specifico-ætiologic relation of pathogenic bacteria to the infectious diseases in general has been forever silenced.

The proof that certain well-characterized micro-organisms are present in some infectious diseases is of a decisive nature for the *diagnosis*. There is no doubt that, even without the proof of specific bacteria, the disease

may be determined with great certainty from its usually very typical morbid phenomena, and it would be wrong to assume that the exact observation of the symptoms at the bedside for purposes of diagnosis would become more and more unnecessary in the future at which period the pathogenic bacteria giving rise to the individual infectious disease can be determined with greater certainty than now. Even if the object that the specific cause for every infectious disease could be recognised bacteriologically with ease and certainty, might be attained, the function of the diagnostician would by no means be completed with this proof. The desideratum would still exist to study the specific action of these bacteria upon the human organism in general, and upon its individual organs in special, and the attempt to determine the dependence and intensity of the anatomical changes and disturbances of function as a result of their action, and the correlation of the separate factors of the morbid process, would still be necessary. Neither dare it be forgotten that the diagnosis of an infectious disease has the stamp of certainty pressed upon it only by the certain microscopic demonstration of the specific pathogenic agent! Even now, the diagnosis of pulmonary tuberculosis, without the proof of tubercle bacilli, under the most favourable circumstances is only looked upon as likely; that of anthrax, without the finding of the anthrax bacilli, which cannot be mistaken, makes the diagnosis entirely untenable.

The fact that we make the diagnosis in individual infectious diseases dependent upon the occurrence of certain bacteria, which can be definitely determined morphologically, places us in a position to differentiate diseases which in their symptomatology closely resemble one another and which, up to this time, had been considered to be identical or mistaken for one another; and, *vice versa*, diseases of apparently unknown cause may be recognised as belonging to one and the same infectious disease, even occurring under another typical pathological picture. To mention a special example in the latter respect, the pneumonias of rag sorters, and certain severe intestinal affections which ran their course under symptoms resembling cholera, were recognised to be cases of internal anthrax, i. e., being the expression of an unusual method of the development of the poison of anthrax in man.

The proof of the presence of pathogenic bacteria only then has a *practico-diagnostic* value if it is possible to recognise them without their being in a pure culture, and that the morphological, especially the tinctorial, properties of the specific organism, as is the case with the tubercle bacillus, are such that mistaking them for other bacteria is entirely excluded.

With the proof that characteristic micro-organisms are the exciting cause of certain infectious diseases, as has already been mentioned, their diagnostic value has by no means been exhausted. On the contrary, the question forces itself forward, *how the individual varieties of bacteria act in the organism*, and whether, by the manner and means in which this occurs, points may be developed which are of value in the diagnosis. Experimental bacteriology in the course of the last decade has principally concerned itself with this question, and has furnished an enormous amount of highly interesting facts. On account of the great amount of the latter and because a great deal is yet by no means clear, and opinions are still being formed, it is extremely difficult to give even a superficial review of these facts. Nevertheless, at l

main points shall here be briefly considered; this is all the more necessary as, in the study of the question regarding the action of bacteria in the organism and its relation to immunity and preventive inoculation, results have been attained, especially lately, which are of the greatest importance in the diagnosis of infectious diseases.

Action of Bacteria.—Among the bacteria which grow and multiply in the organism (the saprophytes, and their action, which do not multiply, are excluded in this consideration) we differentiate according to the manner of their growth and of their deleterious action upon the body: 1. *Bacteria which only multiply at the point at which they find entrance into the organism (and in the surrounding area)* which, however, may nevertheless severely damage the body, so that, originating from the bacterial nidus, an *intoxication* takes place by substances produced by the micro-organisms known as "toxines." Chief representatives of this category are tetanus and diphtheria bacilli. 2. *Bacteria which are widely distributed in the organism during their growth.* This occurs either by metastases, i. e., by way of the lymph and blood channels, due to bacteria being deposited in certain areas of the body which are with a certain regularity principally affected by the metastases (pyæmia, tuberculosis, enteric fever), *or by the uniform distribution of the bacteria through the entire channel system*; by this means the bacteria collect in the capillaries, increase here, grow into the veins and thus eventually reach the general circulation. These are the *septicæmias* in a general sense, to which group belong especially streptococci and pneumococci septicæmias (in the course of phlegmons, diphtheria, severe cases of pneumonia and other affections, relapsing fever, and anthrax).

The action of the bacteria and of their toxic products is partly of a *local* and partly of a *general* nature. The former, the *local* effects, are characterized by *the production of purulent inflammations and exudates*, or by the formation of specifically inflammatory proliferations developing from cellular tissue, "*the infectious granulation tumours.*" We are justified in assuming now that the reactive inflammation is due to the chemical action of the bacteria, in that, by this method, *white blood cells*, and especially the polynuclear leucocytes, which possess motility, *wander into the blood stream from the marrow of the bone*, accumulate in the areas in which the chemotactic irritation has occurred, and, as we shall see farther on, counteract the infectious irritant. The *general* bacteriological actions are those of intoxication of the organism by the poisonous products of bacteria, and this shows itself in certain more or less common resultant phenomena of the infection: Parenchymatous changes, especially degeneration of the cell protoplasm of the internal organs, fever, irritative or paralytic phenomena in the nervous system, greater decomposition of albumin and, as the result of the latter, increased excretion of nitrogen, and especially also of the excretion of hydrated albumin bodies, the albumoses.

The Nature of the Toxines.—A great many of the effects of the infections may be looked upon, finally, as the result of the products of secretion furnished by the pathogenic micro-organisms, and they are designated as "toxines." These show themselves to be very labile substances, the chemical composition of which is by no means definitely determined as yet; their peculiar action may at present only be surmised from their biological properties. Apart from the general toxic effects which have already been alluded to, the toxines possess the property of producing in the organism specific "*antitoxines*," by which they differ vastly from other chemical poisons. The property of the production of such anti-bodies belongs, besides to the bacterial, only to certain animal (snake poison), and also to some vegetable, toxines (abrin, ricin), but, besides, as latest developments have shown, also to other materials which are not poisons at all but are food products. In contrast to other, chemical substances, the toxines which reach the body do not at once show their toxic property, but only after a period of latency of the poison, *the incubation period*, has preceded it, which, even by a very considerable increase of the dose of the toxine, cannot be abolished. As in the course of the individual infections or intoxications certain cell areas of the body are electively damaged, it must be assumed that the protoplasm of certain cells shows a greater affinity for the poison and with these forms a definite chemical combination.

Ehrlich's Theory.—In what manner this occurs is only a subject of hypothesis. The most likely theory, because it can be best combined with known observations, is

the view of P. Ehrlich, which must be explained in its principal points as it promises to be of essential importance for the entire subject of bacteriological action, of the doctrines of immunity and nutrition. As a fundamental principle of the action of the toxine, *a firm chemical combination of the toxine molecule with the protoplasm molecule* of certain cell areas must be assumed. In this respect the toxines would resemble the food products "capable of assimilation," i. e., those which also combine permanently with protoplasm, whereas the action of other chemical substances in the organism, for example the alkaloids, does not depend on a firm, but on a *transitory, loose combination* of the chemical substances with the protoplasm (formation of salts, etc.). The property of the (food material and) toxines, to form a permanent combination with the protoplasm, according to Ehrlich's view, depends upon a peculiar grouping of the atoms of the toxine molecule, in that they contain a chemical group, the "*haptophorous*," by which they become firmly anchored with the corresponding haptophorous side chains of the protoplasm molecule, the "*receptors*." Presuming that such receptors for the detention of the special toxines are not present in the body, or that they are limited to certain tissues, the natural immunity of some species of animals towards some toxines as well as the specific action upon certain organs could be explained. As, further, the occupation of the receptors by the haptophorous group of toxine molecules, according to general biological laws, causes a substitute formation, even a surplus quantity, of newly formed receptors, this amount may become so considerable from the great quantity of toxines which are superadded that the superfluous receptors are thrown off and thus reach the circulation. Thus the blood contains substances, the *antitoxines*, which cause a union of the haptophorous group of toxine molecules and the formation of chemical substances which are indifferent combinations as far as the organism is concerned; the antitoxines are, therefore, able to arrest the toxines in the blood and to keep the toxines from the receptor-carrying cells and, therefore, from the cells exposed to the poison.

These assumptions, however, are insufficient satisfactorily to explain the very complicated question regarding the nature and action of the toxines. Ehrlich further postulates, besides the existence of a haptophorous group, a second group, "the *toxophorous*," in the toxine molecule, which has a toxic action upon the protoplasm of the cells only after the combination of the toxine by the haptophorous group has taken place, which, in the special instance, does not occur at the same time, which would partly explain the difference of the duration of the incubation stage. Besides the toxines, certain naturally or artificially attenuated toxine modifications have become known, "*the toxones*," and the non-poisonous modifications, "*the toxoides*." Both substances are capable of forming a combination with the specific anti-bodies, therefore they possess the same haptophorous group as the toxines, whereas the toxophorous group is more feebly developed or entirely absent. By a plentiful administration of the latter, we are able, in keeping with the presence of the haptophorous group in their atomic arrangement, to produce, by means of the toxones or toxoides, antitoxines, with this advantage that by this means benign substances are utilized for immunization purposes.

Preventive Measures against Infection—Immunity and Immunization.—The intensity of the action of the bacteria and their toxic products upon the infected organism depends upon the virulence and quantity of the bacteria which are active, and, above all, upon the reaction of the infected organism and in how far it is capable of protecting itself from the pernicious action of the bacteria. In this regard the body has control of a number of protective expedients, the knowledge of which is of great importance to the physician, as their observations may open up entirely new roads both regarding the diagnosis and treatment. We must, therefore, concern ourselves somewhat minutely with this condition.

The intact normal human organism shows a certain resistance to *the entrance of micro-organisms into the body*, in that the skin and mucous membranes form quite an opposing wall to their invasion, so that, as a rule, a small loss of substance is necessary to form a flaw for the entrance of the bacteria. The secretions of the mucous membranes, the saliva, mucus, gastric juice, etc., retard the development of bacteria. A certain obstacle, more or less difficult to overcome, is also formed by the

lymph glands, as the bacteria in their migration encounter a retention here which is of a permanent or temporary nature. The surface of certain organs, on the other hand, is less resistant, such as that of the tonsils, the bowel and the alveoli of the lung.

Of more importance than the above-mentioned guards are those which are contained in the body to prevent infection, *natural immunity*, and also after infection has taken place, the operation of *certain factors which act as a defence*.

Natural Resistance

I. Regarding *natural immunity* or, a better term, "*natural resistance*," it is generally acknowledged that some individuals are less susceptible, i. e., are more able to resist, than others. The reason of this greater resistance may be looked for in the fact that such organisms are capable of preventing the action of pathogenic bacteria by a more developed condition of the natural protective arrangements. These, primarily, are furnished by a more energetic activity of the cells. As we know that the cells of the body, especially the leucocytes, have a destructive action upon bacteria, and, on the other hand, it has been determined by Buchner, and others, that also the fluids of the body that do not contain cells, especially the serum, display the same bactericidal property, we may assume that the normal blood contains chemical bactericidal substances which are furnished by the cells and which Buchner has characterized and designated "*alexines*."

Alexines.—These are ferment-like bodies which are distinguished by their labile character, easily destroyed by heat (55° C.–60° C.) and, when opposed to bacteria, develop a fermentative-soluble action. It may be assumed that, as the result of the entrance of bacteria into the body, the leucocytes approach the bacteria by chemotaxis, developing alexines of an increased strength, taking up the bacteria, which are destroyed or have at least been injured in their powers, and render them innocuous ("*phagocytosis*" of Metschnikoff in a modified sense). The nature of the action of the alexines, as far as I am able to judge, should be conceived, according to the latest investigations and according to Ehrlich's theory, in that the alexines, constituted similarly to the toxines, i. e., with a haptophorous and a toxophorous group, by means of the former are enabled to unite with the bacteria receptors, and by the toxophorous group are capable of disintegrating the bacteria. Probably this simple method of influencing bacteria is sufficient in some infections—solely a production of alexines increased by the infection, and with this of the natural resistance—to cause a cure at once, therefore without the production of specific anti-bodies. This, however, is but rarely the case; as a rule, these simple recuperative factors are *not* sufficient to counteract the infection. In fact, the development of *specific protective substances* is usually necessary after infection has taken place, which render the organism capable, not only to overcome the beginning infective process in the body, but also to confer *immunity*, which may be either transitory or permanent ("*acquired immunity*"), to a new infection of a similar nature.

II. **Acquired Immunity.**—This may be *natural* by recovery from the special infectious disease, or *artificial*, by inoculation of infective material according to a preconceived regular plan. The protective material developed in this manner acts partly against the poisons produced by the bacteria, and partly their action is opposed also to the living bacteria themselves, hence we must differentiate between a *toxine immunity* and a *bacterial immunity*.

1. **Toxine Immunity.**—The protective substance in this case consists of the *antitoxines* which have already been described, i. e., the haptophorous group of receptors which are produced in superfluous amount and which are thrown into the blood current. Of this variety of antitoxines several have been definitely determined, such as the diphtheria and the tetanus antitoxines. The antitoxines are contained in the blood of individuals which have recovered from the infection, and, to a greater degree, in the blood of animals that have been treated by diphtheria or by tetanus toxines in such a manner that constantly increased doses of the toxine have been administered to them, thus artificially developing larger quantities of antitoxine. This antitoxine combines and neutralizes the toxines not only in the living body, but

also in the test tube, where it may be noted that, upon the mixture of a serum containing antitoxines with a surely fatal dose of toxine, the action of the latter is neutralized. In general the antitoxines act *specifically*, i. e., tetanus antitoxine acts only upon tetanus toxine, etc., the bacteria themselves remain uninfluenced by the antitoxines. To Behring belongs the credit of having recognised the origin and action of the specific antitoxines and to have utilized them for the specific immunization and the cure of the special affection.

2. **Bacterial Immunity.**—By this we understand the acquired property of the organism to utilize the protective substances against the deleterious effects of the living pathogenic bacteria. As such we must consider, in the discussion of bacterial immunity, *bacteriolysines* and *agglutinines*.

(a) **Bacteriolysines.**—Regarding *bacteriolysines*, it should be mentioned that bacteriolytic protective substances are found, for example, in the blood of persons who have recovered from an attack of cholera or enteric fever, or in the blood of animals which have been artificially treated with cholera or enteric-fever toxine. The action of bacteriolysines, similar to the antitoxines, is *specific*, so that the immunized serum of enteric fever only has a bactericidal effect upon typhoid bacilli, etc. In what manner this occurs may be controlled step by step according to the process of R. Pfeiffer, the discoverer of bacteriolysines, if, for example, immunized cholera serum and living cholera vibriones are injected into the peritoneal cavity of a guinea-pig, and the changes which occur in the bacteria in the exudate taken from the peritoneal cavity are noted from time to time by the microscope. It will be seen very readily that the bacteria soon become immobile, swell and degenerate and, finally, within about half an hour, are completely dissolved; the animal remaining alive in spite of the fatal dose. The method by which the bacteriolysines act is difficult to understand. In the light of Ehrlich's theory, the process may be assumed to be as follows:

As the immunized serum containing bacteriolysines, upon heating to 55°–60° C., becomes inactive, but, on the other hand, upon the addition of small quantities of normal serum regains its activity, it may be assumed that the action of the bacteriolysines is composed of two agencies, namely, of a thermolabile substance and that of the actual immune body. Similar to the toxine immunization (see above), i. e., in the formation of the antitoxine, it may also depend upon an excessive production and desquamation of protoplasm receptors, which circulate in the blood as immune bodies, in the immunization with bacteria. These, however, would differ materially from the antitoxines, according to the convincing statements of Ehrlich, in that they are supplied with two haptophorous groups (representing one "amboceptor") of which one would chain the corresponding haptophorous group of the bacterial protoplasm and the other combine with the thermolabile alexine circulating in the blood. Only the latter would be capable of rendering the bacteria inactive by its toxophorous group (existing side by side with the haptophorus group). The alexine, therefore, complements the action of the immune body, and from this point of view may be looked upon as a "complement" (Ehrlich) and be so designated. If immunized serum is heated to 55° C.–60° C., the complement is destroyed, whereas the thermostabile immune body itself remains intact, but is now inactive, as the complement which has the bactericidal property is absent, and only by the addition of a normal serum containing a complement can the immunized serum again become reactive.

(b) **Agglutinines.**—Besides the above-described bacteriolysines in bacterial immunization another variety of protective substances is found in the blood serum, called the *agglutinines* (Gruber). Their action consists in the fact that typhoid or cholera bacilli, brought in contact with typhoid or cholera serum, causes the bacteria to become immobile, to clump and sink to the bottom, whereupon the fluid which was previously cloudy, due to the bacteria, clears again. In general the agglutinines act strictly *specifically*, i. e., typhoid serum only agglutinating typhoid bacilli, cholera serum only cholera vibriones. It is evident that this has given us a very important aid in *differential diagnosis*, and, in fact, the agglutination of typhoid bacilli (which may be easily noted under the microscope) by the blood of enteric-fever patients, the "Gruber-Widal reaction," has already found its way into practice

and is looked upon as a diagnostic aid of importance. The action of the agglutinines is in no direct or indirect relation with the action of the bacteriolysines; their constitution, according to Ehrlich, is also different. In contrast to the bacteriolysines, the agglutinines are said to belong to the class of receptors which are developed by immunization of the blood, which have but *one* haptophorous group, but, besides, also a toxophorous group, by means of which they are enabled to produce fermentative changes in the bacterium which has been chained by the haptophorus group, i. e., without the aid of a complement.

It is obvious from what has been said that the serum itself is not the source of the production of these protective substances; they are rather the product of the cell activity of the organism, which, reacting specifically to the toxines of the bacteria, produce protective substances that bind the latter and destroy the bacteria, and cause immunization. It appears to me further, to explain the occurrence of immunity, that the assumption is necessary or, at least, the most natural, that, after the change of cell activity (differing from the normal) has begun, which is caused in the course of an infectious disease to combat the same, these substances are continuously produced by the cells either transitorily or permanently—in keeping with their *tendency of perseverance* which has been predicated by me, and which is peculiar to cell activity in general, as a result of which the cells continue to perform their labour, either, as under normal conditions or in the case of immunity, to the profit, or, as in the case of certain diseases of metabolism, diabetes, etc., to the detriment, of the organism.

The effect of the bacterial activity, which causes disease in the human body, does not manifest itself only in those cases in which the patient succumbs, but also in those instances in which the subject remains victor in the battle with the exciter of infection. Then the most varied anatomical and functional disturbances are noted in the body, which are either more or less common to all infectious diseases, or an expression of the specific activity of an individual infective substance. The former determine the assumption of an infectious disease in general, the latter the special diagnosis, to the consideration of which we shall now proceed.

DIAGNOSIS OF INFECTIOUS DISEASES

Certain infectious diseases show an apparent clinical relationship among each other, in so far as the same organs are especially affected. So in the "acute exanthemata" (measles, scarlatina, variola, etc.) an eruption of the skin occurs constantly and in a typical manner, which is the most obvious symptom of the disease and compared to which the other symptoms of the morbid process in a clinical respect appear secondary. Further, whooping-cough and influenza form a group in which the infection shows itself primarily by an affection of the respiratory organs, etc. A grouping of the infectious diseases from this standpoint, however, is impossible and would not even be correct, because, as a result of the infection, the entire body is diseased, and specially localized actions of the poisons in the individual infectious diseases and cases are not absolutely constant, and vary continuously. Nevertheless, these more or less specific localizations which occur in the individual infectious diseases, at least as a rule, are of great importance to the examining physician. Their presence gives the diagnosis the first direction, as a rule, and draws narrower limits for the differential diagnosis. Therefore, we shall in general conform to this practical division principle in the following chapter, but we shall not lay special stress upon strictly carrying it out.

MEASLES—MORBILLI—RUBEOLA

Ætiology—Incubation.—Valuable conclusions may rarely be drawn from the duration of incubation as to the diagnosis of measles in the individual case. From a great mass of absolutely reliable observations it is determined that the average duration of *incubation in measles is ten days*; exceptions to this rule are rare, and in the individual case, if the source of the infection is known, the duration of incubation may be utilized in the diagnosis. Also of value for the diagnosis are: The marked contagiousness and transitory character of the morbilli poison, the fact that the tears, the secretion from the nose, the sputum, the serum of measles patients, as the results of inoculation have proved beyond doubt, are contagious in the *prodromal and eruptive stages*, whereas, in the stage of desquamation, the epidermis scales most likely are no longer capable of giving rise to the disease in the healthy by transference. The contagious principle, as is well known, is contained in the air surrounding the patients, and may be carried by fomites or a healthy third person, although not as frequently as in scarlet fever, the poison of which is less transitory and therefore the more closely adheres to clothes, etc. The proof that the individual in whom the existence of measles is questionable, has already had measles, as a rule, is against the presence of the affection, as a second affection of measles belongs to the rarest exceptions (relapses which occur in a brief period after the apparently complete effervescence of measles must not be considered); it is also against assumption of measles that the affected person has notoriously had no communication with measles patients. The simultaneous existence of another infectious disease is no proof that the exanthem in question does not owe its origin to measles. For enteric fever, scarlatina, variola, etc., but, above all, erysipelas and whooping-cough, have been noted in combination with measles. All the points mentioned, however, must only eventually be used in a subordinate manner as a prop for the diagnosis of measles.

Prodromal Stage.—After the period of incubation has run its course, and this without symptoms, the *prodromal stage*, with well-developed phenomena or symptoms, follows, which, almost without exception, lasts three (in maximo five) days. This sets in with chilliness or with one decided chill, which is followed by fever that rapidly, i. e., on the first day, reaches a height of 102.5° F. to 104° F. On the *morning of the second day the temperature falls to normal and remains normal for the following two days, or at least is but slightly subfebrile.* Towards the end of the prodromal stage it rises again, subsequently, as we shall see later on, to reach its acme. Simultaneously with the prodromal fever the characteristic phenomena of measles appear, *inflammations of the mucous membrane*, especially catarrhal affections of the mucous membranes of the respiratory tracts and its adnexa. Conjunctivæ, nose, larynx, and bronchial tubes are inflamed; the conjunctiva palpebrarum et bulbi appears reddened, swollen (occasionally with marked chemosis), tears run from the eyes, and the eyes are painful; reflex spasm of the lids and photophobia set in. As a result of the inflammation of the nasal mucous membrane there is marked secretion; frontal headache and frequently uninterrupted sneezing occur; the face is bloated. With this there is cough which occasionally is croupy or appears in paroxysms similar to whooping-cough; the voice becomes hoarse. More rarely is there difficulty of deglutition, with slight enlargement of the tonsils and reddening of the pharyngeal mucous membrane, initial vomiting and diarrhœa, in children convulsions. The inflammation of the mucous membrane of the pharynx and palate shows itself in the

form of diffuse reddening in the course of which small, lentil-sized, dark spots are prominent, the swollen follicles are noted as nodules, the size of millet seeds; similar red areas are noted in the prodromal stage, occasionally, upon the mucous membrane of the larynx. Very early, generally upon the first or second day of the prodromal period, we note upon the lips and the mucous membrane of the cheeks small, bluish-white spots with a narrow, red ring (Koplik's *spots*). They are a very constant phenomenon in the prodromal stage of measles, hence they are of value in determining the diagnosis early. The tongue, in contrast to the tongue in

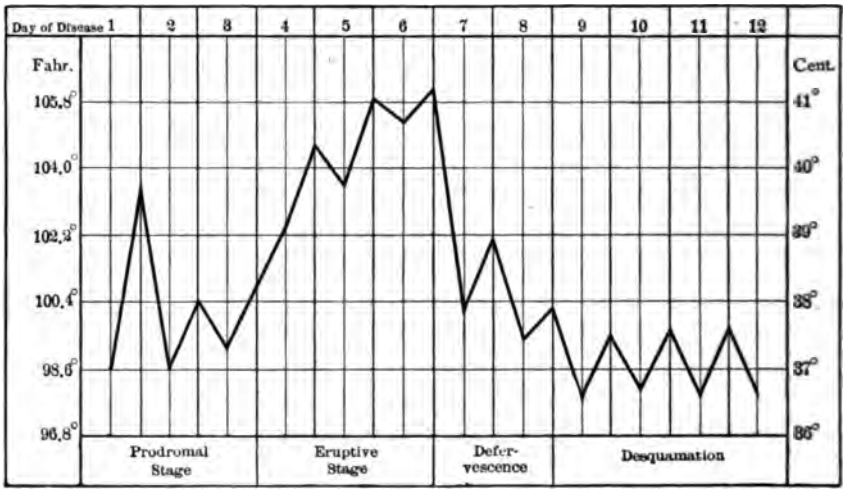


FIG. 73.—AVERAGE TEMPERATURE CURVE IN MEASLES.

scarlet fever, in measles does not show characteristic changes: It is coated, and somewhat turgescent papilli are noted. In this stage the skin generally is still free from any eruption; in some cases, on the contrary, especially in the second half of the prodromal period, small, punctiform nodules appear in the face and at other parts of the body as the commencement of the eruption. The small papules are enlargements at the mouth of sebaceous follicles. In some cases the general health is not affected at all, in other cases symptoms may be observed on the first day.

Stage of Eruption.—The appearance of the *stage of eruption* on the fourth day (from the beginning of the prodromes) is shown by a sudden rise of temperature, which, since the second day of the prodromal period, has been quite, or nearly, normal. The *fever* now rapidly reaches 104° F., on the following day may even rise higher, to 105.5° F., and with this attains its maximum; it falls at the end of the fifth (or sixth) day. With the maximum rise of temperature, at least as a rule, the most marked development of the *exanthem* coincides. The development of the eruption occurs in the beginning or the second half of the fourth day of the disease, so that *at first the face,* which is rapidly followed by the neck and back, later the body and extremities, is uniformly covered with the eruption of

measles; the development of the eruption does not require more than from twelve to thirty-six hours.

The Exanthem of Measles.—The *exanthem* which is so necessary for the diagnosis shows rose-red, more rarely dark-red, smaller or larger (averaging one half cm.) somewhat raised macules, which are sharply demarcated from the surroundings and separated from one another. They are but rarely confluent so that, with a very plentiful eruption, some areas in the skin may appear to be uniformly red. But even then the origin of the individual macules may be noted, and, besides the confluent areas, other points may be observed which show the usual exanthem. Especially characteristic, although not as regularly demonstrable, is the occurrence of minute papules which lie in the centre of the macule and are somewhat raised over the latter. Upon pressure of the finger the macules disappear for a moment unless a capillary hæmorrhage has occurred and the case is one of so-called *hæmorrhagic* measles. Besides the rare form of hæmorrhagic measles, which occurs in debilitated individuals and very small children, with hæmorrhages of the mucous membranes and internal hæmorrhages, and which then runs a severe course, we may differentiate *morbilli læves,* i. e., distinctly isolated, smooth morbilli macules without distinct enlargement of the follicles; further, *papular* measles if the enlargement of the follicles is marked, and *vesicular* measles (*miliares*) if the latter is caused by a fluid exudate. The diagnosis of these varieties of the eruption of measles is not difficult, but it is without clinical value.

The exanthem and the inflammations of the mucous membrane, besides the fever, show the characteristic symptoms of measles. Their *ensemble* entitles the positive diagnosis of morbilli; but the synchronous development of these three phenomena is not absolutely necessary. There are positively demonstrated *cases of measles without affection of the mucous membrane,* which, as we shall see, may be easily confounded with rötheln [rubella], and, *vice versa,* we are compelled to admit a *variety of measles without eruption,* if, during an epidemic of measles, affections of the mucous membrane and fever run the same course in apparently healthy individuals as in measles, but the exanthem does not occur. The diagnosis in such cases, however, is only a probable one; it is somewhat more certain if, as has been noted, in cases of *" morbilli sine exanthemate,"* later a characteristic furfuraceous desquamation occurs, or if enlargement of the spleen can be proven. The *fever* in mild cases may be very slight, *it is, however, never absent.*

Defervescence—Desquamation.—After the fever and the exanthem have reached their maximum on the sixth day of the disease, the temperature falls more or less by crisis, so that *on the seventh or eighth day* it is normal again.

During this time the exanthem also begins to fade, at first upon those places of the body which were primarily attacked, therefore, in the face, later in the extremities; the pale macules still retain a yellowish pigmentation for some time. The affection of the mucous membrane also gradually ceases with the disappearance of the exanthem. With the fall of the fever, but generally some days later, the *stage of desquamation* begins:

The skin commences to separate from those areas in which the eruption was formerly situated, in the form of small furfuraceous scales (*d. furfuracea*); rarely are the scales larger, but they never show such great lamella as in scarlatina. The desquamation is most marked and most constant in the face, less regular and less marked on other portions of the body; it usually lasts several days, rarely a week and longer. The urine in measles shows no special changes; occasionally it contains albumin, as in all infectious diseases. The marked symptoms of nephritis, however, are quite rare in measles.

Complications.—The above-described, typical course of measles is modified partly by certain anomalies of its course, partly by complications, the knowledge of which is important to the diagnostician. ***The fever may be unusually high and protracted.*** Even in the prodromal stage the height of the temperature, not alone on the first but also upon the second and third days, may be very great; in other cases the prodromal stage may show itself markedly prolonged, with high fever until, finally, an unquestioned eruption of measles clears the situation. Unusually high-fever ranges occur even in the eruptive stage, especially if *complications* arise during this period; but the appearance of the latter may also manifest itself by fever in the usually afebrile period of desquamation and during convalescence. Marked deviations from the usual fever course undoubtedly call for an exact examination of the internal organs in order to determine the complications which run their course with a rise in temperature. Complications are many in measles, and of the most important a few shall be considered.

Occasionally the diagnosis is made difficult in that, simultaneously with the eruption of measles or before this appears, other exanthemata appear upon the skin. In all stages of measles, from the prodromal period to convalescence, erythemata are noted to occur which resemble the eruption of scarlet fever. Further, the eruption of the macules of measles may be accompanied with the development of herpes, pemphigus, urticaria, or the formation of pustules; relapses of the eruptions of measles have also been noted occasionally, so that, after the eruption of measles has occurred according to rule and is in the period of disappearance, another exanthem of measles with a new development of fever arises. Of *complications on the part of the mucous membranes* may be mentioned: The exaggeration of the inflammation of the conjunctiva, of the mucous membrane of the nose and pharynx into gangrene, the spread of the inflammation to the cornea, iris, and Eustachian tubes (with subsequent otitis media), further, ulcerations in the larynx and pseudo-croup. Actual *diphtheria* may complicate the morbillous affections of the pharynx and larynx. Whether this group of measles is of a diphtheritic or of a non-diphtheritic nature—both varieties occur—is only determined by the examination of the pseudo-membranes for the Klebs-Löffler diphtheria bacilli. Especially important are the complications on the part of the *bronchial tubes*. In that the inflammation attacks the finest bronchi, the development of *catarrhal pneumonia* is very apt to occur, which is the most frequent and most dangerous complication or sequela of measles. This develops in the stage of eruption or

in the period of desquamation and shows itself by the plentiful loud râles and the almost always present bilateral dulness in the lower posterior portions of the lungs, as well as by a rise of the temperature, by cyanosis, etc. The patient with measles is to be examined daily for this complication, as the early knowledge of the onset of catarrhal pneumonia very frequently determines a life-saving therapy. Rarer complications, besides those already mentioned, are eclamptic attacks, sinus thrombosis, apoplexy, meningitis, endocarditis and pericarditis. Somewhat more frequent is *pleurisy*, the original anatomical substratum of which is perhaps the macular reddening of the pleura, often observed in cases which have come to autopsy; *intestinal catarrh* has also been noted as a frequent complication. It is very probable that the mucous membrane of the intestine regularly takes part in the inflammation of measles, so that, even in the prodromal period, diarrhœa is frequent. It is rare for the catarrh of the intestine to develop more marked dimensions and, in that the dejecta become muco-hæmorrhagic, to show a severe character; if this occurs the affection of the bowels adds greatly to the lethal outcome of the case. *Gastric catarrh* is a rare complication, as is also *arthritis* and *nephritis*, in contrast to scarlet fever, the excretion of the poison of the latter through the kidneys giving rise to more marked irritation and very generally developing an inflammatory condition. Quite rare, in general, in the course of measles, are the development of parenchymatous swelling of the liver and marked enlargement of the spleen, whereas enlargement of the lymph glands is quite common. In rare cases, finally, *septicopyæmia* appears as a complication.

Sequelæ.—As *sequelæ* remain, on the whole but rarely: Valvular defects of the heart, prolonged diarrhœa, chronic arthritis, affections of the ear, especially frequently diseases of the middle ear; further, periostitis of the orbits, neuritis, hemiplegia, etc. More important among the sequelæ in measles are *chronic pulmonary affections* which have been frequently noted as a direct result of morbilli. Here are to be considered pleural exudates which become purulent, chronic pneumonia, and tuberculosis, the latter, as it appears, finding a favourable soil to develop in the pulmonary tissue affected by measles. The same is true of the infection by *whooping-cough*, which is not rarely seen to occur immediately after measles.

Differential Diagnosis.—In by far the majority of cases the diagnosis of measles, with the observation of the clinical phenomena just mentioned, is not difficult. In some few cases, as I must admit from my own experience, the question whether measles or another acute affection is present, is very difficult to decide, yes, in some cases only a provisional diagnosis is possible.

Drug Eruptions.—As the eruption of measles represents a form of roseola, *exanthemata of roseola* of various kinds may be confused with measles, especially if the patient is seen for the first time during the eruptive stage of the disease. It is not so easy as may be believed to differentiate from measles certain eruptions caused by drugs, namely, roseola eruptions after the use of iodine, copaiba, antipyrine, etc. Grave errors sometimes occur especially if, owing to an acute febrile, not yet diagnosticable affection, which may be eventually accompanied with coryza, an antipy-

retic was administered at the onset, and now, in consequence of the action of the drug, an eruption occurs which is absolutely the same as that of measles, and thus a diagnosis which so far had been doubtful appears to become clear. In such cases we must note, above all, whether the fever is marked especially upon the appearance of the eruption after the action of the drug has ceased, whether, besides the exanthem, the ordinary inflammations of the mucous membranes occur which are common to measles, and whether the eruption shows the so important small central papules and the typical course which is characteristic of the exanthem of measles. But, in spite of all this, for a while the diagnosis may be impossible if fever, cough and other symptoms due to another cause are present; but, even if the affections of the mucous membranes are absent, it must not be forgotten that these are occasionally absent also in morbilli (in those varieties designated as measles occurring without inflammations of the mucous membranes). The eruption due to drugs, although it becomes paler relatively quickly after the remedy has been discontinued, similar to the condition in measles, is not so typical as the exanthem of measles, and, on the other hand, becomes more prominent when the drug is again administered.

Enteric Fever and Typhus Fever.—*Roseolar eruptions* due to other causes than drugs rarely give rise to mistakes: The roseola of enteric fever, the eruption of typhus fever, the syphilitic roseola, and, finally, the roseola which now and then is seen and is due to anomalies of menstruation or to unknown causes, especially in children. The last-named varieties of roseola are easy to differentiate from measles, as they occur without fever and certainly without any affection of the mucous membrane. The *roseola of syphilis* is more apt to be confounded with measles, if it runs its course with marked fever (as is occasionally the case). But the implication of the mucous membranes of the respiratory tract: Cough, sneezing, etc., is absent, although angina may occur. Above all, however, the usually dark-red eruption does not disappear rapidly, as is the case in measles, and the consideration of the ætiology, and the presence or absence of other symptoms depending upon a recently acquired syphilis, direct the diagnosis into the right channel. The eruption in *enteric fever* is rarely so markedly developed that it may be confounded with measles; it is more important to remember that the roseola of enteric fever occurs only in the middle of the second week, the fever, therefore, in the case in question has existed for more than three days. It is true that the eruption in the abortive types appears earlier, but we are protected in these forms, as in the common forms, of enteric fever by the relative slowing of the pulse, the extent of the enlargement of the spleen, and the absence of mucous-membrane affections, especially of the specific injection of the palate, and so may make a differential diagnosis between enteric fever and measles. The confusion is more likely to occur with *typhus fever*, as the eruption in both diseases may run a similar course and occur from the third to the fifth day, and, further, conjunctivitis, coryza and bronchitis may appear in both diseases. However, in typhus fever the prodromal fall in temperature does not occur, and the fever does not fall on the seventh day, but later, usually

not until the ninth to the twelfth day, but here, similar to measles, by crisis in the course of two days. In typhus fever the development of the eruption in the face compared with the trunk and the extremities is not at all marked; further, the change of the eruption from a roseolar form into petechiæ, which is the rule in typhus fever, is very exceptional in measles, and just so is the enlargement of the spleen marked, easily demonstrable, in exanthematous typhus, in contrast to measles. The enlargement of the spleen in typhus fever occurs in three quarters of the cases.

Variola.—A confusion of measles with variola at first sight appears to be impossible. Nevertheless, both diseases have some points in common in their development. In variola as in measles the appearance of the eruption occurs upon the fourth day; in both prodromal erythemata occur, and, besides, the eruption of small-pox in its onset is constantly morbilliform, i. e., it occurs in the form of small, red, slightly raised macules, so that in this stage of the two diseases a wrong diagnosis in either direction is possible. But this is prevented by the observation of the prodromal fever, which, in the case of small-pox, rises continuously from the first to the third day, to *drop to normal* with the appearance of the eruption, i. e., under all circumstances, therefore, *an entirely opposite condition to the fever of morbilli.* The other phenomena of the prodromal period are also different in both affections; in small-pox gastric symptoms and pains in the small of the back, which do not occur in the prodromal period of measles are prominent, whereas the affections of the mucous membranes: Angina, coryza and others, are rare and subordinate, if they occur at all, in small-pox. With the development of the vesicles of small-pox and pustules, therefore from the sixth day on, all doubts must vanish.

Scarlatina.—More frequently than to decide between small-pox and measles are we called upon to make a differential diagnosis between *scarlatina* and measles. As there are eruptions of scarlatina which do not, as is usual, become diffuse but occur in the form of discrete roseola or of papules, and, *vice versa*, also confluent measles occur, it is easily understood that the differentiation between the exanthem of measles and scarlet fever may, even to the expert, give rise to great, yes, insurmountable, difficulties. In the differential diagnosis in such cases it should be noted that the exanthem of measles occurs first in the face and is here especially markedly developed, whereas the neck and chest are first affected in scarlatina, and the face, under all circumstances, shows less eruption, especially the region about the mouth is particularly free. Further points for the diagnosis are given by the period of eruption of the exanthem, which, in the case of measles, is on the fourth day, in the case of scarlet fever as early as the first or, latest, upon the second day; further, the peculiarity of the prodromes: The initial vomiting and the severe angina in scarlet fever, the inflammation of the mucous membranes of the respiratory tract in measles, the fever which, in scarlatina, does not show the prodromal defervescence characteristic of measles, and even later is different in both affections. Finally, the tongue also assists in making the differential diagnosis: In the case of measles it has no characteristic appearance, whereas, in scarlatina, after a few days the tongue shows the characteristic and well

known "raspberry" appearance. A complete differentio-diagnostic conception between measles and scarlet fever can only be given after we have carefully considered the diagnosis of scarlet fever.

Rotheln.—The exanthem of *rötheln* is so like that of measles that a differentiation of both diseases from the appearance of the eruption is frequently impossible. It is true, the macules of rötheln are smaller, less serrated and are not as red as the macules of measles, but these differences are not prominent enough to allow us to determine between the two eruptions. The first appearance of the exanthem in the face, and the *ensemble* of the prodromal phenomena, viz., cough, hoarseness, sneezing, photophobia, etc., is the same in both affections when they are mild. But the duration of the prodromes in rötheln is much briefer, rarely lasting over a day, the general health is scarcely affected, and *the temperature is but slightly, if at all, raised.* As in all affections which are to be considered in the differential diagnosis, in doubtful rötheln the fact that the patient had a former attack of measles, in which case almost always an immunity is acquired, points against a new infection by measles. Also the fact that an epidemic of one or the other infectious disease is present at the time, may be used in the diagnosis.

Influenza.—This is especially the case in an epidemic of *influenza,* which affection appears with the phenomena of the prodromal stage of measles. Only the appearance of the eruption will allow us to make the differential diagnosis in this instance; in the first three days the small papular reddening of the pharyngeal mucous membrane may occasionally be of value and point to the diagnosis of measles against that of influenza.

How different would the case stand in those frequent cases in which other affections so closely simulate measles, if it were possible to find the micro-organism which is specific of this affection! But, as it appears, up till now this micro-organism has not been *definitely* determined.

SCARLET FEVER, SCARLATINA

Ætiology.—Scarlet fever is a contagious, infectious disease (from person to person) the poison of which reproduces itself in the body affected by scarlatina. The tenacity of the scarlet-fever poison is acknowledged to be very great; it adheres to clothes, undergarments, to all objects in the surroundings of the patient and is said to be found also in the secretions, especially in the mucus of the mouth and pharynx and, as is well known, in the exhalations of scarlatina patients. The contagion occurs by contact with these effects or by the presence in the room in which a scarlet-fever patient has been, or in which he is still present, or, finally, through a third person, who himself may remain unaffected. If the poison has found access to the body, it circulates in the blood, which, if transferred, produces scarlatina. I was affected by scarlet fever after I had injured my finger at the autopsy of a scarlet-fever subject. The eruption of the exanthem curiously took its course from the point of inoculation, which may probably be explained in that the poison during the incubation period ripened at the point of infection before it was distributed to the rest of the body. Contagion may occur at any stage of the disease, *most easily, as it appears, in the period of incubation,* but the possibility of transference during the period of desquamation, which formerly was supposed to be the most dangerous time and by many physicians to-day is regarded as the most favourable period for contagion, cannot well be disputed.

Incubation.—The *period of incubation* varies; as a rule, it is from four to

seven days, therefore, *on an average one half the time of the period of incubation in measles.* This may be adhered to in the diagnosis, in deciding whether scarlatina is due to this or that source, but it must not be forgotten that we are dealing with an *average number,* that many exceptions occur in which a shorter or longer period of incubation than five days must be accepted (half a day to three weeks). In my opinion, these exceptions to the rule are due principally to *individual susceptibility to the scarlatina infection.* The above-quoted example, concerning my own person, is very instructive in this respect. I must have had a very slight predisposition to scarlatina, was neither affected as a child by scarlatina when my own relatives were affected, nor even later when treating scarlet-fever patients. One day I wounded the index finger of my left hand in an autopsy of a scarlet-fever subject; the post mortem was held upon a person who had gone through an unusually severe course of scarlet fever. On the seventh day after the lesion, the badly healed wound began to pain; only at the beginning of the tenth day malaise and angina occurred; on the eleventh day vomiting and marked fever, and towards the end of the eleventh day a scarlet-fever eruption that, contrary to the usual course, first showed itself at the point of inoculation, following the lymph channels of the left arm upward in the form of a broad red band, then rapidly diffused over the rest of the body. The course of the scarlatina was a medium severe one; desquamation also began on the left arm. Although this case was a pretty clear evidence of inoculated scarlatina, I do not think it right, upon the basis of this experience, to accept the twelve to fourteen days' period of incubation which is named by some authors as the average period of incubation. This is contradicted by evidence just as conclusive from the greatest majority of physicians that the period of incubation is not longer, but, as a rule, shorter, than a week. From the example above quoted, according to my opinion, only this much may be assumed with a certain degree of likelihood, that a person who is not susceptible in the ordinary form to scarlet-fever infection cannot withstand the directly inoculated scarlatinal poison, but, nevertheless, shows his individual, greater power of resistance in that it requires a longer time for the poison to produce the symptoms of the disease, and that, possibly for the same reason, the poison of a very severe case of scarlet fever, even after the direct inoculation, is only capable of producing a relatively mild grade of scarlet fever. I must insist upon this, as from other sources observations have been reported which show particularly a very short period of incubation, one to four days, in cases in which the scarlet-fever infection occurred in persons with open wounds.

Relapses and even two, yes, four attacks of scarlet fever occur; they are, however, as in the case of measles, under all circumstances rare; in the greatest majority of cases one attack of scarlatina confers immunity for the rest of life. The briefly sketched ætiological conditions must be taken into consideration in the diagnosis of scarlet fever. Decisive for the diagnosis, however, is only the observation of the entire symptom-complex of the disease, in which the exanthem is the most important, if not the only, factor from which the diagnosis may be made.

Prodromal Stage.—The period of incubation, as already remarked, runs its course in from four to seven days, without symptoms or, at most, with mild malaise, and now the *prodromal stage* follows. This is ushered in by rapidly rising *fever,* usually accompanied with a chill, with symptoms of cerebral irritation, decided headache, fainting sensations, convulsions (in children), and vomiting. The last-named symptom is diagnostically important as an initial symptom of scarlet fever, as it occurs in no other infectious disease (pneumonia of children excepted) so frequently as in scarlatina. The most characteristic symptom, which is usually first noted by the patient, is *angina,* the objective substratum of which is shown by a reddening of the pharynx, of the tonsils, and of the uvula, which is combined with an enlargement of the lymph glands behind the angle of the jaw.

Course of the Fever.—The *fever* in the prodromal stage is not characteristic. As a rule, it is high from the first day of the disease, between 103° F. and 104° F., rising upon the second day with the appearance of the eruption to 105° F. or over. It remains at this height, with slight remissions, for several days, to decline gradually, i. e., less critically than in the case of measles. There are many deviations from this the usual course of fever in all stages of scarlatina: Scarcely rising above the normal, yes, in exceptional cases even being afebrile in the prodromal period, marked remissions on the second day, *critical* defervescence towards the end of the

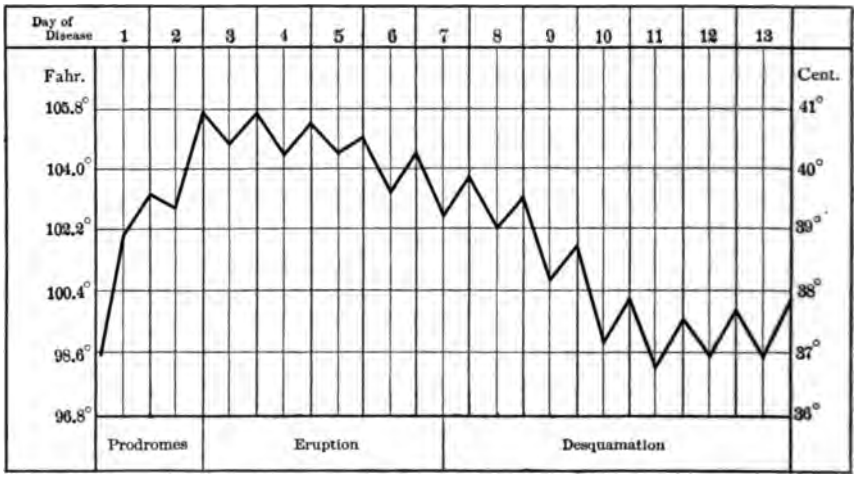


FIG. 74.—AVERAGE TEMPERATURE CURVE IN SCARLET FEVER.

first week, etc. Nevertheless, it is well in the diagnosis to think of the average fever-curve of scarlatina, and carefully to observe deviation, i. e., only then to be satisfied when the most careful examination of the patient reveals no reason for the abnormal behaviour of the fever in the given case.

Stage of Eruption—Eruption.—The same as the duration of the period of incubation in scarlatina is about half that of measles, this also holds good of the prodromal stage. This, as a rule, is not longer than from one to two days; the beginning exanthem is often even noticed in a primary condition upon the first day of the disease. The *eruption of scarlet fever* can, however, certainly be seen upon the second day. It appears with the eruption of numerous macules, varying in size from that of the head of a pin to that of a lentil. They are intensely red, first appearing upon the neck and chest and are rapidly distributed over the entire body, *with the exception of certain parts of the face, especially the regions of the mouth and chin,* which are conspicuous by their pallor as opposed to the rest of the face. In the extremities the regions of the joints and especially the extensor surfaces are more markedly affected than the rest. These individual red points are very close together, so that at least one half of the surface of the skin appears to be implicated. Confluence rapidly occurs through growth of the individual points: The skin now appears uniformly *diffusely*

red, so that upon careful examination the entire skin appears to be suffused with a scarlet-red appearance; however, dependent upon the amount of the eruption, small, pale or more darkly punctated areas may be noted upon close inspection; the skin appears to be slightly infiltrated by œdema. If any hard substance, for example, the nail of the finger, be drawn over the skin, the redness is replaced for a little while by white over the places that have been stroked, a condition, however, which also occurs in other red eruptions upon the skin.

If the exanthem has developed in the above-described manner (scarlatina lævigata), it can scarcely be confounded with any other eruption. Nevertheless, there occur, though by no means frequently, deviations from the usual appearance of the exanthem which must be known in order to avoid errors in diagnosis.

The exanthem may resemble measles, if it should consist of somewhat larger, direct *roseolar* papules. Confluence occurs in these later, so that they are of a uniform red appearance, and then, in contrast to the usual, punctated-confluent exanthem, a macular-confluent redness is seen in which the original roseola may be recognised by a darker colour (*scarlatina variegata*). The roseola of scarlatina is differentiated from the macular eruption of measles in that they are less raised, do not show, as does the eruption of measles, a small red nodule in the centre, and also in that they extremely rarely occur in the face. In some rare cases the œdematous infiltration is limited directly to the discrete areas in which the exanthem appeared primarily, especially at the mouths of the hair follicles; in this case papules form which may be detected by feeling (*scarlatina papulosa*) and which, by a circumscribed elevation of the epidermis, may form into vesicles, and so give rise to *vesicular scarlatina, scarlatina miliaris.* This variety is especially seen in skins that perspire freely, and in some epidemics it has been prominent. If the eruption should be intensely developed, it may be characterized by the exudation of hæmoglobin and take on a livid-violet colour; this is especially the case in the hæmorrhagic diathesis, in which case, as with the other acute exanthemata, petechiæ and greater suffusions of blood occur in the subcutaneous cellular tissue (*scarlatina hæmorrhagica*). Frequently these hæmorrhages of the skin are accompanied with internal hæmorrhages, especially of the nose and of the genital apparatus. Bleeding from the urinary passages is also common in these conditions. These cases are almost exclusively of bad prognosis. Besides the scarlet-fever eruption we find, as with the cases of measles now and then, herpes, urticaria, pemphigus, pustules and other eruptions.

Full Development of the Eruption.—The maximum development of the eruption occurs between the second or fourth days, according to the mild or severe character of the case, sometimes earlier, at other times later. During this time the development of the other scarlatinal changes occurs, primarily of the *angina.* This is the most important localization of the scarlatinal process, because (in contrast to the eruption) it is never entirely absent, although the development may vary considerably in the individual case. Occasionally, during the height of the eruption, the mucous membrane of the palate and of the pharynx appears considerably swollen, eventually covered with diphtheritic membranes; occasionally even abscess formation may take place. With this, a phlegmonous inflammation of the region of the neck may develop (angina Ludovici)—all complications to which I will refer again later on. With the fully developed eruption or somewhat later, usually from the third to the fourth day on, desquamation of the coating of the tongue occurs, and now the *tongue,* which up to this

time has only been red at the point and at the margins, the rest having had a grayish-yellow coating, *appears intensely red, with markedly raised, swollen papillæ* (*raspberry tongue, cat tongue*). Next to the angina, this condition of the tongue is especially characteristic in its full development, perhaps even pathognomonic; the raspberry tongue lasts for nearly a full week.

The above symptoms, viz., the changed condition of the tongue, the angina, the exanthem, and the fever, are sufficient to establish the diagnosis of scarlatina at this stage alone. What else has been noted of the symptom-complex belonging to scarlet fever is of a secondary consideration from a *diagnostic* standpoint, as important as they may be from a prognostic point of view. This refers to the cerebral symptoms: The headache, the delirium, the somnolence, etc., to the higher pulse frequency which, corresponding to the temperature, may be from 120 to 140 and over. The slight enlargement of the *spleen*, which also seems to be present in the severest cases, is likewise of no great diagnostic importance, and just so the enlargement of the lymphatic glands of the neck. It is of greater importance to know that other lymph glands, such as those in the inguinal region, are not infrequently markedly enlarged in the course of the infection. Functional murmurs may occur over the heart, the urine may, even at this stage, contain albumin, on account of a mild irritation of the kidney by the toxines circulating in the blood.

Defervescence.—After the fully developed exanthem has remained at its acme for about one day, the red appearance gradually begins to fade, at first in the upper, and then in the lower, parts of the body. The gradual disappearance of the exanthem lasts from two to four days, as a rule. The other symptoms of scarlatina also gradually disappear with the passing of the eruption. Now, at the end of the first week or at the beginning of the second, the first signs of desquamation appear which, in contrast to the desquamation of measles, occurs in *large lamellæ*, rarely, and then only transitorily, in small flakes. This process may last from four to six weeks, occasionally even longer, and may repeat itself in individual areas.

Polyarthritis; Nephritis.—*Polyarthritis* with inflammation of the tendon sheaths and *nephritis* must be looked for during the period of desquamation, morbid phenomena in the course of scarlet fever, which are quite evidently due to a specific-irritative action of the infectious toxine. The *scarlatinal arthritic affections* are partly *purulent joint inflammations* in which cocci may be found in the joints, partly inflammatory infections of the joints of a more *transitory* nature. The latter may occur either in the large or in the small joints and may disappear after a few hours or days, similar to the condition in acute rheumatic fever, without anatomical changes being noted in the joint post mortem. *Nephritis* occurs with a very varying frequency in different scarlatinal epidemics. During the first nine days it is rare; if albuminuria is noted during this period, it is dependent upon a mild irritation of the renal tissue which is seen more or less frequently in all infectious diseases. On the other hand, a fully developed nephritis, with excretion of blood and epithelial casts, is a relatively [illegible]quent appearance at the end of the second, especially, however, at the

third, and, at the latest, at the end of the sixth week, which is usually in connection with a more marked excretion of the remainder of the scarlatinal toxine which occurs about this time.

Marked differences between the symptoms of this scarlatinal nephritis and an acute nephritis occurring from other causes, do not exist; there are to be expected: Diminished amounts of urine, appearance of œdema (a special predisposition of the skin to the condition being brought about by the preceding inflammation), mild and severe uræmic symptoms, etc. If the nephritis of scarlet fever does not have a lethal outcome, recovery occurs in a few weeks. I have rarely noted chronic nephritis resulting from this condition. Scarlatinal nephritis is similar in this respect to the nephritides occurring in other infectious diseases, for example, after diphtheria and pneumonia, in which I have seen the albuminuria disappear completely even after several months.

Unusual Symptoms of Scarlatina.—The symptoms just described are the usual ones in the majority of cases, on the basis of which the diagnosis may be made without any difficulty. In some cases, however, unusual phenomena occur in the course of scarlatina, which complicate the typical picture of the affection. These so-called "complications" of scarlatina may affect any of the organs of the body; they shall be mentioned according to their importance and frequency and briefly elucidated.

Pharyngeal Diphtheria.—*Diphtheria of the pharynx* in its mild or severe form, according to the epidemic, belongs to the common phenomena of scarlatina. Although scarlatinal diphtheria, as I have previously pointed out (see p. 243), according to my opinion, cannot be identified with the ordinary diphtheritic infection, i. e., at least in the majority of cases (except actual diphtheria which may occasionally occur as a complication during scarlatina), but must be looked upon as a mixed infection, being identical with genuine diphtheria in its gross anatomical appearance; therefore, we need not delay with the description of the membranes. Diphtheria of scarlet fever may also, the same as the other form, extend to the nose, the larynx and the tympanic cavity, and become especially dangerous in developing, secondarily, purulent cocci which subsequently find their way into the deeper structures, producing local abscess and gangrene, or which may give rise to septic phenomena in the entire organism. Especially dangerous is the distribution of these cocci in developing severe inflammations of the cellular tissue of the neck (angina Ludovici), which may be followed by gravitation abscesses into the mediastinum, secondary pleurisy, pericarditis, widely distributed gangrene or erosion of one of the vessels of the neck, thus leading to a fatal termination.

The same as other infectious diseases, so also may scarlatina give rise to *myocarditis* and *endocarditis* (which see). Many a valve lesion dating from early infancy may surely be referred to a previous attack of scarlatina.

Pericarditides are rarer, at least the severe varieties. The other serous membranes also become the seats of secondary inflammation, probably due to the fact that suppurative and pyogenic bacteria more easily find access to the soil prepared by the preceding scarlatinal infection. Thus there are found among the scarlatinal compli-

cations: *Meningitis, pleurisy* relatively frequently, mostly purulent, in rare cases also *peritonitis.* The *eye* is occasionally affected by keratitis, iritis, panophthalmitis, etc.; *affections of the ear* are very frequent, ranging from a simple catarrhal inflammation of the Eustachian tube to a purulent otitis media, by which route meningitis, sinus thrombosis, and cerebral abscesses may occur. Severe forms of *gastritis* and *enteritis,* with hæmatemesis and enterorrhagia, also occur in the course of scarlet fever, sometimes profuse diarrhœa with choleriform phenomena or dysenteric dejecta. Much more rarely than measles does scarlatina give rise to *affections of the air-passages.* We have already spoken of laryngeal diphtheria; there may also be œdema of the glottis, which especially occurs as the result of an angina Ludovici. Marked bronchitis, on the whole, is rare, as are lobular and lobar pneumonias; they may be accompanied with œdema of the lungs, gangrene or abscess of the lungs, and be complicated by empyema. Finally, the severe inflammations of the genitalia must be mentioned, which occasionally show a diphtheritic or gangrenous character. Periostitis and ostitis (most frequently of the petrous portion of the temporal bone) also occur.

Should these secondary, purulent processes attain marked development, should the organism, in the face of the general invasion of the accompanying micro-organism and their products of metabolism, not be capable of producing sufficient antitoxine to withstand such an onslaught, the clinical picture of *septicopyæmia* appears in the course of scarlatina, due to the secondary purulent inflammations.

Sequelæ.—It is easily understood that, on account of the mass of complications from local processes during the scarlatinal process, "*sequelæ*" take place: Lymph-gland enlargements, chronic purulent arthritis, caries, chronic middle-ear disease with and without paralysis of the seventh nerve, etc. Especially noteworthy among the sequelæ of a severe attack of scarlatina is the marked *general debility or a hæmorrhagic diathesis.* Similar as subsequent to other infectious diseases, so also in scarlatina we may note severe disturbances of the *nervous system*: Hyperæsthesia and anæsthesia, motor palsies, such as hemiplegia and paraplegia, paralysis of peripheral nerves, chorea, hysteria, psychoses, without there being the least possibility up to this time to analyze these occurrences in the nervous system in a satisfactory manner in each individual case. The *skin* may also for a long time afterward show decided feebleness of its structure owing to the effects of the scarlatina: A marked tendency to the development of furunculosis, to circumscribed or general œdema. This *œdema of the skin which arises without the presence of a renal inflammation* is very much rarer than that occurring subsequent to nephritis, and the diagnosis of the same is only justified after repeated examinations of the urine have demonstrated the total absence of albumin. Finally, the development of tuberculosis may occur after scarlatina, but not nearly as frequently as in the case of measles.

As we review the great mass of symptoms which may arise from the scarlatinal process, it would appear that the diagnostician had sufficient criteria at once, or at least after a longer or shorter observation of the case, to detect the disease with great certainty. In fact, the diagnosis of scarlatina belongs to the very easiest as soon as the exanthem has developed in a characteristic manner, the angina is at least indicated, if, in the second half of the first week, the scarlet-fever tongue is noted and the fever runs its typical course. If then, in the further course of the affection, the desquamation of large scales appears and, farther on, in the third week a nephritis takes place, no other disease but scarlatina can be diagnosticated. The greatest majority of cases shows this *ensemble* more or less well developed; in a few cases, however, the diagnosis may be difficult.

Anomalies in the Course of Scarlatina.—Occasionally the onset of the affection appears to occur in a normal manner; in its further course, how-

ever, some few symptoms of scarlatina may develop in an extraordinary way, and with great intensity: The fever, the angina, the lymph-gland enlargements, which then form abscesses, and are accompanied with a phlegmonous inflammation of the cellular tissue of the neck. These anomalies in the course of scarlet fever give rise to no great difficulties in diagnosis. It is, however, very different if the course of scarlet fever is *irregularly "abnormal" from the onset.* The prodomal phenomena with intense throat, nervous, and digestive symptoms may last unusually long, so that the exanthem appears not on the first or second, but only on the third or fourth, day, or even later. The eruption may exceptionally be limited to certain parts of the body, or it may appear in an unusual form, such as miliaria, pustules or pemphigus vesicles, and so give rise to difficulties in diagnosis. In other cases the exanthem is of so fleeting a nature that the patient does not notice it; and in rare cases the eruption may even be absent entirely (*scarlatina sine exanthemate*). An angina may then be all that such a scarlatinal infection produces; and yet, this angina must be designated as of scarlatinal origin, if it occurs after indubitable contagion from scarlet fever. Such cases may also prove themselves to be of scarlatinal origin in that, in spite of the absent eruption, desquamation or anasarca or a scarlatinal tongue appear later. On the other hand, neither the severe general affection, nor the high fever, nor an additional nephritis by no means prove the scarlatinal origin of such angina, as all these symptoms may occur in the course of an angina which is not due to scarlet fever. The opposite, *scarlatina sine angina,* also occurs especially if the eruption is but feebly developed.

It is also claimed that cases have been seen in which angina and eruption were absent and scarlatinal infection manifested itself only in nephritis and gastro-enteritis! Whether these are unusual cases of scarlatina cannot be decided until we succeed in isolating the toxine of scarlet fever; until then we must be guarded with our opinion in regard to the character of such cases. The same is true of other cases which are also counted as scarlatina, in which, during a scarlatina epidemic, outside of very rudimentary local phenomena which, however, as such cannot be with certainty declared to be scarlatinal, severe cerebral symptoms are present: Most violent headaches, eclamptic attacks, coma, persistent vomiting, excessive febrile temperatures, in which the patient succumbs with manifestations of collapse on the first day of the disease, sometimes even in the course of the first twelve hours.

It happens in isolated rare cases that articular inflammations, which otherwise do not take place until the desquamative period sets in, appear during the initial stages of scarlatina, thus producing an unusually morbid picture reminding us of acute rheumatism or sepsis.

Differential Diagnosis.—Of course, it would be of the greatest importance, in order to differentiate scarlet fever from other affections which present similar symptoms, if we would succeed in isolating the toxine of scarlet fever and to determine its character. Although researches have been made repeatedly for the bacteria of scarlet fever, and either bacilli were found in the blood of scarlatina patients, or *streptococci* were constantly demonstrated in the tonsillar membranes and also in the internal organs of patients who died of scarlatina, yet we are not in a position to-day to declare a certain kind of bacterium to be the positive generator of scarlatina. Therefore, we are restricted in the differential diagnosis up to the present

e solely to utilize the development of certain symptoms in the morbid picture, the rse of the fever, etc., to differentiate scarlet fever from other affections.

Erythema.—If the eruption of scarlet fever forms a *diffuse redness*, as is usually the case, the confusion with *erythema* may occur. But in the latter the uniform red does not contain those red points with which the scarlatinal eruption commences, and which do not blend with the general red until it has become very intense. Besides, in erythema the redness recedes very rapidly, whereas in scarlatina it persists usually for days; furthermore, in erythema there are no angina and no swellings of the cervical glands. With the appearance of the raspberry tongue later on, every doubt regarding the presence of scarlet fever is removed. The fever as a symptom of differentiation is of less importance, because rises of temperature occur also in erythema, and, on the other hand, it may happen that the fever may be absent entirely in some cases of scarlatina or be only very insignificant; if it adheres to the course typical of scarlatina, of course, the differential diagnosis is easy.

Erysipelas.—It is only possible, upon superficial examination, to confuse *erysipelas* with scarlatina, and only when it is a question of a very limited eruption of the scarlatinal exanthem. Whereas the punctated appearance of the cutaneous redness is in favour of scarlatina, an inflammation of the skin which is accompanied with diffuse uniform, but restricted, redness, and, above all, the strictly defined demarcation of the red, furthermore, the *slow* progress of the inflammatory boundary, as well as the œdematous swelling and painfulness of the exterior skin towards pressure, are characteristic of erysipelas.

Measles.—If the eruption is *roseolar* or roseolar-confluent, measles and such eruptions as have been mentioned in the diagnosis of morbilli are to be considered. For purposes of comparison the differential points in both diseases have been classified below; it must, however, be mentioned that only the *average condition of both affections* has been taken into consideration.

Differential Diagnosis

MEASLES	SCARLATINA
Incubation: Duration ten days.	*Incubation*: Duration five days (4–7).
Prodromes: duration three days. Affections of the mucous membranes, especially of the respiratory tract (coryza, conjunctivitis, bronchitis), Koplik's spots upon the mucous membranes of the cheeks and lips.	*Prodromes*: Duration one day and a half. Vomiting, angina, enlargement of the lymphatics of the neck.
Fever: High upon first day, on the second and third days of the prodromal age decreasing.	*Fever*: High from the onset, not falling during the prodromal stage.
Eruption: In the form of *discrete roseolæ* with central nodule, appearing and uniformly *in the face.*	*Eruption*: *Punctated-confluent, first on the neck*, back of the neck and ch *the face less affected*, the region aro the mouth free from eruption.
Maximum of the eruption from the to the second day after its appear- o sixth day of the disease).	Maximum of the exanthem second to fourth day of the aff decrease lasting two to four days.

MEASLES	SCARLATINA
Maximum of the temperature on the fifth to sixth day of the affection.	Maximum temperature from the second day of the disease, lasting several days.
Critical defervescence of the fever, so that normal temperature is reached from the seventh to the eighth day.	Defervescence by *lysis* towards the end of the first week, so that the temperature is usually normal towards the middle of the second week.
Tongue not characteristic.	Tongue from the third to the fourth day of the disease red, with swollen papillæ (raspberry tongue—cat tongue).
Desquamation commencing at the onset of the second week, in small flakes.	In the second week in LARGE LAYERS.
Complications and sequelæ—especially affecting the *respiratory organs* (catarrhal pneumonia, pleurisy, tuberculosis).	Appearing in the most varied organs, especially as pharyngeal diphtheria, endocarditis, septicopyæmia; *late complications*: polyarthritis, nephritis.

Rotheln.—*Rötheln* must also be considered in a differentio-diagnostic respect. The fact that in rubella the prodromal stage is merely indicated, the affections of the mucous membranes are of an exceedingly mild character, fever is absent, as a rule, and the eruption is especially developed in the face, makes the diagnosis of rubella (rötheln) certain. In fact, this is an affection so totally different from scarlatina that mistakes are scarcely possible, and the following description of rötheln will elucidate this sufficiently.

RÖTHELN, RUBELLA, GERMAN MEASLES, FRENCH MEASLES, EPIDEMIC ROSEOLA

Even up to the most recent time the character of *rötheln* as a substantive affection has been disputed in that it has been looked upon as a mild unusual variety of measles. Despite the fact that this opinion has always been defended, the majority of physicians have reached the conclusion that rötheln represents a *morbus sui generis*, arising epidemically by contagion, producing only rötheln, never measles and scarlet fever, conveying no protection against the latter affections, but showing an immunity against a new affection of rötheln in the individual. The course of the disease compared with scarlatina and measles is also entirely different.

The *period of incubation* probably lasts twice as long as in the case of measles, i. e., from two to three weeks, being without symptoms; the first phenomenon noticed in the disease is the appearance of the characteristic exanthem, *as a rule, without fever and without prodromal manifestations that are worth considering.* At any rate, the latter, when they are indicated, cannot last longer than from a few hours up to a day; they may then consist in affections of the mucous membranes of an exceedingly mild grade; Koplik spots never occur; if they have been noted, it must be very exceptional.

Eruption.—The chief diagnostic characteristic of the disease is the *eruption*, which, similar as in the case of measles, occurs first in the face and also particularly about the mouth (in contrast to scarlatina), then extending to the upper extremities and, finally, to the lower extremities. The red macules appear and disappear rapidly, a few hours after they have been fully developed; *in this manner it may occur that the eruption in the face may have already disappeared while the exanthem in the lower extremities is still in its acme*, whereas in measles, at the time of the full development of the exanthem, it is found to be uniformly distributed over the entire body. The eruption is rose-red, round, usually not serrated, and about the size of a lentil, rarely larger or smaller; they show no tendency towards confluence and to hæmorrhage. The eruption in the case of measles disappears with desquamation of fine scales, which is not the case in rötheln; as a rule, there is no itching.

Other Symptoms.—The other symptoms which occur besides the eruption are diagnostically less important. As has already been indicated, a mild catarrh of the mucous membrane of the respiratory tract may occur at the onset. There may also be affections of the conjunctiva and of the nasal mucous membrane; further, a partial reddening of the mucous membrane of the palate is regularly noticed, and almost constantly an enlargement of the lymphatic gland, most constantly of those lymph glands belonging to the back of the neck [posterior chain]. The appearance of the tongue is not characteristic, the urine extremely rarely contains albumin.

Of great diagnostic importance is the *condition of the temperature* in rötheln. In many cases there is absolutely no fever during the entire course of the affection; in a smaller number of cases there is mild fever, from 100° to 101° F., as a rule only in the onset, sometimes during the period of eruption. In keeping with the absence of fever (or the appearance of very slight fever), as well as with the fleeting action of the infection, the general health of the patient is very slightly disturbed. Complications and sequelæ are absent. On the other hand, relapses occur in rötheln, in which it has been noticed that the relapse is apt to be severer than the original attack.

If the symptoms of the disease are carefully considered, as well as the fact that rötheln occurs in epidemics independently of measles or scarlet fever, the *differential diagnosis* does not offer any difficulties. Doubts concerning the presence of scarlatina occur only rarely; but the differential diagnosis from measles should also be easy, especially if it is noted that a much longer period of incubation and an exceedingly brief prodromal period exist in the case of rubella, and that fever is absent. The differentiation of rötheln from other roseolar eruptions should be made in a manner similar to that followed in the differential diagnosis of measles (see Measles); I do not enter upon it here to avoid repetition. As the eruption itches in some rare cases, it may be confounded with urticaria; but mistakes will not occur in this case if the entire, generally typical course of rötheln is duly considered.

VARIOLA—SMALL-POX

The *source of contagion* must always be considered in the diagnosis of *small-pox.*

Ætiology.—Small-pox is an exquisitely contagious disease, the infection taking place either directly through the contents of the efflorescences of the pox, through the scabs and also through the blood (whereas the secretions and excretions of small-pox patients—sweat, saliva, fæces, urine, etc.—do not contain the small-pox poison), or through the exhalation of small-pox patients, so that a temporary presence near the patient, even without contact, is sufficient to acquire variola. The contagion may further be carried by fomites: Garments, letters, etc., and by corpses of those that have died of small-pox, as well as by a third person that may have remained perfectly well; the fœtus in uterine life may be infected through the mother, so that children may be born with small-pox or show the remains of an attack. Contagion exists during all stages of the disease; it is most pronounced during the stage of eruption, but it is also marked in the prodromal period; a transference of the affection to healthy persons is possible even in the last days of the period of incubation.

Incubation.—After the poison of small-pox has entered a susceptible organism it requires from ten to thirteen days until the first symptoms of the disease manifest themselves. Almost exclusively does the *period of incubation run its course without giving rise to symptoms*; only in rare cases are vague symptoms noted of a disturbance of the general health, pains in the small of the back and symptoms of pharyngitis, which in the prodromal period become more pronounced, and occasionally show themselves even towards the end of the period of incubation.

Nature of the Poison of Small-pox.—The clinical course of the affection commences with the onset of the prodromal stage, and it is so characteristic that it allows a diagnosis of variola to be made with positiveness. We are the more compelled to observe in the diagnosis primarily the individual peculiarities of the clinical

picture, as the *nature of the small-pox poison* has as yet not been definitely determined. It is certainly questionable, according to investigations, whether the *cocci* that have been repeatedly found in the pus of the pustules in variola, may be regarded as the specific virus of the affection. The same is also true of the bacilli which were on several occasions demonstrated in the contents of the papules occurring in small-pox and in the vaccine lymph. Of more importance are the findings of Guarnieri, L. Pfeiffer, and others, according to whom certain *protozoa* (*cytoryctes variolæ*) can be demonstrated in the blood of febrile variola patients, and which, later, are noted as small shining bodies in the epithelial cells of the skin at the areas of the variola efflorescences; they showed an increase by division into two parts and also possessed slow amœboid movements. It is to be hoped that, if we succeed in growing cultures of these protozoa better than was possible up to now and in utilizing these cultures in experiments upon the living organism, the ætiological significance of these cytoryctes regarding the variolous process will become clear.

Prodromal Stage.—The *prodromal stage* lasts from two to four days, in the majority of cases three days. It begins with a decided chill, or more frequently with several, less marked chills, and with this is associated a rapid rise in temperature to 102° F. to 104° F. The fever increases during the next few days, with slight morning remissions, so that 105° F. to 107° F. may be reached upon the evening of the third day. This condition is accompanied with a pulse frequency of 120 to 140 beats per minute, with marked lassitude, thirst, increased respiration, dyspnœa, nausea, and, eventually, vomiting; in short, with the symptom-complex which may be noted in any severe infectious fever. Somewhat characteristic are: *Intense headache* with delirium, the latter somewhat resembling that occurring from the excessive use of alcohol, ringing in the ears and vertigo, *pharyngitis,* transitory motor paralyses, and pain in the limbs, especially, however, *very troublesome pain in the loins and in the small of the back,* which in other infectious diseases is never so prominent as is the case in variola. The pains in the small of the back are also of prognostic value in so far as, the milder they are, the slighter the course of the affection may be expected to be. The *spleen* is said to be enlarged even during this stage; I have not paid particular attention to this point and, therefore, can give no opinion from my own observations. These pains in the head and back, in contrast to the condition in measles, are more prominent in comparison with the affections of the mucous membranes (coryza, photophobia, bronchitis) which appear towards the second half of the prodromal stage. On the other hand, the condition of the skin in the prodromal stage is of great diagnostic importance.

In a certain number of cases—varying in different epidemics as it appears—on the second day, less frequently on the first or third, various forms of eruption, the so-called "*initial exanthems,*" appear. They occur in two varieties, either in the form of a diffuse scarlatinal erythema or as a *roseola* resembling measles ("*rash,*"), or as closely crowded *petechiæ,* eventually upon an erythematous base. The roseolar exanthem usually appears on the second day, first upon the face, later upon the rest of the body, especially upon the extremities, and disappears completely in a short while, i. e., in about twenty-four hours. This eruption is found almost exclusively in mild cases (varioloid), and on this account is of prognostico-diagnostic

importance. A more unfavourable prognosis must be made in the secon variety of the initial exanthem, the *scarlatiniform, hæmorrhagic erythem in the prodromal period of small-pox,* which, as a rule, occurs on the fir day, is less fleeting and shows distinct localization. Its point of selection the lower abdominal region, reaching from here to the genitalia and to t inner surface of the thighs down to the knees, so that Th. Simon, to who we owe most of our knowledge of these prodromal exanthems, has spok of a "thigh triangle," the apex of which is formed by the closed thigh and the base of which corresponds to a line drawn across the abdomen the height of the navel. Another favourite place is the extensor surface the extremities and the axillary space with the adjacent regions of the upp arm and chest ("arm-chest triangle").

Stage of Eruption—Eruption.—At the end of the third day, or at th beginning of the fourth, the *specific small-pox eruption* appears, causin the diagnosis, which had been more or less probable before, to become abs lutely certain. The exanthem occurs in the form of small macules th size of a millet-seed, first in the face, upon the forehead, on the nose an the hairy skin of the head, etc.; in a short time, in the course of the fourt day, it spreads to the body and, finally, to the extremities. These macule become raised on the fifth day and change into conic papules; on the sixt to the seventh day a vesicle filled with a pale fluid appears at the apex o the nodule, and the contents of this vesicle gradually become turbid an more purulent upon the eighth to the ninth day; the form of the pustule then becomes globular and shows a depression at its apex (*umbilication*).

If the pustule is pierced by a pointed instrument, it allows its contents to ooz out *gradually*; this is due to the partition-like build of the pustule, which again i dependent upon a flake-like (necrobiotic) transformation (Weigert) of the cells o the *rete Malpighi*, which lose their nucleus and, being separated by the fluid con tents, form irregularly arranged trabeculæ. These connect the floor and top of th pustule and, in their further growth, allow the extension of the same more markedl towards the periphery than in the centre in which usually the excretory duct of a sweat gland is met with. These conic nodules remain solid in some few cases, an a small vesicle develops only at the top, after the desiccation of which the papul persists (*variola verrucosa*); other cases occur in which the originally small pus tules transform into large blebs with sero-purulent contents (*variola pemphigosa*).

Stage of Suppuration.—The skin in the surrounding of the pustules i markedly reddened. It is inflammatorily œdematous. Diffuse œdema oc curs in cases in which the pustules are closer together, so that especiall the face becomes thick, due to the swelling, is covered with pustules and with overhanging, spongy eyelids and narrowed lid space, and presents terrible appearance. The marked swelling and tension of the skin due t the œdema produces severe pains, especially in places in which the skin i normally tense, so especially on the hands. Further, at those areas in whic the epidermis is thickened by callosities, the accumulation of pus cannot, a in other places, bulge hemispherically—here the pocks, therefore, are flat with a grayish-yellow centre shining from the depths.

Mucous-Membrane Eruption.—Simultaneously with the development o the eruption upon the skin, the exanthem also appears upon the *mucou*

membranes. The oral and pharyngeal cavities are primarily and most markedly affected by the exanthem, which here at first forms only small red macules, then papules and, finally, whitish-gray vesicles that, scarcely developed (as the thin epithelial cover rapidly falls off), become ulcers which, if confluence of the eruption takes place, may assume larger dimensions. From the mouth the eruption of the mucous membrane spreads to the nose, to the œsophagus, to the larynx and to the trachea, causing salivation, dysphagia, hoarseness, and cough, eventually perichondritis and œdema of the glottis. If the eruptive process affects the tongue, which is by no means always the case, the latter swells to a shapeless, unwieldy mass. A formation of pocks occurs also upon the eyelids, and, as the result of this, ulcerative conjunctivitis and ectropium may develop; metastatic panophthalmitis has also been noted. An eruption of the pocks in the neighbourhood of the Eustachian tube and the extension of the inflammation to the middle ear will produce dulness of hearing, which may also be due to continuance of the process from the skin to the external ear, or to propagation of the inflammatory swelling to the drum. At the genitalia the eruption may find its way into the vagina; efflorescences of the eruption may also occur at the urethral opening and at the lowest part of the rectum, with troublesome consequences regarding the evacuation of urine and fæces. The *affections of the mucous membrane* occasionally develop earlier than the eruption upon the skin, so that sometimes, by a careful, early examination as to the appearance of the mucous membranes, important points may be obtained for the diagnosis.

Stage of Exsiccation.—As has already been mentioned, from the ninth day of the disease suppuration in the pock is in its fullest development, and the accompanying phenomena which have been described increase more and more in intensity. This stage of suppuration lasts about three days, and gives place, upon the twelfth day, to a new stage, the *exsiccation stage.* It may be remarked here that it facilitates the description to divide the course of small-pox into separate "stages"; in reality there is no marked demarcation between them, one stage gradually merging into another without giving rise to special phenomena. The stage of exsiccation is characterized in that the pustules which are filled with purulent material become flat and smaller by resorption or by an exit of their contents. The dry pus masses form crusts (stadium "crustosum"), which adhere tightly to their lower layers and, as they mechanically irritate their surroundings, give rise to irresistible itching; the ulcers of the mucous membranes heal by a new formation of the epithelium. Under the crusts of the pocks of the skin the epidermis regenerates itself from the papilli which have remained, or, if the purulent process has gone somewhat more deeply and the papillary body has been destroyed, a well-developed cicatricial tissue is formed. If now the crusts fall off, which usually occurs about the sixteenth day of the disease (stadium decrustationis), there remain, in the latter case, deep, white, striated scars which the patient usually retains for life. The loosening of the crusts is accompanied with falling off of the hair, which, however, grows again provided the process has not gone too far or too deeply and has thus given rise to the destruction of the hair follicle.

The falling off of the crust requires a longer time, so that, in a medium s vere case, it takes at least from four to five weeks before convalescence established.

Course of the Fever.—We have seen that the fever which ushers in t prodromal period may reach 104° F., and upon the second or third d may even be higher, reaching perhaps 108° F. *Coincident with the appea ance of the eruption, the temperature falls* now, in contrast to all of t other eruptive diseases (measles, scarlatina), *rapidly* to normal or to bel the normal. In general it may be said that, the more rapidly and co pletely the temperatu falls when the eruptio appears, the milder t case. The afebrile perio however, only lasts t suppuration occurs; no with the eighth or nin day, the temperature ris again to 103° F. or 10 F., the higher, the mo marked the purulent co dition of the skin. does not, however, reac the high range of the pr dromal period, as a rul Nevertheless, even thi "secondary fever" is ac companied with headache delirium, especially als with delirium tremen yes, sometimes even with symptoms of amnesia. The suppuration feve falls more or less rapidly to the normal with the appearance of exsiccatio (twelfth day of the disease). The course of the fever is, as may be noted, very characteristic one; although it may be modified by the severity of th affection, yet in the severest as in the mildest cases the general type of th temperature curve may always be observed as being to a certain extent typi cal (see curve, Fig. 75). Mild cases which run an afebrile course (variol non-febrilis), with an unquestioned, typical eruption, belong to the greates rarities; *vice versa*, there are cases of severe, "confluent" small-pox i which the prodromal fever shows only slight remission at the time of th eruption and at once passes into the fever of suppuration.

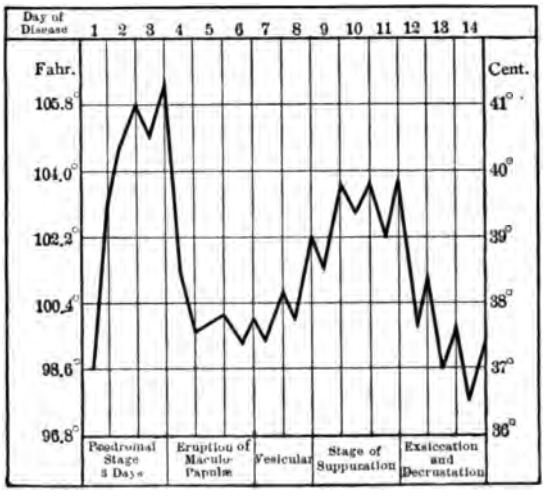


Fig. 75.—Fever Curve in Variola.

Different Varieties of the Course of Small-Pox.—The individual case of small-pox show other peculiarities, besides the fever, in regard to th severity of the clinical picture. We may differentiate the mildest form b the names of the *abortive variety* and *varioloid*, the medium severe variet as *variola*, and the more severe form as *variola confluens*, as *variola pus tulosa hæmorrhagica*, and, finally, the most severe form as *purpura vario losa*. However, a strict demarcation of the individual varieties from on another, apart from the last-named, the most severe form, in which th

genesis is by no means cleared up as yet, is impractical; it is useless, therefore, diagnostically to attempt a definition whether the individual case belongs to this or that group. Nevertheless, the separation of the above-named individual varieties from one another has its use, especially in regard to prognosis, and for this reason we shall briefly sketch the course of the different varieties.

Abortive Variety.—During an epidemic of small-pox cases are observed in which, after infection has taken place, although well-developed prodromal phenomena with fever arise, yet the eruption either fails entirely to appear on the fourth day or only a few pocks, going through their regular metamorphosis, become visible. In other cases the development of the pocks ceases with the papular stage, without giving rise to suppuration. It is clear that, without the appearance of papules or pustules, in the so-called *febris variolosa sine exanthemate*, the correctness of the diagnosis of variola, even if the ætiology be clear, can always be disputed, and that, accordingly, the existence of this variety of small-pox, as not demonstrable, can be denied. However, it dare not be gainsaid that it would be curious indeed why such abortive cases should be absent in a disease like variola, whereas their existence in other infectious diseases has been proved to be an absolute fact.

Varioloid.—*Varioloid*, since the obligatory introduction of vaccination and revaccination, has become the most frequent form of variola. The clinical picture of varioloid is less typical; the prodromal symptoms are sometimes milder than in variola vera, occasionally, however, they may be very pronounced, although generally of a shorter duration, in spite of the later milder course of the disease. In rare cases the fever may be absent in the prodromal stage, or at least be insignificant; on the other hand, it may attain a high range. But then it falls abruptly with the eruption and never rises again to a *secondary fever, which, if it should be noted at all, is at most indicated in varioloid.* Especially frequent in the prodromal stage of varioloid are the *initial exanthems.* Curschmann assumes that the purely erythematous, respectively the roseolar, eruptions are almost pathognomonic of varioloid, whereas the petechial ("triangle of the lower extremity") point to the development of variola vera. The *eruption* of the efflorescences of varioloid is *sparse*; there are but few macules, these occur precipitately and are irregular (occasionally not appearing first in the face). The development of the exanthem in its individual stages is less complete, the contents of the pustules is more sero-purulent, the suppuration is of a briefer duration so that exsiccation may eventually occur as early as on the eighth day; the affections of the mucous membrane also are much milder than in variola vera.

Variola Vera.—The picture which has been previously described of the course of small-pox conforms to so-called variola vera. It only remains for me to describe two varieties of variola vera, which, although unnecessarily, are diagnosticated and described as separate forms: The confluent and the hæmorrhagic forms.

Variola Confluens.—The erupted pocks from their onset are extraordinarily close together; the pustules which form from the papules are found on this account to merge into each other, so that the epidermis, while forming large vesicles filled with a purulo-serous fluid, is detached over large areas of the skin. This is especially noted on the hands and even more so in the face, which in this manner appears as if covered by a grayish-yellow "parchment mask" and is fearfully distorted, especially on account of the confluent pocks giving rise to great œdema. The mucous membranes show conditions similar to those of the skin. In fact, the highest grade of the development of the pocks may occur here, combined with diphtheritic ulceration, with glossitis variolosa, perichondritis, etc. The temperature, which in the prodromal stage is from 105° F. to 107.6° F., remains high in spite of the eruption appearing, or a remission to normal may occur which, however, is very transitory. Serious complications are very common in confluent small-pox; gangrene, nephritis, pneumonia, pleurisy, etc., may occur. Death may be due to these complications, or may take place after the clinical picture of pyæmia has

been noted (the pyæmia in this case is due to suppuration which, later on, occurs in the deeper layers of the cutaneous tissues); recovery is rare.

Variola Hæmorrhagica—*Variola pustulosa hæmorrhagica.*—This variety is characterized in that a varying number of the pocks, at the onset of their development or after they have become pustular, show their contents to be bloody, usually at first in the lower extremities. Hæmorrhagic efflorescences also form upon the mucous membranes, and to this are added hæmorrhages from the gums, the nose and the respiratory passages, as well as from various internal organs: The stomach, the bowels, and especially frequently from the uterus, and from the pelvis of the kidney. In keeping with these debilitating blood losses there are: A temperature course with a tendency to collapse, cardiac weakness, and an abnormal frequency of the pulse; recovery is even rarer than in the case of confluent variola. If the tendency to hæmorrhage occurs as early as at the beginning or at the end of the prodromal stage, before the development of the eruption has taken place, this variety, the severest of all, is usually designated by a special name, purpura variolosa.

Purpura Variolosa.—On the first day of the disease or at the beginning of the second, an intense prodromal exanthem occurs which at first is not suspicious, but which rapidly becomes hæmorrhagic and no longer disappears upon pressure of the finger. These hæmorrhages form small macules upon the extremities, and confluent suggilations upon the body, so that the latter shows a blue-black surface. This condition is accompanied with severe angina with a "diphtheritic" histolysis, with hæmorrhages from the gums, *hæmoptysis* and *hæmatemesis*, and also with profuse metrorrhagia; fæces and urine are discoloured by blood. Death occurs rapidly upon the fourth or fifth day of the disease, with the signs of collapse, which also manifests itself by a relatively low temperature even before a pustule has appeared. Occasionally some few hæmorrhagic pocks may form, showing plainly that this purpura variolosa belongs to the small-pox process. In other cases, in which this diagnostic mark is absent from the clinical picture, the recognition of the disease must depend upon ætiological data and upon the circumstance that small-pox is prevalent in the community at the time, that the malady began with fever, with intense pains in the head and small of the back, and with uninterrupted vomiting. Such diagnoses will always remain probable ones, if we mean to be thoroughly critical, but they equal many other diagnoses as to "certainty."

Complications and Sequelæ.—That a malady like small-pox, in which the morbid process is so far-reaching and in which the deeper structures are so frequently involved, gives rise to many *complications* and *sequelæ*, is self-evident.

Of the *complications*, in so far as they have not already been dwelt upon in the previous description of the disease, only the most important are to be mentioned. As in all other infectious diseases in which the respiratory organs are affected, so also in small-pox we note the occurrence of catarrhal pneumonia, and, as it seems, a tendency to tuberculosis, as well as pleurisy, mostly with a purulent exudate; further, serous or sero-purulent arthritic inflammations, in part with persisting ankylosis, parotitis either of a benign, or of a septico-metastatic, nature; more rarely pericarditis, endocarditis and myocarditis. *Orchitis* has lately been very frequently noted as a complication of small-pox, and, finally, also various kinds of *disturbances on the part of the nervous system.* The occurrence of delirium, and especially of delirium tremens, has been previously mentioned; various forms of mania may also be sequelæ of the variolous process, as well as meningitis and encephalitis; relatively less frequent than the last-named complications are the affections of the *spinal cord* as a result of small-pox, such as myelitis (disseminata) with paraplegia and especially ataxia (Westphal), acute ascending paralysis (Leyden); variola is followed, furthermore, by neuritis, peripheral paralyses, especially of the muscles of the palate, similar to diphtheritic paralyses. The occurrence of neuro-retinitis variolosa, besides the already mentioned complications on the part of the eye, should also be enumerated, and it must again be emphasized how very frequently the organ of hearing shows anatomical changes in the course of small-pox, so that permanent difficulty of hearing or complete deafness may often remain after recovery from variola.

Albuminuria is found in the severe cases both in the prodromal stage and in the further course of the affection; however, a severe acute nephritis with the appear-

ance of blood and casts is by no means a frequent complication of variola, and even less frequently is a chronic nephritis noted as a result of small-pox. The other changes in the composition of the urine which occur in the course of the disease are of an indifferent nature and as yet of no importance in the diagnosis.

Differential Diagnosis.—To confound variola in the stage of suppuration which has run its usual course with any other affection is absolutely impossible. This is, however, only true of the cases that run the regular course; as soon as variola appears in an atypical form, great difficulties regarding the diagnosis may arise.

As long as no eruption has made its appearance (therefore, during the first three days), the diagnosis is always questionable; and yet, in the interest of prophylaxis it would be of the greatest importance to be able to diagnosticate variola with certainty particularly in this stage, as it cannot be questioned that the transference of the disease to others is possible as early as in the prodromal stage. The abrupt rise of the temperature, with chills or with a decided chill, in the midst of complete health, and, further, the severe disturbance of the general health *upon the first day,* without special implication of any of the organs of the body, prove at once that an infectious disease has attacked the organism. But which infectious disease is present cannot be decided from the negative objective findings. I believe that it is theoretically and practically incorrect, if this condition is present, to review the various infectious diseases, miliary tuberculosis, enteric fever, etc., from a differentio-diagnostic standpoint. In practice we must say "non liquet" in such cases and—wait. It is suspicious, it is true, if *severe lumbar pains* are present as early as on the first day, especially if an epidemic of small-pox prevails at this time, as lumbar pains, with perhaps the exception of epidemic cerebro-spinal fever, are not so prominent in other infectious diseases as they are in variola. But if this condition is supervened by a *prodromal exanthem,* especially if it be *petechial* and in the form of a femoral triangle, the diagnosis of small-pox is as good as certain. The development of an *erythematous-roseolar* prodromal exanthem on the second day is not calculated to give the diagnosis a firmer support, as the question may arise whether measles or scarlatina may be present. In fact, the regular *eruption of scarlet fever* occurs already on the second day; but in measles, too, we sometimes note small maculo-papular exanthems as early as in the prodromal period, viz., on the second or third day. Naturally, the presence of affections of the mucous membrane (angina, photophobia, coryza, etc.) is more in favour of scarlatina or measles; but mild symptoms of an implication of the mucous membrane, especially pharyngitis, occur also in small-pox as early as in this period of the disease, i. e., at the end of the prodromal stage. However, *one* symptom of the prodromal period of measles is regularly absent in small-pox, namely the prodromal fall of temperature; *on the contrary, the temperature rises more and more on the second and third days in the case of variola, only to fall with the appearance of the eruption*; if, therefore, a fall in temperature is absent during the second half of the prodromal stage, this decidedly points to small-pox. It is in favour of scar-

latina and against small-pox if the temperature should reach its fastigiu with the appearance of the exanthem, and remain there.

If the eruption of small-pox has appeared (on the fourth day), the ci cle of the differential diagnosis is narrow enough so long as the exanthe is maculo-papular. Now, small-pox may only be confused with *measles* c eventually, with *typhus fever*. Measles and small-pox have a prodrom period that is almost equal; in both the eruption appears first in the fa and during this time has about the same appearance. As in the prodrom period, so also in this first stage of the eruption, the condition of the fev is a valuable differentio-diagnostic aid. *In the case of measles the te perature, which has fallen in the second half of the prodromal period, aga rises rapidly with the eruption of the exanthem; in the case of small-po on the contrary, the course of the temperature is just the opposite, as t fever falls with the appearance of the eruption.* This latter condition the fever also differentiates small-pox in the eruptive stage from typh fever, in which disease, as in measles and in small-pox, the roseolar exa them may also appear already on the fourth day, and among the initi symptoms of which the lumbar pains, which generally point to small-po may eventually be more prominent.

After suppuration of the exanthem has occurred, the diagnosis gair more and more in certainty. It is scarcely possible to confuse small-po in this stage with other pustular exanthems, especially not if the entir course of the disease is considered. Pustular eruptions due to drugs, suc as croton oil, antimony salve, etc., are never so uniformly distributed ove the body as in the case of variola, and usually also in varioloid, and, in con trast to the condition in small-pox, the suppurative stage is without feve The fever is also usually absent in impetiginous skin eruptions, in pustula syphilis, etc., in which, besides, the course is much slower, so that thes pustules only at the first sight of patients covered with them can be con sidered to be caused by variola. A mistake is much more likely to happe if pock-like eruptions occur in the course of another infectious disease, a is the case in glanders and in *septicopyæmia*. If the source of the septi infection cannot be discerned (cryptogenetic variety of septicopyæmia) the severe constitutional disturbances of the patient may give rise to doub as to whether we are not dealing with a case of small-pox in such an in stance. It is in favour of septicopyæmia that at this time no small-pox i known to be present in the locality, that the curve of the fever show marked remissions and exacerbations, that chills occur during the cours of the fever, that endocarditis, osteomyelitis, joint inflammations an ecchymoses of the eye-ground are demonstrable; all these symptoms do no occur at all, or at least are very rare, in small-pox. In glanders there ar always larger infiltrations, besides the pustules.

If the exanthem of small-pox is absent during the entire course of th disease, i. e., if the disease terminates in recovery, as the *abortive type* with the end of the prodromal stage or a few days later, without an erup tion having occurred, the diagnosis can only be made with a certain degre of probability—with great probability if at least head and lumbar pain and prodromal exanthems were present and if, besides, the history of th

case points to a variola infection. In the severest cases, in which death rapidly takes place under the previously described picture of *purpura variolosa*, the diagnosis can also be made only with a certain probability, although more so than in the mild abortive cases of *variola sine exanthemate*. It is true, it is not always possible to differentiate purpura variolosa from those cases of hæmorrhagic scarlatina which early terminate fatally. But a differential diagnosis between purpura variolosa and non-variolous purpura fulminans (in which death occurs in a few days, and also with the development of large blue-black ecchymoses) is absolutely impossible so long as we do not understand these obscure, pernicious varieties of purpura better than is the case to-day. The onset of the affection with severe pains in the head and in the lumbar region and the history may, it is true, direct the diagnosis to purpura variolosa, but it will not become certain until, on the fourth day or thereabouts, characteristic hæmorrhagic pustules are noticed here and there, besides the purpura exanthem.

The *division of the various forms of small-pox*: Varioloid, variola vera, confluens, and hæmorrhagica, presents no difficulty, but it has, as has already been indicated, very slight clinical value, especially as transitions between the individual varieties of small-pox are very common.

The differential diagnosis between varicella and varioloid will be found in the next chapter.

VARICELLA—CHICKEN-POX

That varicella is a *morbus sui generis* and that it is independent of variola, is probably admitted everywhere to-day. It is an affection confined to infancy; it never, by contagion, gives rise to small-pox, but always to varicella; it is quite independent of any connection with epidemics of small-pox and is noted in certain cities annually (as I can prove from personal observation), whereas not a single case of small-pox occurs at the same time. Vaccination does not confer immunity to varicella, the recovery from varicella does not protect from an attack of variola and, *vice versa*, vaccination of children that have just recovered from varicella, gives a positive vaccination result—all facts of experience the correctness of which is absolutely determined.

The period of incubation appears to be somewhat longer than that of small-pox, from thirteen to seventeen days. The disease very commonly sets in at once with the appearance of the eruption, without prodromes having preceded; the latter may be indicated in other cases, which, however, are very rare, by a brief, mild fever (which lasts only a few hours), lassitude, anorexia, etc.

The *exanthem of varicella* shows very characteristic peculiarities. It sets in with a roseolar eruption, which occurs in the upper half of the body (but by no means always first in the face) and then extends to the extremities. Without going through a papular stage, these macules change in a few hours, rarely lasting from one half to an entire day, into vesicles, the size varying from that of a lentil to that of a pea, which are tightly filled with a transparent, light fluid and without exception are *not umbilicated*. The clear, serous contents of the vesicle may later become turbid from an admixture of the leucocytes; but it will almost never become pure pus. The vesicles occupy the entire area of the previous roseolar eruption, so that they remain with a reddened areola; they are discrete, confluence is scarcely, if ever, noted. The duration of the individual vesicles is short: After from one half to one day they become flabby or burst, and small yellowish-brown crusts form from the centre, which desquamate in a few days without leaving deeper scars. *It is especially characteristic that new roseolar macules appear between the vesicles that have already developed, to change into vesicles*, a condition which does not occur

in the development of the exanthem of small-pox. The last-developed roseoles cha less and less into varicella vesicles; many may become pale without having gi origin to a vesicle. The entire period of eruption, as a rule, does not last longer t two or three days, so that, usually upon the fifth day, the entire eruption shows cr formation.

Fever.—The eruption occurs either without fever or, what is much more quent, with very slight rises of temperature, from 101° to 102½° F. This n fever lasts a few days and has a critical defervescence; as long as there is fe relapses of eruption are apt to occur.

Other Symptoms.—Besides the eruption upon the skin, efflorescences occur (oc sionally, as it appears, even earlier than the skin eruption) upon the mucous m branes of the oral cavity and of the pharynx, upon the hard and soft palate, on tongue, etc. Combined with the exanthem in the pharynx is slight difficulty in de tition; there may also be enlargement of the lymph glands of the pharynx. Affecti which run their course during an attack of varicella have been noted; but severe c plications and sequelæ rarely occur in this harmless infectious disease, except oc sionally otitis, bronchial affections, etc.; relatively often has nephritis been obser to occur as a result of this affection.

Differential Diagnosis—Varioloid.—It is possible to confound varicella w other diseases which show a similar eruption. The most has been said and writ about a confusion of varicella with the *pustules of variola*. The marked differen are: The almost exclusively clear or only slightly turbid appearance of the c tents of the varicella vesicles compared with the purulent composition of the vari pustules, the rapid development of the usually non-umbilicated varicella vesicles fr roseolar maculæ, without the intervening stage of papules, the eruption of varice in successive crops (so that, besides the fully developed, yes, even besides t incrusted varicella vesicles, fresh roseoles may be found), compared with the *unifo development of the eruption of small-pox*; further, the short duration of the in vidual life of the varicella vesicles and the course of the *prodromal stage*, which, the case of varicella, is entirely absent or at least only indicated, whereas, in varic and varioloid, it is accompanied with very severe conspicuous phenomena (headacl lumbar pains, prodromal exanthems) and lasts several days. Characteristic, mo over, is the conduct of the fever which is absent in varicella prior to the appe ance of the eruption or, at most, exists but a few hours, and usually does n decline with the appearance of the eruption, provided the disease does not run afebrile course. The differential diagnosis between varicella and varioloid in pr tice is even more simple still, in that varicella does not occur in adults at all b only in children under ten years of age, and that the epidemic prevalence at the ti of one or the other disease is apt to influence the diagnosis in the proper direction.

Pemphigus, Herpes, etc.—The *exanthem of pemphigus* may very closely resemb varicella, especially as some few varicella vesicles may develop more from th periphery and attain the size of a silver dollar. But the *course* in pemphigus und all circumstances is more *protracted* than in varicella, in which disease the eru tion has ceased after two or three days, whereas in the case of pemphigus relaps may occur for weeks, and the vesicles are sometimes much larger in circumferen (the size of a hazel-nut or larger) than those of varicella. Otherwise the course the disease in pemphigus may closely resemble varicella, the eruption may run i course with fever and the mucous membrane of the mouth also be implicated.

Mistaking varicella for the *eruption of herpes* may be prevented if we consid that the vesicles of herpes in general are smaller, usually limited to the course of nerve, and form in groups, whereas varicella, almost exclusively without any u formity, are distributed all over the body. The vesicles of *miliaria* are also mu smaller, upon the average; their contents have an acid reaction, those of varicel vesicles being neutral or alkaline. Certain varieties of *syphilitic exanthems*, whic however, are extremely rare according to my experience, show the external appea ance of varicella completely ("varicella syphiliticæ"); but they may easily be di ferentiated by their more protracted course and by the simultaneous appearance other syphilitic phenomena in the same individual. Finally, there are *artifici* eruptions which may resemble varicella, produced by burns or by the use of certa

drugs (cantharides, etc.). But they may be differentiated from varicella by their localization upon circumscribed areas of the body, which have come in contact with the drug, and by the inflamed condition of the regions adjacent to the vesicles.

ERYSIPELAS—CRYPTOGENETIC ERYSIPELAS—ST. ANTHONY'S FIRE—THE ROSE

Erysipelas is an infectious disease, the cause of which, in contrast to those which have been described up to now, is known with certainty. Erysipelas is due, as Fehleisen has shown, to the action of cocci and especially of streptococci, which, finding their way into the skin and mucous membranes, produce a specific inflammation of the same. The mode of invasion can frequently be determined, as the streptococci may use an open wound as their mode of entrance ("surgical erysipelas"). However, the route by which the infection has taken place is not always found, even after the minutest investigation; on the contrary, we could scarcely go too far in assuming that, in about one half of the cases, the erysipelas cocci have entered the susceptible organism without the route being in the least manner demonstrable, similar to the condition in septicopyæmia in a great number of cases. These cases of "idiopathic" or "medical" erysipelas should more suitably be termed *cryptogenetic erysipelas*. The diagnosis of this latter shall concern us principally, as surgical erysipelas belongs to the domain of surgery.

Streptococcus Erysipelatis.—After various observers had determined the fact that micro-organisms are found in the tissue of the *erysipelatous* skin, Fehleisen, in 1882, discovered a *streptococcus* which proved to be the specific cause of erysipelas. This micro-organism forms long chains (consisting of from ten to twenty links or more), can be grown upon gelatine without liquefying it; the streptococci grow quicker and more luxuriously at the heat of the body upon agar, blood serum and in bouillon. These streptococci cultures, taken from erysipelatous patients and subcutaneously inoculated into animals or man, produce erysipelas, i. e., a redness and swelling of the skin which advances from its periphery. Upon microscopic examination these cocci are found nearest the external margin of the diseased tissue, and almost exclusively in the lymph spaces and lymph channels of the skin, completely proliferating in them and clogging them. The period of incubation of inoculated erysipelas, from the time of the injection to the initial chill, respectively to the time of onset of the eruption, was from fifteen to sixty hours. Erysipelas has also been noted upon the mucous membranes, as we shall see, and occasionally the affection of the mucous membranes is the primary one: Hartmann has in fact noted that the erysipelas cocci are capable of accumulating upon the mucous membrane of the upper part of the intestinal canal and of the respiratory organs, but even here the route of selection is by means of the lymph channels.

As erysipelas usually runs its course without pus formation and septicopyæmia, the long chains of streptococci which were found uniformly in these three disease processes, have been looked upon as different varieties of streptococci, so that one variety only produces erysipelas, the other local pus formations, and the third septicopyæmia. This opinion has lately been more and more abandoned, as the results of a great variety of investigations have proven the *identity* of the cocci found in these various affections, in a morphological as well as in a pathogenic respect. We find streptococci as the cause of disease not only in erysipelas, in purulent conditions and in sepsis, but also in various forms of puerperal diseases, in meningitis, endocarditis, periostitis, etc., and, further, as the cause of mixed infection in other infectious diseases, as in scarlatina, diphtheria and certain varieties of pulmonary tuberculosis. Undoubtedly, it is only the varying grade of virulence of the streptococci which determines the picture of the disease in the individual case: In a rabbit the *least virulent streptococci produce* local *purulent conditions*; the *more virulent, erysipelas*; and the *most virulent, septicæmia*, usually without local changes at the point of inoculation. It has further been shown that we are capable, by inoculation and reinoculation from animal to animal, to change the virulence of the special streptococcus variety in such a manner that the variety which originally

gave rise only to local pus formation, now produces erysipelas or even sepsis. According to this we now understand that, although erysipelas usually retains its character from the onset to the end of the affection, nevertheless, according to the virulence of the streptococci, the nature of its development and the condition of the involved tissue, occasionally pus formation or even septicæmia may occur subsequent to the course of true erysipelas.

In judging the course of erysipelas, these facts, which we owe to experimental-bacteriological research, are to be remembered, even if they are not directly concerned in the diagnosis of erysipelas. For this affection occurs with such characteristic symptoms that the diagnostician is rarely in doubt whether he has to do with erysipelas or not. The phenomena which are the result of the infection are partly of a local, partly of a general, nature; the latter are unquestionably the result of the chemical poison produced by the streptococci.

Exanthem.—After a brief (judging by inoculation experiments, one to three days) *period of incubation*, and after a questionable, as a rule entirely absent, *prodromal stage*, the affection sets in with chilliness or with a decided chill, occasionally also with vomiting; at the same time there appear, if it is a question of erysipelas of the skin, on some parts of the surface of the skin certain changes which show the disease to be erysipelas. The skin of the face is most frequently affected (erysipelas faciei); in the affected area in which the lesion has occurred, or if it be a case of cryptogenetic erysipelas, usually upon the saddle of the nose, a large red area appears which distributes itself to both sides to the cheeks, forming the figure of a butterfly. The affected skin is hot, *markedly reddened*, often bright red, shining, œdematously *swollen*, the folds have disappeared and, what is especially characteristic, there is *a sharp line of demarcation from the surrounding healthy tissue*. A sensation of painful tension is present, and the *pain* is markedly *increased by pressure*. The *margins* of the reddened and swollen skin are raised, in advancing erysipelas tongue-like or serrated extensions invading the healthy tissue. The *surface* is either smooth or covered with small vesicles (erysipelas miliare, vesiculosum), rarely with larger vesicles (erysipelas bullosum, pemphigoides) which contain a clear, serous or yellowish, yes, even hæmorrhagic, fluid. After the inflammatory swelling and redness of the skin have lasted for two or three days, they have attained their maximum. The skin gradually becomes paler, and a desquamation takes place either of small or of larger scales; the vesicles burst and crusts form. The *distribution* of erysipelas occurs in all directions from the margin of the swelling, according to the tension of the skin; *but at some parts of the body it finds a natural obstacle, especially in those regions in which the skin is more tense and tightly bound to its lower layer.* Therefore, it is noted that facial erysipelas is almost always limited by the *chin*, the back of the *neck*, or by the border of the hairy scalp or somewhat under this; erysipelas of the trunk is limited by *Poupart's ligament* or by the crest of the ileum, by the gluteal fold, etc. However, erysipelas does not always stop at these natural boundaries; it rather advances, in individual cases, slowly and continuously from place to place: From the face to the back of the neck, from there to the trunk and to the extremities (erysipelas "migrans"). Very marked inflammation and tension of the skin cause gangrene (especially at the eyelids) as a result of the severe cir-

culatory disturbance. The entrance of the streptococci into the deeper cellular structure of the skin gives rise to severe suppuration (erysipelas phlegmonosum); the lymph glands in the neighbourhood of the inflammatory process are enlarged.

Other Symptoms.—Besides the changes in the skin, the erysipelatous infection shows itself by more or less severe general phenomena. The fever, in certainly two thirds of the cases beginning with a chill, rapidly reaches 104° F. or over, and remains at its acme (104° to 106° F.), with very slight remissions, until the erysipelatous process has run its course; this is usually the case in from ten to twelve days. In other cases the exanthem may last for weeks or months, the fever also continuing, but interrupted in these cases by longer or shorter intermissions (in keeping with the temporary recession of the inflammation of the skin); the defervescence of the fever as a rule is critical. In rare cases erysipelas may run its course *without fever,* to which I can testify.

The sensorium, as a rule, is markedly affected: Headache, delirium, insensibility are rarely absent in erysipelas; apparently dependent less upon the height of the fever than upon the effect upon the brain of the poison produced by the streptococci. I have occasionally even noted transitory aphasia. The brain may be found to be normal at the autopsy in spite of pronounced *cerebral manifestations intra vitam. Meningitis* occurs in other, but very rare, cases, due eventually to a sinus thrombosis or an inflammation of the orbital connective tissue. This last complication has frequently given rise to sudden blindness and atrophy of the optic nerve. *Albuminuria* is found in erysipelas as a result of the toxic irritation of the kidneys; to determine from the excretion of albumin that an infectious *nephritis* is present, an often-repeated examination of the urine for epithelial casts is necessary. Well-developed nephritis is not frequent in erysipelas, as the toxines of the latter affection are not a poison which specially irritates the kidney; nephritis may be absent even in the severest cases of erysipelas, according to my experience. Should nephritis occur, it disappears more or less rapidly during the course of convalescence; it becomes chronic as rarely as the complicating nephritis of other infectious diseases. Systolic murmurs are frequently heard over the *heart,* which in the majority of cases must be designated as functional. Nevertheless, they are occasionally a sign of endocarditis due to the fact of erysipelas cocci having found their way into the blood. This connection has been made plausible by experiments; the same is the case with the metastatic development of purulent *arthritic inflammations,* which occasionally occurs in erysipelas. Intravenous injections of pure cultures of the streptococcus erysipelatous into the blood of rabbits were regularly followed by purulent inflammations of the joints; pus taken from an inflammation of a knee joint occurring in the course of erysipelas in man has been shown to contain pure cultures of the pathogenic streptococcus. A further action, and as it appears a specific one, has been claimed for the erysipelas coccus upon the mucous membrane of the *upper portion of the intestinal tract,* producing an enteritis and ulcers in the course of which occasionally diarrhœa occurs, the stools containing an admixture of blood. From the oldest times

the frequent coincidence of *gastric disturbances* (signs of dyspepsia, coated tongue which dries rapidly and becomes fuliginous, bilious vomiting, etc.), and erysipelas has been noted. For the present we can only look upon these complicating gastric phenomena as due to a secondary intoxication, as well as the mild enlargement of the spleen, but which, according to my experience, is by no means constant. Rarer complications are ***pericarditis, peritonitis, purulent pleurisy,*** and *pneumonia.*

Erysipelatous Pneumonia.—Regarding the latter, certain forms of pneumonia, the so-called "*wandering pneumonias,*" have for a long time been brought in connection with a general erysipelatous infection, in which localization takes place in the lungs. It is true that certain pneumonias are characterized by a serpiginous advancement in the lung, similar to the gradual advancement of erysipelas upon the skin, and by exacerbations of the fever which go hand in hand with the local process. These pneumonias are said to have been noted especially in those times in which erysipelas of the skin was very frequent. The possibility that the erysipelas streptococcus may colonize in the respiratory organs, develop there and give rise to an erysipelatous pneumonia, cannot be put aside, according to experimental investigations. A positive diagnosis, however, could only be made if in the individual case it were possible to find pure cultures of streptococci in the sputum of such pneumonia patients. This serpiginous character of the infiltration, the exacerbations of the fever, the enlargement of the spleen, etc., prove nothing regarding the erysipelatous nature of the pulmonary inflammations; all these symptoms, according to my experience, occur comparatively frequently in simple croupous pneumonia.

Erysipelas of the Mucous Membranes.—Of greater importance, especially from a prognostic standpoint, is the implication of certain *mucous membranes* in the erysipelatous process, be it that they are primarily affected or that a cutaneous erysipelas in its development has affected the mucous membrane. The mucous membranes of the mouth and pharynx are particularly exposed, as well as that of the female genital organs. *Pharyngitis erysipelatosa* is characterized by a more marked swelling and deeper redness of the mucous membranes of the mouth and pharynx, usually including the tonsils; vesicles and small losses of substance may be noted upon the inflamed surface. The process then spreads to the mucous membrane of the nose and finds its way to the external skin, and in this manner the erysipelatous character of the pharyngitis becomes manifest. So long as the external skin is not affected, the diagnosis cannot be made with certainty, as the other differential symptoms—enlargement of the spleen and albuminuria—according to my experience, occasionally occur with other varieties of angina. The observation that fever, which may last for days, precedes erysipelas of the face, might be explained by the reason that erysipelas of the pharynx may have been overlooked in such cases. Should erysipelas which has established itself upon the mucous membrane of the pharynx, develop downward, laryngitis (erysipelatosa) and the complication of erysipelas which is rightly so much feared, *œdema of the glottis,* may occur. The trachea and bronchi may also be affected by the erysipelatous process, as autopsies have proven, which may occasionally lead to terminal pneumonia. The second and much more important form of erysipelas of the mucous membrane is that affecting the *female genital organs.* These organs are especially predisposed when they are in the *puerperal* con-

dition. The virus, finding its way through the tubes into the peritonæum, develops a severe streptococcus, puerperal fever. This has lately been clinically differentiated from other varieties of puerperal fever. We cannot enter into a discussion of this subject here.

Sequelæ.—*Sequelæ* as a result of erysipelas are rare, as a rule; nephritis and endocarditis have already been mentioned; a transitory *falling out of the hair* has been commonly noted after erysipelas. After frequent relapses—erysipelas belonging to those infectious diseases the recovery from which predisposes to relapses—occasionally a hyperplasia of the lower-cell tissue of the skin and a dilitation of the lymph channels is noted which is known by the term *elephantiasis*; scar formation occurs after erysipelas provided the process has run its course with deep abscess formation and gangrene of the skin.

Differential Diagnosis.—Erysipelas is rarely confounded with other diseases, it being a malady which, if once seen, is recognised at first glance. In individual cases the *ætiology* is of value in the diagnosis; thus the fact that the affected person has been in contact with a case of erysipelas a few days previously, or was in a ward of a hospital next to an erysipelas patient, etc. I am compelled regarding this point to assert, whether I wish to or not, that the *transmission of erysipelas cocci is possible even from some distance.* The point of invasion may occasionally be noted in the markedly swollen, reddened, painful portion of the skin showing the characteristic margins, as a *læsio continui,* a small scratch wound of the nose, upon the head, etc. As easy as is the diagnosis of erysipelas in the majority of cases, it must, however, be stated that cases occur in which the diagnosis may give rise to great difficulties.

Phlegmons.—*Phlegmonous inflammations* may be easily mistaken for this affection. Fever, swelling, pain and reddening of the skin, lymphatic enlargement are at first sight very similar to erysipelas. Upon careful consideration, however, some points of difference are noted: The phlegmonous swelling of the skin is board-like, the redness darker, and, what I consider the principal difference, is *never* noted to end with a *sharply defined, serrated border* in contrast with the healthy skin, as is the case in erysipelas; later, when the infiltration passes into pus formation, the differentiation of both conditions is less difficult.

Lymphangitis.—Further, especially in erysipelas of the extremities, the differential diagnosis must be made from *lymphangitis,* as circumscribed swelling and reddening and pain of the skin, fever and, occasionally, gastric symptoms are also present in this condition. Commonly, however, lymphangitis is characterized by the fact that the redness, at least in its later course, becomes more bluish and is markedly *striated,* whereas in erysipelas the colour is light red and is very diffuse, and at most some few stripes may be seen to stretch from the border into the healthy cutaneous tissues. Besides, the inflamed lymph vessels, as a rule, may be felt as hard bands being painful in their course, as well as the lymph glands belonging to them. It is rare for lymphangitis to show a reticular instead of a striated form concentrating itself upon the lymph capillaries. The differentiation from erysipelas is then somewhat more difficult; it is possible if

the circumstance is observed that the redness in the reticular form lymphangitis is composed of the smallest red areas, therefore is less ui form and less sharply demarcated as in specific erysipelas. Further, the are commonly vesicles upon the affected cutaneous surface in this ca whereas in lymphangitis multiple small abscesses may form in the neig bourhood of the inflamed lymph channels. Finally, I should like to e phasize the fact that it must be remembered in the differentiation of infla mation of the lymph channels from erysipelas, while having clinical valu that both affections are due to the same cause, being produced by strept cocci.

Anthrax.—Occasionally *anthrax* may give rise to diffuse swelling a reddening of the skin resembling erysipelas. The anthrax tumour, ho ever, is hard, becoming œdematous towards the periphery, with a dee gangrenously scabby centre. This is surrounded by a red-violet infiltrat base and a border of vesicles which are filled with a reddish-black fluid. I doubtful cases a microscopic examination of the blood and of the fluid o the œdema must be undertaken for the detection of anthrax bacilli, an inoculations of the blood or of the pure cultures of bacilli into animal (guinea-pigs, mice) must be made to establish the diagnosis with certainty

Erythema.—*Erythema* can scarcely be confounded with erysipelas. The *diffus varieties of erythema* have a more *superficial* and *fleeting* character than erysipela and are scarcely to be considered in a diagnostic respect. But erythema exsudativun multiforme and nodosum may also be easily differentiated from erysipelas. *Ery thema multiforme* consists of several red efflorescences and forms very characteristi figures, due to the fact that the centre of the eruption sinks and becomes bluish-re whereas the markedly red periphery extends and coalesces with the neighbouring eruption (erythema gyratum, iris). In contrast to erysipelas, the dorsa of th hands and feet are the points of election of erythema multiforme; the erythematou parts of the skin are *not painful* upon pressure, and fever and constitutional symp toms are absent, as a rule, in this cutaneous affection. However, these latter phe nomena, similar to erysipelas, are pronounced in *erythema nodosum*; also the cuta neous changes (pale to blue red nodules) which arise in the course of this affec tion, are painful upon pressure; the erythema nodules, however, occur at once in great numbers and do not enlarge, as the erysipelas inflammation, by an advance of the infiltration at the periphery. The nodes disappear by changes of colour, a common extravasations of blood, and it may then by a superficial, careless examina tion be mistaken for the residues of trauma, but they can scarcely be confounded with erysipelas.

TYPHUS FEVER—TYPHUS EXANTHEMATICUS—SPOTTED FEVER—SPOTTED TYPHUS

Exanthematic typhus easily ranges itself with the previously described "acut eruptive diseases" in every respect; with enteric fever, with which it was formerly brought in relation, it has absolutely nothing in common outside of the severe dis turbances on the part of the nervous system and the occasionally occurring enlarge ment of the mesenteric glands and of Peyer's patches. As measles, scarlatina, etc. typhus fever is a *markedly contagious* disease, contagious from person to perso and by the exhalations of the sick, being capable of transmission by fomites as wel as by a third person that may have remained free from the disease, and, of course occurring in endemics and eventually in epidemics. The *contagious principle itself* is as yet *unknown*.

Prodromal Stage.—After a *period of incubation* which varies greatly (from hours to weeks, usually reckoned to be from one to two weeks), running its course without symptoms or upon the last days prior to the appearance of the disease giving rise to vague phenomena—malaise, insomnia, headache, eventually also coryza—the disease begins suddenly with a decided chill or with repeated chilliness, frequently also with vomiting. The temperature *at once* rises to 104° or 105° F., and with this the usual accompanying phenomena of high fever appear: Heat, thirst, anorexia, turgescence of the face; the tongue is coated and soon becomes fuliginous. To this there are added symptoms which in their intensity and peculiarity cannot be regarded as being dependent upon the degree of fever, but must be looked upon as the expression of the infection: Conjunctivitis, coryza and angina, laryngitis and bronchitis appear and, above all, unusually marked disturbances on the part of the nervous system: Vertigo, tinnitis aurium, headache, great muscular weakness and pains in the limbs increased by movement; further, early marked sopor, wild delirium and hesitating speech. Even in this stage, which may be looked upon, in analogy to other acute exanthems, as the *prodromal stage,* an enlargement of the spleen may also be recognised. The pulse, in keeping with the fever, is increased in frequency (120 and over, a minute); it is certainly not relatively slow and only exceptionally dicrotic, as is the case in enteric fever (typhus abdominalis).

Stages of Eruption and Florition—Exanthem.—In the second half of the first week, i. e., from the fourth to the sixth day, the *exanthem* appears: Roseola macules, as a rule, appearing first upon the belly and thence spreading to the extremities (especially upon the extensor surfaces) and, although less frequently, to the face; upon pressure they become pale, whereas later, after a few days, upon pressure with the finger some hæmoglobin remains in place of the eruption. The entire exanthem may on this account obtain a more livid appearance ("petechial typhus"). The number of spots varies greatly—from several thousand to but a few; yes, Lebert claims that he has observed undoubted cases of typhus fever in which the exanthem was absent altogether. The *duration* of the eruption is about a week, somewhat longer if petechiæ have occurred. With the disappearance of the eruption in the third week desquamation of the finest scales occurs, rarely of large flakes.

With the appearance of the eruption, therefore towards the end of the first week, the severest stage of the eruption is ushered in (status typhosus), being characterized by profound stupor or coma, high temperature, increased pulse frequency (up to 140), distinct splenic tumour (frequently not painful upon palpation), involuntary evacuation of urine and fæces, bronchial affections, and generally also by the voidance of urine containing albumin, by increase of the weakened heart action and the appearance of functional systolic murmurs. If the course of the disease be favourable (mortality about 15 per cent), at the end of the second week or at the beginning of the third, a change takes place, which is rapid, as a rule, with a critical defervescence, or it may be somewhat prolonged but at least occurring in the course of about two days. Quiet sleep and perspiration set in,

cough disappears, the eruption fades, the urine loses its albumin—con valescence makes its appearance.

Fever.—Of importance for the diagnosis is the *course of the temperature curve in typhus fever*, especially in distinguishing it from enteric fever, the symptoms of which at the height of the affection may show some resemblance to typhus fever. In contrast to the course of the curve in enteric fever, the temperature in typhus fever does not rise step-like but *suddenly*, remains at its acme, 104° F. or 105° F., with slight morning remissions, to fall *comparatively rapidly* upon the tenth to the fourteenth day, rarely a few days earlier or later. *Crisis* is either complete in a half

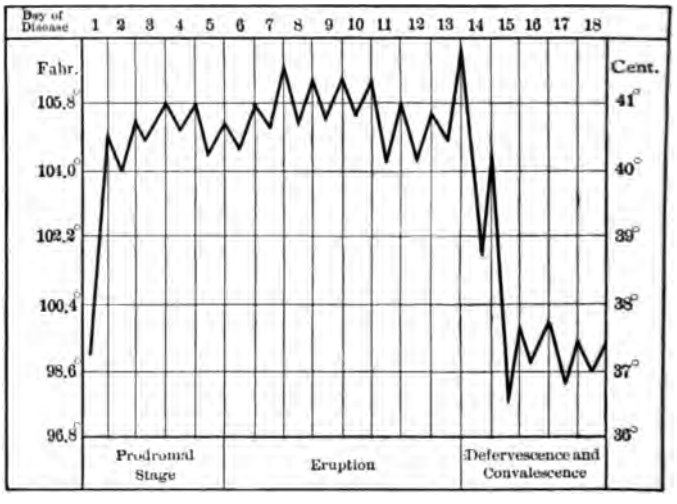


FIG. 76.—AVERAGE TEMPERATURE CURVE IN TYPHUS FEVER.

day or may be somewhat more protracted, lasting two or three days. Occasionally, before the crisis occurs, a so-called precritical rise precedes, with transitory excessive rise in temperature, vomiting and chills. The opposite is also seen, namely that the temperature before the crisis may fall several degrees, then again rising to its old height, and now defervescence occurring with crisis [precritical fall]. The fall in temperature may occur with collapse, and the crisis directly be the cause of the fatal termination; more frequently, however, death occurs at the height of the disease, most commonly on the twelfth day.

Complications.—Severer morbid phenomena than those described which are in keeping with a middle severe course of typhus fever, so-called "*complications*," are not infrequent; they are to be looked upon as the result of a more powerful action of the infective principle, and partly also as the effects of severe prostration and inanition. They make the prognosis more unfavourable and increase the mortality. The most important complications are: *Pneumonia*, catarrhal as well as fibrinous, pulmonary gangrene, pleurisy; further, *laryngitis*, *pharyngitis with ulcer formation* and diphtheritic deposits. Relatively frequently have been noticed *furuncles*, *catarrh of the middle ear* and, especially, unilateral and bilateral *parotitis*. Gastric and intestinal catarrh, now and then with enlargement of Peyer's patches and of the mesen-

teric glands, are shown clinically by dyspepsia, jaundice and diarrhœa, which, if they are noted at all, occur in the course of the second week. Hæmatemesis has been observed in some few cases, its anatomical substratum being partly tears of the mucous membrane (Virchow), partly punctiform hæmorrhages of the mucosa of the stomach. On the part of the nervous system there occur as complications: Purulent *meningitis*, cerebral embolism with hemiplegia, neuritis; but, in general, in spite of the severe disturbance of the nervous system, very few changes are found anatomically. Also, as it appears, in spite of the frequency of albuminuria, no severe inflammations of the renal structure are noted. On account of the great prostration of the patient and owing to the marked cardiac asthenia which is noted in the pathological picture, the occurrence of gangrene of the extremities and *bedsore* is not to be wondered at. Besides, the symptoms which take place may be noted even in convalescence: Sopor, loss of memory, intestinal catarrh, etc., also bronchitis, which occasionally may be the starting-point of a tubercular infection.

Differential Diagnosis.—The direct diagnosis of typhus fever depends in the main upon the fact that at the time in which individual patients are observed an epidemic is prevalent. Under such circumstances, if the above-described symptom-complex be noted, typhus fever cannot be mistaken for any other affection. The condition is altered, however, if the first cases of an endemic are observed, or if sporadic cases come under observation! Here typhus fever may be confused with other infectious diseases, especially *measles* and *enteric fever*. From the latter typhus fever may be differentiated partly by the eruption, partly by the condition of the pulse, the fever type, and by individual symptoms which are much more characteristic of typhus than of typhoid fever. The eruption in enteric fever, as a rule, is much more sparse and upon the average appears about a half a week later (as a rule only in the middle of the second week) than in typhus fever. Deviations from this usual condition occur, i. e., the eruption in typhus fever may be less markedly developed, and, *vice versa*, in the case of enteric fever it may be unusually copious and appear earlier. The development of the eruption may be so abundant in enteric fever that even the extremities may appear as if covered by them. I have frequently seen such cases, especially during the Franco-Prussian war. Nevertheless, these are great exceptions; but it is equally rare that the roseolar eruption in enteric fever should become petechial, as is a rule in typhus fever. Of more importance for the differential diagnosis is the character of the pulse and the course of the fever. The pulse in enteric fever, according to my observations, in comparison with the height of the temperature, is relatively slow, i. e., instead of a pulse of 120 or thereabouts with a temperature of 104.5° F. or 105° F., it is quite usual that the pulse in enteric fever should be about 100 or less, despite the fact that the temperature is 104° F. or 105° F. I regard this condition of the pulse to be of greater diagnostic importance in enteric fever than the frequently mentioned dicrotism which occasionally occurs in the case of typhus fever as well as in other febrile diseases. In typhus fever the pulse is especially characterized by its considerable, and occasionally its disproportionately great, frequency. Further, whereas in enteric fever the temperature rises step-like and requires four to five days to reach its acme, at which it persists for weeks, the rise of temperature is rapid, in from one to two days in the case of typhus fever; the deferves-

cence is also noteworthy. In the case of enteric fever, during a week longer of remissions and intermissions, the temperature falls, whereas the case of typhus fever the defervescence is critical. (Compare Figs. and 78.) Common to both affections are bronchial phenomena; in the ca of typhus fever, however, there are also coryza and conjunctivitis, sym toms which are almost exclusively absent in the case of enteric fever. great diagnostic value should be placed upon the earlier alteration of t cerebral condition, and upon the less frequent appearance of diarrhœa typhus fever compared with typhoid fever; diarrhœa may also occur typhus fever and be very pronounced, whereas, on the other hand, to whi fact I can testify from a rich experience, it may be absent for weeks in e teric fever, in spite of the severest course. A most important differenti point is, finally, the Gruber-Widal reaction, which is almost exclusive absent in the case of typhus fever.

Measles.—To differentiate typhus fever from *measles*, which have common the appearance of the copious eruption, the affections of the m cous membrane of the upper portions of the respiratory tract and of i adnexa, as well as the sudden rise and fall of the temperature, the pr dromal fall in temperature in measles should be particularly noted, whic is absent in typhus fever and also the early disappearance of the feve after the exanthem of measles has reached its height (early, from the fift to the sixth day). A sudden change by no means occurs in typhus fev after the appearance of the exanthem, but, now, for a long time, about week, the severest period of the disease, the real status typhosus, manifest itself. The prevalence of the eruption in the face in measles is valueless i a diagnostic respect. It is of more importance for the practitioner to re member that measles is especially a disease of children, whereas typhu fever in children under five years of age belongs to the greatest curiositie of medicine.

It would be of fundamental importance for the diagnosis if it were possibl to discover the specific virus of typhus fever. Up till now, a distinct micro-organism as the cause of typhus fever has by no means been generally accepted (in spite of th positive proof of bacilli in the blood by Moreau and Cortez, Hlawa and others, an of capsulated small diplococci in the blood and sputum of typhus patients found b Dubief and Brühl).

It is therefore a much more satisfactory condition and of decisive importanc for the diagnosis that our knowledge of the origin and nature of the micro-organism giving rise to relapsing fever, which will now be discussed, is known.

RELAPSING FEVER, "RECURRENS"—TYPHUS RECURRENS

Relapsing fever is an infectious disease characterized by *febrile attack with intervening afebrile periods,* being undoubtedly *contagious.* The *con tagious principle* is found in the *blood* of the patients; this is proven wit great certainty by the positive results of inoculations which have been mad with the blood of relapsing-fever patients upon monkeys (lower animals ar not susceptible to relapsing fever). Yes, Mogzutkowsky and others hav proven this even in man. The presence of peculiar *spirilli* in the blood o

relapsing-fever patients distinguishes it from all other blood; they are never absent in the blood and have up till now not been found in any other affection.

It is therefore more than likely that these spirilli, which were detected by Obermeier in 1873, are the actual cause of relapsing fever, especially as the inoculation of blood containing the spirilli into other organisms has caused relapsing fever to appear and an enormous increase of the spirilli in the inoculated blood. However, *one* condition for the faultless proof that these spirilli are in fact the cause of relapsing fever could as yet only be complied with in an unsatisfactory manner, namely, to cultivate these spirilli outside of the body and to inoculate pure cultures into the animal organism. We are not absolutely certain at present how the spirilli find entrance into the organism and there show their pathogenic action. However, the material which has been gathered by investigation in reference to the distribution of the spirilli in the body of the relapsing fever patient and their relations to the various stages of the disease, etc., is so great that we are entitled to draw some deductions from the life changes of the spirilli in the diseased body.

[Tictin found the spirilli in bedbugs and reproduced the disease in monkeys by injecting blood sucked by a bug from an infected monkey. It is thus possible that bedbugs are the intermediate host of the spirilli.]

The spirilli of relapsing fever are very delicate, not articulated, wavy, long strands (about seven times as long as a red blood corpuscle), showing windings similar to a corkscrew; they are actively motile and glide between the blood cells. *The spirilli are only found in the blood and especially between the blood corpuscles, never in them or inclosed by them.* They can be readily demonstrated in the blood on microscopic examination by the use of the immersion lense, more plainly in cover-glass preparations which have been treated with watery aniline colour, best with fuchsin.

Of special importance is the fact that the spirilli of relapsing fever, despite the great number of examinations that have been made, *have only been found in the blood of patients during the period of fever,* and *were constantly absent in the afebrile periods.* It must, however, be remarked that their presence in the blood only in general conforms to this law, i. e., that they may appear a short time before or after the onset of the fever, and also may disappear from the blood a short period before the fever has ceased, or, again, they may persist in the blood at the very first onset of the afebrile stage.

Formation and Death of the Spirilli in the Various Stages of the Disease.—*Why the spirilli, which are present in such large quantities during the fever period, disappear so rapidly and without leaving a trace during the crisis* (by crisis produced artificially they do not disappear), to recur in the blood with the onset of the fever, is very difficult of explanation. According to the investigation of Metschnikoff it has become likely that the spleen plays an important part in this process. Whereas the spirilli, during the height of the fever, are only found in the blood, i. e., in the vascular system, but not in the secretions or in the tissues of the body, according to Metschnikoff's findings, they gather in the spleen before and after the crisis; here they are destroyed, as it seems, by chemical protective material which is secreted by the cells of the spleen, and thus they perish. If all of them are not destroyed, a new attack occurs, probably in the manner that the spirilli which survived, multiply, again to invade the blood in great quantities after a certain time, thus producing a second attack.

As the spirilli of Obermeyer, on the one hand, are constantly found in the blood of relapsing-fever patients during the febrile period, and, on the

other hand, are never found in any other affection, it is obvious what an enormous diagnostic importance is due to these structures. They have the same importance in the diagnosis of relapsing fever which tubercle bacilli have in tuberculosis. It is evident that the most exact observation of the clinical phenomena of relapsing fever cannot be dispensed with by the diagnostician. The clinical symptoms give the impulse for the examination of the blood for the spirilli, and even in finding the spirilli nothing is known of the course of the individual case and the changes in the different organs and of their functions in the course of the disease!

Course of the Affection.—It has been proven experimentally that the blood at the acme of the fever is directly contagious; but this method of contagion will scarcely ever be noted in practice. Contagion is more likely to occur from person to person by contact, by the exhalations of the patients, by fomites and by a third person that may have been the carrier of the disease without having been affected by it. After a *period of incubation* (and *prodromal period*) which runs its course without symptoms or, at the most, with very insignificant morbid phenomena (lassitude, anorexia, etc.), and which upon the whole is from five to seven days in duration, the affection begins *suddenly* with marked fever, in the majority of cases being ushered in by a chill. At the same time there occur headache, vertigo, tinnitus aurium, vomiting, mild delirium, sensations of great fatigue, pains in the back and loins, and, above all, drawing, tearing *pains in the muscles*, especially in the calves which are also sensitive to pressure. While the headache diminishes during the first days, the general muscular pains continue and induce the patient to lie quietly. Soon pressure and tension in the epigastrium, especially frequent in the left hypochondrium, take place, possibly due to the enlargement of the spleen. The latter may be very markedly enlarged and is then easily palpable—I have never seen the spleen so large in any other infectious disease as in a case of relapsing fever; the liver is also enlarged and painful, and slight jaundice may usually be noticed.

That we are dealing with a severe infectious disease, is not to be questioned under such circumstances. Which disease we are confronting, however, cannot be determined without further observation, but the chill and the facts that the hot skin may early show perspiration, that the tongue is thickly coated, that the frequency of the pulse is 120 and more, make us reject the diagnosis of a possible enteric fever and lead us in another direction; the tearing muscular pains, especially if an epidemic be present at the time, invite us at once to consider relapsing fever. From the fourth day on, at which time the characteristic eruption has become noticeable in the acute exanthemata and also in typhus fever (in this case usually a day or two later), the circle of the infectious diseases which are to be considered in a diagnostic respect becomes considerably smaller if no exanthem appears in the case in question; now the examination of the blood can no longer be delayed, if it has not been taken earlier for the purposes of examination. With the demonstration of spirilli in the blood every doubt disappears—the diagnosis of relapsing fever then becomes the easiest and most certain in the entire realm of acute infectious diseases. The fever curve

does not show anything characteristic during the first five to seven days; a continuous type of fever is present, ranging from 104° F. to 106° F., with slight or marked remissions (up to three or four degrees), similar to what is noted in all other acute infectious diseases.

About the sixth day, however, the picture suddenly changes, after occasionally a precritical rise has occurred (eventually with repeated chill and vomiting): The high temperature falls abruptly from 104° F. or 106° F. in the course of from six to twelve hours to normal or subnormal ranges, temperatures of 95° and less have been seen, and the drop in temperature may amount to seven, nine, or twelve degrees! *In general, such an abrupt*

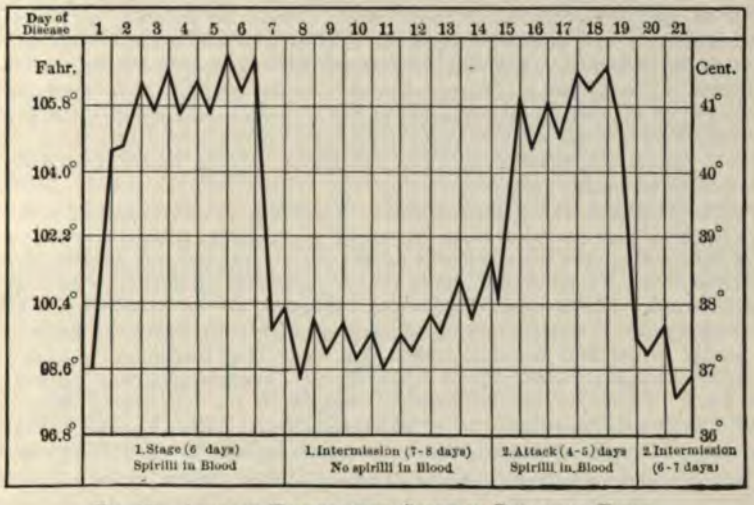


FIG. 77.—AVERAGE TEMPERATURE CURVE IN RELAPSING FEVER.

defervescence is not noted in any other infectious disease as in the case of relapsing fever; it is, therefore, somewhat pathognomonic of relapsing fever, especially if it be considered that the preceding fever, not as in the case of malaria, was of short duration, but has existed almost continuously for a week. With the defervescence marked sweating occurs, the frequency of the pulse suddenly lessens, the spleen becomes smaller, *the blood free from spirilli,* the subjective phenomena disappear rapidly, except the pains in the extremities which may continue for some time; the patient appears to be convalescent and in fact is so in a small proportion of the cases. As a rule, however, usually after seven days, another attack of fever occurs, resembling the primary one, accompanied with chill, headache, etc., after a slight increase in temperature and in pulse frequency usually had announced the appearance of a new attack towards the end of the period of intermission. This represents an exact reproduction of the first attack; the temperature may eventually even be higher than during the first stage, whereas the duration of the second attack is apt to be shorter (about five days). The second attack also disappears with copious sweating and a critical fall of temperature. Then a third, a fourth, and in rare cases

even a fifth, attack may occur, after an afebrile period which may last for several days has regularly been present between the attacks. The later attacks of fever are in general briefer than the first one; the afebrile periods of intermission are prolonged inversely with the frequency of the attacks.

Number and Duration of the Attacks.—It must be especially remarked in regard to the conditions of frequency and time of the individual attacks that, according to the observations of various investigators, the following has been determined: In about one fifth of the cases the disease showed *one* attack, in not quite one half of the cases (about 40 per cent) two, in one third of the cases, three, and in about 5 per cent four attacks were noted; only in about 1 per cent of the cases a fifth attack occurred. The duration of the attacks and the periods of intermission varied greatly in different epidemics and in different cases. Thus the *duration of the attacks* varied from one to fourteen days; upon the average, the first attack lasted six days, and the second four and a half days, the third three days, and the fourth one and a half days. The *period of intermission* also varied greatly, from three days to three weeks; on an average the duration of the afebrile period between the first and second attacks was from seven to eight days, between the second and third attacks, from six to seven days, between the third and fourth attacks, finally, frequently even from ten to eleven days.

Special Symptoms and Complications.—Of special symptoms and complications which are particularly prominent in the course of relapsing fever something further must be said. Although the *symptoms on the part of the nervous system*, above all the headache and pains in the joints which especially irritate the patient, and although even actual neuralgias may occur, the brain, on the whole, is less affected by the action of the infection than in enteric fever or typhus fever; the patients complain mostly of vertigo; delirium occurs, but it is very transitory as a rule. In some cases meningitis, especially pachymeningitis hæmorrhagica, was present as a complication. There are but insignificant changes in *the respiratory organs*, only bronchitis is a usual symptom. Of severe complications which may be the cause of death, catarrhal and fibrinous pneumonia must be mentioned. Less constantly affected is the digestive tract, if slight gastric and intestinal catarrhs are excluded. The tongue is markedly coated from the onset; it rarely becomes dry and fissured during the attack. As has already been remarked, the *liver* is constantly enlarged and painful, and slight jaundice is almost always present. Under certain circumstances these conditions may reach a more marked grade, in which case the affection takes on a different, more severe type to which reference will be had later on ("*bilious typhoid*"). The *spleen*, as has already been mentioned, is constantly and usually markedly enlarged; swelling during the course of the attack, it declines in the period of intermission, to enlarge again with a new attack. This increase in size of the spleen is probably due to the periodic development of a plentiful supply of leucocytes in the organ which is probably in relation with the leucocytosis which different authors have observed in the course of relapsing fever. Infarcts arising on account of venous thrombosis may excessively increase the size of the spleen. *Rupture of the spleen* in a comparatively large number of cases has occurred, either abruptly or as a result of previous softening and purulent condition of the infarct. Albumin and hyaline casts are commonly found in the *urine* during the periods of fever as the manifestation of an irritation of the kidneys (in keeping with the granular discoloration of the epithelia of the convoluted uriniferous tubules and the changes in the endothelium of the capsules which have been noted post mortem); but a more marked irritation of the kidneys with well-developed symptoms of nephritis and severer diseases of the kidneys are rare. If, in the latter case, blood occurs in the urine, spirilli are also noted to be present in the urine, whereas the urine, under other circumstances, is constantly *free from spirilli*. Other changes of the urine (diminution of chlorides, etc.) are of no diagnostic importance. The *skin* shows no constant characteristic changes; of course, roseola, herpes, erythema, petechia or œdema were noted now and then—but they are all of minor significance and

by no means of regular occurrence. Rare complications are: Iridochorioiditis, endocarditis, inflammation of the joints, gangrene of the extremities, etc.

Bilious Typhoid.—More important than the development of the previously mentioned complications is the fact that, in some epidemics and in some individual cases of relapsing fever, the disease assumes a malignant type with the picture of severe jaundice, to which the name of "*bilious typhoid*" has been given. The identity of both affections, which has been made probable by Griesinger, the classical interpreter of the disease, has been ascertained by the presence of spirilli in the blood upon numerous occasions, and unquestionably proven by Mogzutkowsky, who inoculated the blood of a patient with bilious typhoid into a healthy person and in the latter produced simple relapsing fever. The course of the affection, according to Griesinger, is as follows: Onset with chill, severe headache, and pains in the extremity, gastric pains and vomiting; great lassitude, apathy and cerebral involvement are present from the beginning. The tongue becomes dry and fissured, diarrhœic stools, containing bile pigments, set in; simultaneously there is considerable enlargement of the spleen and liver, both organs being sensitive to pressure. From the fourth to the sixth day *jaundice* occurs; the pulse which has been frequent up to now, becomes slower, the spleen constantly increases in size. In some of the cases death now occurs, or rapid amelioration with apparent convalescence, then a relapse with all the earlier symptoms, but which now increase and often rapidly terminate in death. In other cases, after the jaundice has appeared, a severe typhoid condition develops, with prostration and stupor, quiet or loud delirium, frequent pulse, dysenteric or diarrhœic discharges which may be of pure blood, difficulty in deglutition due to diphtheritic angina, bronchitis, pneumonia, petechiæ and miliaria upon the skin. Now, with the symptoms of collapse and in convulsions, *death* occurs, or *recovery* may take place *rapidly* within two days or (especially if pneumonia and dysenteric intestinal affections, etc., exist) *gradually*, with the cessation of all the symptoms and a slow decline in the size of the spleen. Convalescence in Griesinger's cases was relatively rapid and comparatively mild; in other cases it is protracted, and combined with severe sequelæ.

The duration of these severe cases of relapsing fever is from five to fourteen days, the mortality is much greater than in simple relapsing fever; whereas here it always shows a very low percentage, it rises in the cases of bilious typhoid to from 60 to 70 per cent. The localization of the relapsing-fever process in the latter is more distributed and severer. Complications, which are entirely foreign to simple relapsing fever, although they do not occur in bilious typhoid, are all more marked and of a more pernicious nature in the latter, in keeping with the severe changes of the organs which have been found post mortem. Thus we note hæmorrhagic and diphtheritic inflammations of the mucous membranes and serous coverings, multiple formations of abscess in the spleen, the liver, the kidney and the brain, as well as parenchymatous inflammations of the liver, of the heart, etc.

The characteristic spirilli are *always* found in the blood of patients

with bilious typhoid: The identity of this affection with relapsing fever, according to this and to the positive, already mentioned results of inoculation, is unquestionable. The name "bilious typhoid" should, therefore, be definitely done away with, and the designation "*septic-bilious relapsing fever*" substituted. Diseases which show a similarity to the septic-bilious relapsing-fever variety, in which, however, the proof of the presence of spirilli is not possible, must on principle be rejected from the category of "bilious typhoid."

It is not necessary to enter into a special differential diagnosis of relapsing fever, for the above-stated reasons. After what has been mentioned of diagnostically important symptoms, above all of the presence of spirilli in the blood of patients in certain stages of the affection, the diagnosis of this disease can be made with more certainty than that of any other acute infectious disease. Usually *one* microscopic examination, i. e., the proof of the characteristic spirilli which do not occur in any other disease, is sufficient to dispel all diagnostic doubt.

ENTERIC FEVER—TYPHOID FEVER—TYPHUS ABDOMINALIS

The diagnosis of enteric fever, in the majority of cases, can be made without difficulty from the complex of the clinical phenomena; but in a small number of cases this may only be done by *exclusion*. The diagnosis is materially assisted by the proof of *typhoid bacilli* in the blood and in some of the excreta of the patient, and of the presence of an agglutinating action of the serum upon typhoid cultures (Gruber-Widal reaction, which see); further, by a careful noting of the *ætiologic* conditions of the individual case. We shall, therefore, first briefly discuss the pathogenesis of enteric fever in so far as it is of value in the diagnosis.

Typhoid Bacilli.—The *typhoid bacilli* which were discovered by Eberth, in 1880, and which were first obtained in pure cultures by Gaffky, are generally admitted to-day to be the cause of typhoid fever. They represent small, feeble staffs with very markedly developed movements (due to numerous flagella) which may be coloured intensely (but *not* according to Gram) and which may be cultivated upon gelatine and especially upon potatoes. Although the dependence of enteric fever may be unquestionably looked upon as due to these bacilli, it has been more and more developed lately that the absolute proof of these micro-organisms is by no means easy, and that a confusion with other typhoid-like bacilli, especially with those belonging to the group of the bacterium coli commune, is very difficult to avoid. The *practical* application of the determination of the typhoid bacilli in the diagnosis is extremely limited up to this time. The typhoid bacilli, as is well known, colonize with preference in the lymphatic organs: In the mesenteric glands, in the spleen and especially in the solitary follicles and in Peyer's patches of the intestine, in which they produce inflammation and necrosis of the tissue. The circumstance that the typhoid bacilli are distributed in the body in a metastatic-infectious manner is the cause that they are also found in other portions of the body, especially in the kidneys and upon the skin (the eruption). They are also met with in the secretions, the urine, the sputum and perhaps also in the sweat. More important as carriers of the bacilli are the *fæces*; however, the proof of the bacilli in the excrements can rarely be obtained and usually only in the course of the second week. This is due to the fact that the typhoid intestinal infiltrations which constantly and plentifully contain typhoid bacilli, begin to form crusts at about this time. The finding of the bacilli

in the blood of the eruption is likewise only possible during the course of the second week, i. e., during the time the eruption begins to appear. It is clear, therefore, that, especially during the first week and a half in which the diagnosis is mostly in the balance, we cannot obtain much aid from bacteriological investigations. We are more certain to arrive at a result in trying to obtain cultures from the urine; most certain to do so by the examination by culture of the blood taken from the spleen; but what practitioner would regularly use such methods for purely diagnostic purposes?

Distribution by Means of the Fæces.—*It may be stated as unquestionable that the infective principle of the typhoid poison is contained in the urine and especially in the fæces of enteric-fever patients.* In favour of this is not only the positive result of the examination of the dejecta for typhoid-fever bacilli, but an enormous array of professional observation which shows that the clothing, bed-pan, closets, etc., which have been soiled by enteric-fever dejecta, have spread the infection. Especially is this the case by drinking-water and milk. In this regard there is a great number of absolutely certain examples which, according to my opinion, absolutely prove that the affection has only occurred in persons who have obtained their drinking-water from the same water-supply or their milk-supply from the same dairy, in which cases later a communication of the water-supply could be shown with privies into which the dejecta of enteric-fever patients were thrown. This has been positively determined. The possibility of a propagation of the germs of enteric fever by means of drinking-water has also been proved from an experimento-bacteriologic standpoint, after it has been demonstrated that the possibility of life of the typhoid bacilli in water may last many weeks.

On the other hand, enteric fever is not contagious from person to person, i. e., individuals who are exposed solely to the exhalations of enteric-fever patients, or who are in close association with the patients without coming in contact with their dejecta or secretions, run no danger of being attacked by enteric fever. I depend upon my own observation of over two and a half decades—but in *one* of my cases could I establish *direct* contagion. This occurred in a female who, being paralyzed, remained for months in a ward in which also enteric-fever patients were present. Whereas never before or afterward a single case of enteric fever was noted the origin of which could be referred to the presence of the affected person in this ward of the hospital or in the hospital itself, the patient previously mentioned one day was attacked by an unquestioned, mild case of enteric fever. As was afterward shown in the case of this patient, *accidentally the same thermometer was used to take the temperature per rectum with which, a short time previously, the enteric-fever patients who were lying in this ward, also had their temperatures taken!*

Course of the Infection.—If the bacilli find their way into the digestive tract of a healthy person, the course of the infection is very probably as follows: In the stomach there is no soil which is favourable to their development; they are, therefore, rapidly destroyed here, which may be assumed according to investigations which have been especially directed to this point, provided the gastric juice contains the normal amount of hydrochloric acid. If this is not the case or if the typhoid bacilli reach the stomach during the time in which no acid is secreted, they leave the stomach in a condition capable of germinating, and find entrance into the bowel of the person, in which, under conditions favouring their retention, they lodge (*individual predisposition*), find their way into the lymph glands, from there into the blood, and thus into the spleen, liver, kidneys, and marrow of the bone. A part of the bacilli are later, thoroughly capable of existence, cast off in the dejecta, and these bacilli which are discharged in the fæces, urine, etc., form the nidus for new infections. As we have seen, this may occur without difficulty by means of drinking-water and food material, and thus the explanation is clear. In favour of this view is not only a large amount of clinical observations, but also the circumstance that the principal field of the pathogenic activity of the bacilli is in the intestinal canal, in which arises primarily an enlargement of the solitary follicles and of Peyer's patches, with medullary (cellular) infiltration, which latter may either diminish with fatty degeneration by absorption or, much more frequently, lead to scar formation (second week of the disease) and development of necrosis (third week).

The question regarding the details of the dissemination of typhoid fever is by no means so thoroughly plain in all instances as in the case mentioned from my own observation of direct transmission of the bacilli into the intestine of the healthy individual or as in cases of an unquestioned importation of the bacilli with drinking-water, etc. On the contrary, we must admit, if we desire to state the case impartially, that the method by which the transmission to the healthy individual has taken place, can frequently not be proved. It is *possible* that, with the fæces of typhoid patients which have reached the soil of the earth (even if the supply of oxygen be entirely cut off, although to a less extent than if oxygen were present, it is possible for the bacilli to flourish), typhoid bacilli may continue to develop here. And this may be facilitated in different regions and at different times (predisposition, regarding *place* and *time*—maximum of the frequency of enteric fever in the autumn and during the first months of winter). However, this is but a *possibility*, as the strict proof of the further development and multiplication of the typhoid bacilli in the earth has as yet not been furnished. On the other hand, it must be admitted that typhoid bacilli in the ground may remain capable of development for a long time—up to half a year—and may withstand external influences such as drying and cold (up to — 10° C.), whereas higher grades of heat (75° C.) and direct sunlight destroy them after a brief period. There is, therefore, no doubt that the enteric-fever poison may remain in the earth for a long time without losing its faculty of growth, and from here may get into ground-water or well-water retaining all its faculties, or may be deposited upon food material and in this way be imported into the human organism. Contagion by means of exhalations coming from the ground, by the "ground air," is impossible. Even if it has been proved that typhoid bacilli may be carried by dust and thus reach the human body, this method of transportation must in reality be looked upon as very rare; and the possibility of flying of the bacilli or an inhalation of these, as a flying poison, is not worth consideration. Pettenkofer's proof that the *frequency of enteric fever is in a reciprocal relation with the condition of the ground-water, so that the low condition of the ground-water involves a growth of enteric-fever cases,* remains under all circumstances an indisputable, important fact, which is undoubtedly correct for some, if not for all, epidemics of enteric fever. This is by no means in contradiction to the bacteriological standpoint, but, according to above explanations, may be combined with it without the slightest difficulty.

The duration of the time from the entrance of the typhoid poison until the phenomena of the disease appear is, on an average, two to three weeks. This period of incubation runs its course without symptoms provided those occurring in the prodromal period are not included.

Prodromes.—By no means in all of the cases is it possible, by carefully considering the ætiological conditions, to gain points of importance for the diagnosis of enteric fever. We are usually compelled carefully to weigh and to analyze the entire *symptom-complex.* As a rule, before the onset of the pronounced febrile period, a so-called *"prodromal stage"* occurs which varies greatly in individual cases, lasting hours, days, yes, even weeks, and which is characterized by general malaise, feebleness, pains in the extremities, by anorexia, vertigo, headache, disturbed sleep, tinnitus aurium, and mild rises of temperature; rarely also by abdominal symptoms (abdominal pains and diarrhœa). The inconstant occurrence and the vague nature of these symptoms would make it appear more proper to forego their separation from the incubation period; the establishment of an individual prodromal stage has absolutely no value in a diagnostic respect.

Course of the Fever.—Enteric fever begins, as far as the diagnostician is concerned, with the occurrence of the *fever,* which rarely sets in with a single chill, repeated chilliness being usually noted. The course of the

fever in enteric fever has been determined by thousands of individual observations and by innumerable measurings by means of the thermometer. In the first half of the first week, often even a few days longer, a gradual, *step-like rise of the curve occurs,* so that the temperature in the evening is about a degree and a half to two degrees higher than that of the preceding evening, and about a degree more than upon the following morning. (See temperature chart, Fig. 78.)

After the temperature has reached its acme of 103° F. to 105° F., in from three to five days, the fever shows a *continuous type,* i. e., the temperature remains at about 104° F., with slight morning remissions (cor-

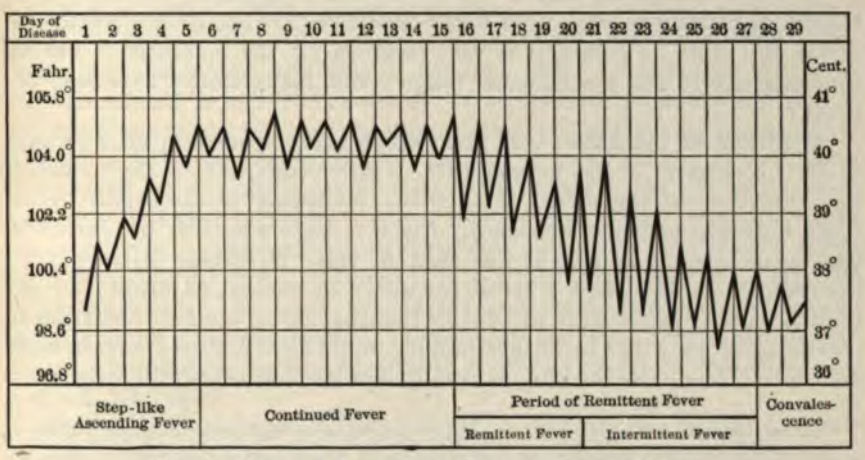


FIG. 78.—AVERAGE TEMPERATURE CURVE IN ENTERIC FEVER.

responding about to the morning remissions in the normal temperature curve). In the course of the third week usually more marked morning remissions occur, which may be looked upon as the first signs of improvement in the case. The morning temperatures in this *remittent stage of the fever* may be between 100° F. and 103° F.; the evening temperatures, however, are always higher. Gradually, in the course of the fourth week, the morning temperature reaches to 100° F. or below, whereas the evening temperatures still reach high grades. It is of importance to the physician to understand this *stage of marked, intermittent temperature curves* in the course of the fever, so that he does not erroneously consider the high evening temperatures a severe sign of a new exacerbation of the disease. In that, now, the evening temperature gradually becomes lower day by day, in the fifth week a time occurs in which the evening temperature no longer rises above 99° F. or 100° F. and gradually reaches normal. The first day in the course of the disease in which this is the case may be looked upon as the commencement of convalescence. The temperature curve as sketched (see Fig. 78) is in keeping with the course of the temperature in a medium severe case of enteric fever. It is obvious that slight deviations may occur at the bedside; upon the whole, however, the temperature shows this course with conspicuous regularity, and *marked deviations* either a[1]

or below (for example, an unusual fall of the evening temperature below the morning temperature in the stage of the intermittent or remittent course) *are by no means to be looked upon as irrelevant circumstances,* but usually as the expression of complications to which we shall refer later on. In a diagnostic respect regular notes of the temperature are to be taken (at least three times every day), and an exact observation of the temperature course in typhoid is absolutely of the greatest importance, yes, even of fundamental importance for the physician, as the prognosis as well as the necessity to adopt therapeutic measures are often materially determined by the course of the fever curve.

[It should be noted that the course of the temperature during convalescence is extremely labile, that slight conditions are apt to produce fluctuations and rises. Further, it is very common for the temperature during convalescence to run a subnormal range which may amount to several degrees.]

Condition of the Pulse.—Of great diagnostic importance, next perhaps to the observation of the temperature, is the recognition of the condition and frequency of the *pulse.* This shows, according to the height of the fever, a greater or lesser frequency. *But it is important that the pulse frequency in typhoid in toto is less than in other infectious diseases,* i. e., whereas we would expect a pulse frequency in general of about 120 per minute with a temperature of 104° or thereabouts, in similar conditions in enteric fever only 110 to 90 per minute would be noted, yes, occasionally even less! The pulse, besides, is usually dicrotic and extraordinarily weak.

Dicrotism.—The reason of this peculiar condition of the pulse frequency may be looked for in that the increase of the pulse, as is usual in fever so also in typhoid, depends directly upon the height of the body temperature, but that, besides, it should be noted that the typhoid intoxication shows an inhibitory action upon the frequency of the pulse in this disease. A weakened condition of the arterial wall due to paresis of the vaso-constrictors should also be considered as a result of the infection, producing the softness and dicrotism of the pulse in enteric fever, especially during the second and third weeks. *Dicrotism* has not nearly the diagnostic importance which is usually given to it, as it occurs in many other afebrile infectious diseases. Besides, I regard the diagnosis of dicrotism by mere palpation of the radial artery (without the use of the sphygmograph) as of slight importance; if the results of the feeling of the pulse are frequently compared with the sphygmographic examinations, we will be astonished how rarely the expected "slight dicrotism" appears in the sphygmogram.

Relative Slowing of the Pulse.—Of much greater importance than eventual dicrotism is the previously mentioned *relative slowing of the pulse frequency.* In no other infectious disease is this incongruity between pulse beat and temperature curve so markedly developed and so frequently pronounced as in the case of enteric fever. [Except in the case of yellow fever.]

As a rule, the pulse is found, with temperatures of 101° F. to 104° F., amounting to 80 to 100 per minute, in contrast to other febrile diseases in which, if the temperature be 104° F., the pulse, as a rule, will amount to 120 beats per minute. I may state from a rich experience that, in cases which were very difficult to diagnosticate, the observation prolonged through the course of several days of the relative slowness of the pulse has more than once assisted me in the diagnosis and later has usually proved itself as a correct inference. It must be remarked, however, that this incongruency of height of temperature and pulse frequency only occurs in mild cases during the entire course of the disease, whereas in severe cases it may also be noted

during the first stages, but later, as cardiac asthenia develops more and more, it gives place to greater pulse frequency, yes, the rapidity of the pulse may even become excessive. This, however, does not detract anything from the importance of the symptom in diagnosis, as in the majority of cases the question of differential diagnosis only produces difficulties in the first stages of the disease.

First Week of the Disease.—Combined with the fever and the just-described changes of the pulse are the other signs of a febrile affection: Lassitude, headache, restless sleep, hot skin, rarely perspiration (well-developed perspiration almost always is *against* the presence of enteric fever, although in some few exceptional instances I have seen typhoid-fever patients almost bathed in sweat); further, anorexia, thirst and a diminished excretion of urine. With this the symptoms of the typhoid infection are mingled: Marked headache, vertigo, mild *delirium, tinnitus aurium*; relatively frequent is also *epistaxis* in the first week. The tongue is coated, but this coating is soon lost in certain areas, and upon the margins and at the point, extending more or less towards the back of the tongue, the organ becomes red; as the tongue is protruded, it is noted that it is tremulous. Frequently there is early *constipation*; only towards the end of the first week does diarrhœa occur, as a rule. The belly becomes tympanitic, painful upon pressure, especially in the ileocæcal region, and gurgling is noted upon palpation (ileocæcal murmur)—a symptom absolutely without value in the diagnosis. The *spleen* is enlarged even in the first week, which can be determined upon palpation. Percussion of the lungs shows normal conditions, auscultation reveals the signs of a mild or moderate bronchitis with medium-sized râles.

Second Week.—In the *second week*, after the fever has reached its acme and the continued type of the fever curve is noted, the disease develops to its complete height, to the so-called *status typhosus*: Stupor, somnolence, difficulty of hearing, difficulty and indistinctness of speech. The patient is apathetic in a sort of semi-comatose condition, frequently murmuring to himself, picking at the bedclothes, showing involuntary contraction of the muscles (subsultus tendinum), and he is in a generally stupid condition ("typhus stupidus"); in other, rarer cases the patient is subject to illusions and hallucinations, loud and lively delirium ("typhus versatilis") occurs, he jumps up, yes, even maniacal attacks may take place. I was compelled, a couple of years ago, to put a patient into the strait-jacket! Involuntary evacuations of urine and fæces occur, the tongue is dry, red, fissured and fuliginous, is protruded with difficulty, trembles, and often remains out, the patient forgetting to draw it into his mouth again; the belly is tympanitic. Usually, now, there are well-developed *diarrhœas*, the thin stools *resembling pea-soup*, i. e., light yellow, separating in two layers, an upper, fluid, and a lower, more consistent, mass. The urine, as a rule, contains albumin, the spleen is distinctly enlarged. At the beginning of the second week, usually *about the tenth day*, the *eruption* occurs in the upper abdominal region, a diagnostically very important symptom. The bronchitis is more marked, the fine râles are concentrated to the lower posterior portions of the lungs, and here, not infrequently even now, a unilateral or bilateral mild dulness may be noted upon percussion.

Third Week.—The *third week,* in the severe cases, shows the acme of the asthenia and of the stupor; this is the period of the severe complications: hæmorrhages and perforations of the bowels, hypostatic pneumonias, parotitis, and bedsores, etc. In mild cases a change for the better occurs in this week, in that the continuous type of fever becomes remittent and later intermittent, and the marked symptoms of the severe infection commence to abate. As a rule, the roseola ceases with the end of the third week.

In the *fourth week,* finally, the symptoms of the disease become gradually less and less marked. The apathy disappears, there is control of urine and fæces; the tongue becomes moist, diarrhœa stops; the eruption which has last appeared becomes pale, the spleen smaller. The pulse becomes stronger and slower; not only the morning temperature, but also that of the evening no longer rises beyond 100° F.—convalescence sets in, which may, however, be interrupted by various sequelæ or by relapses.

Among the symptoms of enteric fever which have been thus briefly sketched, there are of great diagnostic importance, besides the fever and the condition of the pulse: The *eruption,* the *enlargement of the spleen* and the *appearance of the tongue*; of less diagnostic importance are diarrhœa, the gurgling in the right iliac fossa, the nervous phenomena, the bronchitis, and the condition of the urine. We shall have to consider the symptoms mentioned individually and somewhat more in detail.

The Roseolar Exanthem.—The *eruption of enteric fever* is absent in about one quarter of the cases, at the most; it is scarcely different from other varieties of roseola, showing small, round, slightly elevated, pale-red areas which disappear upon pressure with the finger. Somewhat characteristic of the typhoid character of the eruption is only its position. In the majority of cases it only occurs, or at least at first, upon the lower parts of the chest and the upper parts of the abdomen; as a rule, there are but about twenty spots. However, in other, but rarer, cases the skin of the body and extremity appears as if covered with the eruption. It is important to note that *the eruption, as a rule, appears first at the onset of the second week, according to my experience, however, almost always only towards the middle of the second week* (*tenth day*). Therefore, we cannot expect in the first ten days that the appearance of the eruption should remove any diagnostic doubt: And, *vice versa,* the diagnosis of enteric fever should not be looked upon as unlikely during the first ten days if no eruption appears. The individual rose-red spots disappear in from three to four days, whereas new eruptions appear for a week or two longer (up to the end of the third or the beginning of the fourth week), occasionally even recurring in convalescence. Only unquestioned maculo-papular roseolar eruptions must be utilized for diagnostic purposes (not vesicles nor conical papules); but if only a few *unquestioned* rose-red spots are found in the epigastric region, this is a most important auxiliary aid in the diagnosis of enteric fever. [The eruption is also frequently noted upon the back, between the shoulder-blades. It is even observed here when it is absent upon the abdomen, rarely is the eruption seen upon the face and extremities.]

Splenic Enlargement.—*Splenic enlargement,* of less importance in the

diagnosis than the eruption, is almost always present, certainly in 90 per cent of the cases. This may be demonstrated not only by percussion, but also by palpation, provided we are careful and, without pressing, place the right hand in the left hypochondrium at the height of inspiration, while the left hand forces the lower thorax wall towards the other hand. The enlargement of the spleen cannot be noted, either upon percussion or palpation, only in the case of marked meteorism. The consistency of the organ is conspicuously soft, rarely painful; if the latter is the case, the occurrence of a splenic infarct or abscess should be thought of.

Condition of the Stools.—The *gurgling in the right iliac fossa,* the significance of which was formerly often overestimated, appears as soon as fluid intestinal contents containing gas bubbles can be palpated in the ileocæcal region; this symptom has absolutely no diagnostic value. Of more importance are *the diarrhœic stools.* Diarrhœa is absent in the course of enteric fever at most in about one third of the cases: It usually alternates with constipation or with normal stools and only exists continuously in one quarter to one third of the patients. The absence of diarrhœa should not be looked upon as an important symptom in making the diagnosis of enteric fever questionable.

According to my opinion and experience, the diarrhœa in enteric fever is a nervous-infectious phenomenon and not the direct result of the intestinal ulcers. This may be easily proved in that diarrhœa is in no relation with the intensity and extent of the ulcerative process. There are cases in which the bowel is found post mortem to be covered with ulcers and in which, nevertheless, constipation was noted up to the time of death; and, *vice versa,* other cases exist in which *intra vitam* one diarrhœic stool followed the other, but in which only a few insignificant ulcers are noted at the autopsy. It is, therefore, probably due to the sensitiveness of the nerves of the intestines whether they react to the typhoid virus by giving rise to greater peristalsis or not. The *composition of the diarrhœic stools* is also by no means pathognomonic, even if the pea-soup-like appearance be suspicious, i. e., in so far as a well-developed separation of the fluid into more compact parts (the latter consisting of small crummy portions) and thin stools rarely occurs in the other forms of diarrhœa. The reason of this condition, namely the deficient suspension of the more compact constituents of the stools in the fluid, is not quite clear. The sediment contains, besides epithelia and round cells, fat crystals and triple phosphates and, frequently, but not before the ninth day of the disease, *typhoid bacilli.*

Condition of the Tongue.—Of greater diagnostic import, according to my experience, is the *appearance of the tongue.* The tongue, which is at first sticky and moist, and uniformly coated, after a few days becomes dry and partially red. As desquamation of the coating occurs at the margins and at the point, the tongue takes on a peculiar appearance. The gray covering which lies in the middle of the back of the tongue is surrounded by flesh-coloured borders and anteriorly by a triangular red area which occupies the tip of the tongue. In the second week even this coating upon the middle of the tongue disappears; the entire tongue now becomes red, resembling a piece of raw meat, and, on account of the marked dryness, has become fissured, here and there it is covered with dry, bloody mucus—"fuliginous"; the lips are also frequently covered by sordes and are also fuliginous.

Respiratory Organs.—*Bronchial catarrh* may be noted in the most, if not in all, cases early, i. e., in the first week; in the lower posterior parts of the lungs, especially in the later stages of the disease, the râles are relatively quite numerous. The progress of the bronchitis into the last-named portions should be observed every day, as it is very likely that after a longer or shorter period hypostatic conditions and pneumonias may develop, and an early recognition of these complications may save life.

Phenomena on the Part of the Nervous System.—*Nervous symptoms* are constant in the course of enteric fever, but their intensity varies greatly in different individuals ("nervous fever"). Whereas in some individuals the psychical condition is scarcely altered, in others complete stupor develops, in again others unrest, convulsions, delirium, etc., which may remind us of the picture of a maniacal attack.

This changing condition of the nervous phenomena may be explained from the individually varying reaction on the part of the nervous system to the *fever heat* and the *typhoid infection*; for the phenomena of the nervous system, especially those of the brain, are to be directly referred to these two factors. With the lowering of the temperature by antifebrile measures, primarily by baths, the nervous phenomena frequently disappear in a marvellous manner (an enteric-fever patient treated according to modern methods in general shows a very different, much milder picture than the markedly delirious enteric-fever patients or those in complete stupor, of former times); but they do not, as a rule, disappear entirely. Even before the fever occurs in typhoid, nervous symptoms set in, pains in the limbs may be noted, etc., and even later, if for any reason whatever the high temperature, which is almost a constant phenomenon of enteric fever, remains absent for a time, marked symptoms on the part of the nervous system may still be present; I have seen a case of an afebrile enteric-fever patient with marked delirium. Evidently in these cases we have to do with brains readily influenced by irritation, which react markedly to the chemical typhoid poison (similar to the individually varying reaction to narcotics, etc.), so that it does not require the cumulative irritation of the heated fever blood and of the typhoid poison, but the latter alone is sufficient to produce the severe nervous phenomena.

Other Symptoms.—What other morbid phenomena may be noted in the course of a medium severe attack of enteric fever, is of minor diagnostic importance.

Enlargement of the Liver.—It may at least be briefly mentioned that *the liver is generally found enlarged* in the severer types of enteric fever, according to my experience, and (in keeping with the anatomical changes in such cases, with the cloudy swelling or the advanced fatty degeneration of the organ) it may be felt as a tumour of soft consistence.

Composition of the Urine.—*The condition of the urine* also shows but few changes which may be regarded as differing from other infectious diseases. *Albuminuria*, which disappears with the fever and with the disease, is quite common, a more marked irritation of the kidney producing an actual nephritis is generally rare in the case of typhoid (opposed to scarlet fever, diphtheria and others). For a time it was believed that the *diazo reaction*, which, with the urine of enteric-fever patients, shows an intense red colour, would furnish us a sign which was important in the diagnosis of typhoid. However, it was shown that the diazo reaction of the urine of enteric-fever patients is occasionally, although not often, absent (in about one quarter of the cases at most) and, on the other hand, that it is not infrequently present in tuberculosis and in other infectious diseases. Nevertheless, I should advise, in questionable cases in which the diazo reaction is absent, to be careful with the diagnosis of enteric fever, especially as the reaction sometimes becomes positive late, not until the second week of the disease.

Complications.—The *complications and sequelæ* are more numerous in

enteric fever than in any other infectious disease. Their exact knowledge is of great importance in the diagnosis of enteric fever, especially in certain stages, and they are to be mentioned briefly. The most important are those which are due to intestinal ulcers, especially *intestinal hæmorrhages* and *intestinal perforations.*

Intestinal hæmorrhages are not very frequent in enteric fever (from 5 to 10 per cent of the cases); they are of varying intensity; occasionally only traces of blood are found mixed with the stools, at other times large masses of blood are evacuated; the stools then assume a reddish-brown or black colour. Accordingly, the subsequent phenomena also vary: Under some conditions there are no symptoms which call attention to this slight blood loss, and its existence is only noted upon a casual examination of the fæces; in other cases a marked intestinal hæmorrhage will be followed by collapse, with pallor of the face, cold extremities, smallness of the pulse and a fall in temperature. The latter, the fall in temperature, is occasionally the first sign that an intestinal hæmorrhage has occurred, i. e., if the blood which was extravasated into the bowel is not discharged in the stools and, in the midst of a temperature of a continuous type, normal or subnormal ranges should be noted, which always points to an extraordinary incident, generally a severe collapse. The intestinal hæmorrhages usually take place between the third and fourth week, especially at the time when the necrotic crusts are thrown off. Intestinal hæmorrhages which occur earlier, i. e., in the first and second week, are due to parenchymatous hæmorrhages of enlarged Peyer's patches, whereas those which appear in the fifth or sixth week are due to sluggish ulcers.

Intestinal perforations occur most frequently between the second or fifth, rarely during the sixth, week or still later, by the necrotic process extending to the serous layer of the gut which may tear, due to stronger peristalsis or dilatation by gas. As a rule, the tear is found in the ileum, more rarely in the large intestine or in the vermiform process. The symptoms of perforation are ushered in by severe pain in the abdomen or by collapse; this is followed by tympanites and pain upon pressure of the abdomen, displacement of splenic and liver dulness by the air which has entered the abdominal cavity from the gut, in short, the entire symptom-complex from so-called "perforative peritonitis," the detailed explanation of which must be looked for in the differential diagnosis between perforation of the bowel and enteric fever and *meteorism* (which see), which so commonly occurs in typhoid fever. The almost invariable result of these severest complications is death, but recovery is not opposed to the correctness of the view that perforation may have taken place. I have seen several patients with perforative peritonitis get well; these were cases in which the diagnosis could be controlled by the gradual return of hepatic and splenic dulness and also by a friction sound which occurred over the liver during the period of convalescence. Perforative peritonitis without the entrance of gas into the peritoneal cavity may result from the rupture of purulent mesenteric glands or of a splenic or hepatic abscess. A simple purulent peritonitis may also be due to the typhoid ulcers and occur without rupture.

Secondary Pus Formation.—The last-named formations of pus in the course of enteric fever, as well as the *meningitis, pleuritis,* the *pulmonary abscesses* and the *cerebral abscesses, furunculosis* and larger *cutaneous and muscular abscesses* (especially in the regions of the gluteal and gastrocnemius muscles), purulent *mediastinitis, retropharyngeal abscess, suppurative parotitis,* abscess formation of acutely arising goîtres, *lymph-gland suppurations, purulent arthritic inflammations,* etc., and, finally, well-pronounced pyæmia, of which I have seen several examples, owe their origin to a mixed infection, i. e., they are probably due to the fact that, according to experience, the chemical products of the typhoid bacilli, more than in the case of other infectious diseases, prepare the soil in which pus-producing bacteria, usually streptococci, more rarely staphylococci, finding their way into the body, display their action. These *mixed infections* are quite common occurrences in severe cases of enteric fever. Naturally, in some few cases, which have been very carefully examined, of collection of pus in the course of typhoid, pure cultures of typhoid bacilli were found, without the usual pyogenic organisms, staphylococcus and streptococcus pyogenes being present. Under such conditions it must be assumed either that the typhoid bacilli themselves are pyogenic or that in these foci active staphylococci and streptococci which were originally present, having been brought in contact with typhoid bacilli, have perished. The latter assumption, however, cannot be regarded as being always correct, as it has been proved that exclusively typhoid bacilli were present in fresh deposits of pus, and as unobjectionable experiments of various investigators have shown that the subcutaneous injection of typhoid bacilli can produce pus. The decision in which manner suppuration has occurred in the individual case can only be found by an exact bacteriologic examination and culture.

Other Rarer Complications.—After having discussed the most important and most frequent complications of enteric fever, the other complications shall be briefly mentioned, according to the organ to which they are directly related.

DIGESTIVE ORGANS

In the *oral cavity* in the course of typhoid, superficial and deep ulcerative processes are noted (partly due to pressure of the teeth), in the later course of the disease aphthous stomatitis. Similar conditions are seen upon the *mucous membrane of the pharynx*; besides simple angina with the formation of superficial erosions in the palatine arches and tonsils, angina of a phlegmonous and diphtheritic character may occur and give rise to ulceration. *Parotitis* is especially feared, which arises partly from a continuation of a catarrh of the mouth to Steno's duct and which may end in disintegration or may be partly of a purulent nature and is then brought about in the method which has been previously described. Suppurative conditions in the parotid are dangerous on account of angina Ludovici, venous thrombosis and pyæmia, which may sometimes follow it. The facial nerve may also be destroyed, and paralysis of the nerve, from which there is no recovery, may be the result. Complications on the part of the stomach are, in general, rare if the dyspeptic symptoms and vomiting be excluded. I can remember, in reviewing the great number of cases that I have observed, but two rare complications on the part of the stomach which deserve mentioning—*hæmatemesis,* to which post mortem only a marked type of hyperæmia of the mucous membrane corresponded, and the formation of *ulcers of the stomach* in an individual whose gastric mucous membrane, analogous to the mucous membrane of the intestine, contained numerous agminated follicles; the latter, as well as

Peyer's patches, were infiltrated with medullary substance and ulcerated. The enlargement of the *spleen* may attain such enormous dimensions that it may burst, but this is a very rare cause of *rupture of the spleen*; more frequently it occurs as the result of abscess formation due to *infarcts in the spleen*. These latter again are partly the result of emboli which come from the heart and partly due to thromboses which are produced by the circulation which has become deficient in the later course of typhoid, in which case the enteric-fever bacilli may act as pyogenic agents. As in the spleen, so, in rare cases, may *abscesses* occur in the *liver*. *Jaundice* is much rarer in enteric fever than in other infectious diseases (in scarcely 2 per cent of the cases); hence, the presence of icterus, in a questionable case, is decidedly against the existence of enteric fever. In some few rare cases the toxine of enteric fever has given rise to a severe fatty degeneration of the liver which clinically resembled the process of *acute yellow atrophy of the liver*.

RESPIRATORY ORGANS

Coryza is so rare in the course of enteric fever that, if a *nasal catarrh* occurs at the onset of a questionable febrile affection, enteric fever may be *excluded*; however, *epistaxis* in the course of the first week is quite common. *Laryngeal ulcers* are comparatively frequent in severe cases which terminate fatally (in about 20 per cent of the fatal cases); they occur between the second and eighth week and are either the result of diphtheritic changes or (in the presence of adenoid tissue in the mucous membrane of the larynx) the product of actual typhoid medullary infiltration. As a rule, these laryngeal ulcers run their course without symptoms, in other cases they give rise to hoarseness, difficulty of deglutition, etc., occasionally they produce perichondritis or œdema of the glottis; I have seen an apparently completely convalescent enteric-fever patient die of œdema of the glottis which was due to a latent typhoid ulcer. We have already referred to the frequent occurrence of catarrhal, hypostatic *pneumonia*; frequently deglutition pneumonia also develops, more rarely actual *fibrinous pneumonia*, evidently due to mixed infection, in which case, besides enteric fever, the characteristic course of the pneumonia is at least indicated, and the sputum is bloody, curiously even more markedly so than in the usual fibrinous pneumonia. In such cases (as in a case observed in my clinic) *typhoid bacilli* are also found *in the sputum*. Eberth bacilli were also seen in the sputum of enteric-fever patients in whom *lobular pneumonia* had developed, whereas in the sputum of bronchitis they seem to be constantly absent. *Gangrene* of the lung may also be superadded to pneumonia; but gangrene of the lung occurring in enteric fever as a complication is more frequently the result of emboli from ichorous or gangrenous areas of the periphery. *Pulmonary phthisis* is not at all a rare sequelæ of enteric fever, although its frequency has no doubt been exaggerated. Usually, in such cases in which tuberculosis is joined to enteric fever, the conditions are probably such that tuberculosis already existed in its incipiency before typhoid was acquired, and the latter only prepared the soil for the speedy development of tuberculosis in making it more favourable. In quite a number of cases, lately, in my clinic, in the early stages of typhoid *pleurisy* was noted, which was certainly present, as friction sounds and small exudates appeared (proved by puncture). According to my experience, the existence of a pleurisy should not be considered so rare a complication in enteric fever as to use its presence in deciding against enteric fever from a differentio-diagnostic standpoint. In the pleural exudates, the serous as well as the purulent, typhoid bacilli have been found repeatedly.

ORGANS OF CIRCULATION

As rare as endocarditis and pericarditis are (I have seen but very few cases of typhoid endocarditis), so common are degenerative processes of the *heart muscle* (especially parenchymatous degeneration and interstitial myocarditis) in the course of enteric fever, even from the end of the second week on. As a result of them, weakness in the contractility of the heart results with its consequences: *Dilatatio-*

of the heart, thrombus formation, engorgement and severe collapse. This sometimes causes rapid death; we find in such cases post mortem complete fatty degeneration of the heart muscle, but occasionally *no* anatomical changes in the heart, and the cause of the fatal collapse has then probably been due to a toxic ***paralysis*** of the ***vaso-motors***. The examination of the blood shows, besides the above-mentioned rare finding of typhoid bacilli, ***no increase in the white blood cells***, in contrast to the condition in most of the infectious diseases, especially pneumonia, in which leucocytosis occurs. Nevertheless, here and there an increase in leucocytes is noted also in the course of typhoid, provided inflammatory affections complicate the disease.

URINARY AND SEXUAL ORGANS

Of the frequently transitory albuminuria we have already spoken. This toxic irritation which probably is always present, rarely increases, as has already been marked, to an actual nephritis, which, if it does not lead to death, recedes again, similar to the condition in other infectious diseases, even if it has persisted for weeks. Typhoid bacilli are not rarely noted in the albuminous (nephritic) urine. Whether they may also give rise to cystitis, has not been positively demonstrated; it is certain, however, that now and then acute catarrh of the bladder occurs in the course of typhoid. *Orchitis* is often noted in men, *œdema* and *gangrene of the vulva, abortion*, and *metrorrhagia* in women, as sequelæ of enteric fever.

NERVOUS SYSTEM

The nervous system, as has been frequently mentioned, is constantly to a stronger or milder degree affected by the typhoid intoxication. Severer *phenomena*, due to special *anatomical* changes in the central nervous system, are: *Aphasia*, which is not rare in the course of enteric fever; hemiplegia, the result of hæmorrhages into the brain substance (caused by a greater faculty of tearing of the walls of the vessels, apparently acquired in enteric fever); further, the symptoms of sinus thrombosis and especially those of *meningitis* which may sometimes, according to my experience, although rarely, make the diagnosis extremely difficult; paraplegia (as a result of myelitis typhosa), phenomena of multiple sclerosis, etc., finally, paralyses of individual nerve trunks as a result of *neuritis*—in general, however, rare sequelæ.

SKIN, MUSCLES, AND BONES

Besides the suppurations which have already been referred to, there are to be mentioned as complications and sequelæ of enteric fever: Erysipelas, cutaneous hæmorrhages, *gangrene* of the various peripheral parts of the body, *periostitis* which lately has been frequently seen as a sequela. An almost constant change in the *muscles* in enteric fever, to which Zenker first called attention, is a *parenchymatous degeneration* of the voluntary muscles, showing itself in the form partly of fatty, partly of waxy degeneration, and, as is well known, especially marked and constant in the adductors of the femur and in the rectus abdominis. The brittleness of the musculature brought about in this manner often causes tearings of the same and widely disseminated outpourings of blood into the muscle substance.

Finally, that so long-lasting an infectious disease as enteric fever, which so markedly affects the organism, also induces injurious *changes in nutrition*, is self-evident. Therefore, we see at the acme of the disease, or after it has run its course, the development of a hæmorrhagic diathesis, and the complete convalescence of the patient may be deferred for a long time on account of a well-marked marasmus.

Varieties and Types.—Before we discuss the differential diagnosis of enteric fever it is necessary to mention several varieties of the course of the disease which under some conditions may make the diagnosis of the affection very difficult. I shall entirely disregard all those cases of enteric fever

in which certain organs are particularly affected and so prominently dominate the scene that it might appear as if a pulmonary, cerebral, or renal disease were the one in question; under these circumstances a *pneumotyphoid* or *cerebral typhoid* or *renotyphoid* is spoken of. I do not believe that this is proper, as a prominence of some individual symptoms occurs in every infectious disease without our being justified on that account to differentiate separate forms of the respective infectious disease and give it a different name. In such cases it is the duty of diagnosis to separate the essential in the picture from the entire symptom-complex and correctly to recognise such prominent symptoms as being due to enteric fever.

Those cases of enteric fever are more difficult to recognise in which the course of the affection is so mild and many of the prominent symptoms are so obscure or the affection runs such an unusual course that the disease is not recognised (*typhus levissimus, typhulus, abortive typhoid, ambulatory typhoid*). In the case of *typhus levis* the intensity of the symptoms, in spite of the disease running its usual course as to duration, is insignificant; the fever does not reach high ranges, and most of the symptoms are but scarcely indicated, etc. In *abortive typhoid* the disease begins with marked symptoms, but the duration of the individual periods of the malady is so conspicuously brief that already after one to two weeks the patients are free from fever—the entire course of the affection being a precipitated one. Combinations of typhus levis with the abortive variety are so frequent that the differentiation of both forms depending upon the intensity and duration of the course, is frequently impossible in practice and—is valueless.

In rare cases enteric fever, as the latest investigations of Liebermeister and Gerhardt have shown, may run its course *without fever* (*afebrile typhoid*), so that only enlargement of the spleen, eruption, conspicuously severe, general constitutional symptoms, diazo reaction of the urine, dicrotism of the pulse, and, eventually, the ætiology of the special case point with certainty to the existence of enteric fever, in spite of there being no rise in temperature during the course of the affection which frequently lasts for weeks. In such cases the proof of typhoid bacilli in the blood, in the urine, or in the intestinal evacuations, and, above all, a positive Gruber-Widal reaction (see below) decide the question and make of the probable diagnosis a *certain one*, whereas this has been impossible up till lately unless an intestinal hæmorrhage occurred in the course of the affection or for other reasons the disease terminated fatally.

Relapses.—In certain cases a mild course of the affection is followed by a mild or even a severe relapse, which is also of importance for the diagnosis of these cases. It is well always to be prepared for *relapses* in every case of typhoid during the first two weeks after convalescence has begun; it is rare for relapses to occur later. Why the period of intermission varies so much in the various cases, why the frequency of relapses is very much greater in some epidemics than in others, cannot be explained at present, a probable reason cannot even be given.

Diagnostic Value of Individual Symptoms.—In the greatest majority of cases the diagnosis of enteric fever can be made with required certainty '

excluding all other diseases. This is the case if the *course of the temperature* has been noted from the beginning or at least from the second day on, if the *pulse is rapid but, nevertheless, slow in proportion to the height of the temperature,* if the *spleen* can be demonstrated to be *enlarged* in the first week, and if an *eruption* appears in the course of the second week. All other symptoms of enteric fever, such as the diarrhœa, the bronchitis, the nervous phenomena, etc., have less diagnostic value than the above four phenomena, as they are either not constant or they are developed in other diseases in the same manner as in enteric fever. For the last-mentioned reason the *enlargement of the spleen* is the least important of the *four cardinal symptoms*; it is true, it is a constant phenomenon but cannot always be definitely determined, but, above all, it is a symptom which occurs in the other infectious diseases as well as in enteric fever. This is also true of the *fever* after it has reached a continued type; it then differs but very little from the fever of other infectious diseases until the third or fourth week is reached, when the remissions set in and defervescence occurs in a special form which serves to differentiate enteric fever from relapsing fever or from typhus fever, miliary tuberculosis, etc. (compare the various fever types, Figs. 76, 77, 78). Especially important in a differentio-diagnostic respect is, in my experience, the *relative slowing of the pulse,* i. e., that the pulse beats, with a temperature of 104° F. and over, are constantly between 90 and 110 per minute; the dicrotism of the pulse has no specific diagnostic value. The *eruption,* finally, is more important than the other cardinal symptoms as regards diagnosis; questionable enteric-fever diagnoses attain certainty only with the development of some well-pronounced roseolas (a single spot is of extremely slight value in the diagnosis). Only exceptionally, as has already been explained previously, is the proof of typhoid bacilli of practical value in the diagnosis of typhoid. On the other hand, *serum diagnosis,* i. e., the test whether the blood serum of enteric-fever patients shows an agglutination with typhoid bacilli, has lately shown itself as a method which is of great value in *ascertaining* the diagnosis of the disease (Gruber-Widal typhoid reaction).[1]

Gruber-Widal Typhoid Reaction.—The reaction may be conducted in the following manner: A small quantity (about one half cc.) of serum which has been taken from the blood of enteric-fever patients before coagulation has occurred, is mixed with a fifteen times, or, as we shall see later on, better with a fifty times larger quantity of a fresh typhoid culture in bouillon (this culture should not be over twenty-four hours old). The mixture will appear cloudy on account of the suspended typhoid bacilli and should now be placed in the incubation oven. Then it should be noticed whether the fluid becomes clear in the course of the next twelve to twenty-four hours, whereas the agglutinated bacilli will be found at the bottom as a thick sediment (*macroscopic reaction*). Instead of this the *microscopic reaction* is commonly used now, i. e., a drop of the serum the size of which can be easily measured by the use of a very fine pipette, is diluted with fifty times as large a quantity of fresh typhoid culture in bouillon and the mixture then put under the microscope in a

[1] The designation "Gruber-Widal reaction" appears to me to be correct, as *both* investigators have done so much in the development of the question of the agglutinating property of typhoid serum with typhoid bacilli and have both shown the value of this fact in the diagnosis of enteric fever.

hanging drop. The immobilization and agglutination of the typhoid bacilli can be noted *at once* in this preparation, or is at least very pronounced after two hours, provided the serum shows agglutination with typhoid bacilli.

If the test gives a *positive* result, *the more diluted (50–100–1,000 dilution) the blood or serum is and the quicker and more powerfully the positive reaction occurs, the more is a certain conclusion as to the presence of typhoid fever permissible.* It has been shown that the blood of healthy persons, and of others suffering from other diseases, also shows an agglutination with typhoid bacilli, but to a much milder degree, so that a serum which is undiluted or in a 1: 15 (yes, even 1: 30) dilution, of patients not affected by enteric fever, although it often shows marked agglutinating properties, yet that this property is lost in a 1: 50 dilution, and also, even if the undiluted blood or serum of blood were used for the test, that the bacilli which showed marked motility before the test, never become immobilized and are agglutinated to one mass as suddenly as is the case, at least usually, with the serum of enteric fever. A further circumstance which, in the utilization of a positive result for the diagnosis of enteric fever, should enjoin us to caution, is that the reaction may be positive in the sense of a typhoid reaction also in non-typhoid fever patients, if the patient has had a previous attack of enteric fever and this not too long since. The blood of such persons evidently retains the agglutinating property for typhoid bacilli usually for a longer time (months, yes, as it appears, for many years). But, as it is well known that enteric fever may run its course in an unusual way or with symptoms so feebly developed in the form of typhus levissimus, an ambulatory typhoid, etc., it is clear that the positive result of the reaction as a certain sign of existing enteric fever loses greatly in value.

But, on the other hand, even the *negative* findings cannot at once be used in the diagnosis. It appears that in some few cases the serum of enteric-fever patients does not possess more marked agglutinating properties than the serum of non-typhoid-fever patients; further, it has been shown that the reaction, in certain cases of enteric fever, has been negative for days and weeks, in exceptional cases even up to the convalescent period, only then to become positive and of value in the diagnosis. It has been observed, elsewhere and also in my clinic, that the reaction, while negative at the beginning of the second week, became strongly positive a few days later. Especially marked was this in the following case: A servant in the pathologico-anatomical institute was taken sick, fourteen days after an autopsy of a typhoid-fever case had been held, with well-pronounced symptoms of enteric fever: Enlargement of the spleen, eruption, diarrhœa, etc. The reaction which was tried upon the sixth day of the disease proved negative, also upon the seventh day and on the thirteenth day. Intestinal hæmorrhages took place on the fourteenth day, and on the eighteenth day a weak positive reaction was noted; *on the twenty-first day, finally, a very marked positive reaction!* If the patient had died on the seventeenth day, as was easily possible, we should have had an autopsy of a marked case of enteric fever in the third week, in which the Gruber-Widal reaction did not occur.

But just in the fact that the reaction may first be negative and, as the disease advances, gradually become positive, the chief value of the test is found, according to my opinion, for the diagnosis of enteric fever. In such cases in which the reaction with a 1 : 50 dilution of the serum is first negative and in the later course of the disease shows a positive result, enteric fever may be diagnosticated with certainty, and other diseases excluded. In other cases we must be very cautious, as I believe, with the diagnostic use of this test whether it be positive or negative. If all the points are well kept in mind which have been mentioned in reference to the peculiarities of the Gruber-Widal-serum diagnosis, a valuable aid is present in the diagnosis of enteric fever.

The more of these cardinal symptoms are present at the same time, the less questionable is the diagnosis of enteric fever. It is completed by the possible presence of other symptoms which, arranged according to their diagnostic value, shall be briefly mentioned. First, after the four cardinal symptoms: The enteric-fever stools, the appearance of the tongue, the

bronchitis; then the nervous phenomena, the enlargement of the liver, epistaxis; further, the ætiology and the complications. Of the latter the following have marked diagnostic value: Intestinal hæmorrhages, intestinal perforations, and the laryngeal ulcer; of less value are: Parotitis, abscess formation, periostitis, and the other so numerous complications of enteric fever which always play a secondary *rôle.*

Against enteric fever in general, although not absolutely, is the prominence of coryza, of marked sweating in the first stages of the disease, of herpes (in scarcely 5 per cent of the cases), and of endocarditis; further, retraction of the abdomen, constipation existing from the beginning and lasting for weeks, and the constant absence of the diazo reaction in the urine.

Differential Diagnosis.—As will have been gathered from what has been said and, above all, from what is taught by daily experience at the bedside, the diagnosis of enteric fever can scarcely be made in the first week after a single examination. As a rule, prolonged observation and a *differential diagnosis* which follows certain directions are necessary, which we shall now discuss in detail.

Enteric fever is most frequently confused, as all diagnosticians have emphasized from the earliest time, with *acute miliary tuberculosis* and, further, according to my experience, with *cryptogenetic septicopyæmia.*

Miliary Tuberculosis.—The fact that the body may be suddenly overwhelmed with the tubercular poison, which often occurs in persons who have been apparently in the best of health up to that time, and that this infection runs its course with enlargement of the spleen and high fever, but frequently, as is also the case in enteric fever, furnishes very ill-pronounced or scarcely any positive signs for the diagnosis upon examination of individual organs, easily shows that the differential diagnosis of the two conditions under some circumstances may give rise to enormous difficulties. As an eruption also occurs in rare cases of miliary tuberculosis, and as, further, bronchitis and cerebral phenomena, as a rule, belong to both affections, it is clear that there are cases in which it is best for some time to render no diagnostic decision at all. Nevertheless, the difficulty of the differential diagnosis is certainly exaggerated, in my opinion; in by far the majority of the cases it is possible, rather, as I can say from a long experience, to make the diagnosis of one or the other of these affections with sufficient certainty. It is self-evident that, as regards the differential diagnosis, the Gruber-Widal reaction should be tried at once, and utilized in the method as given above. Further, the sputum and the urine are to be examined for tubercle bacilli; however, only in the rarest cases of an acute-infectiously arising tuberculosis does one attain results by this method. Most frequently I have been very much aided in the differential diagnosis of complicated cases by making a very careful examination of the respiratory organs. The slightest deviation of the notes at the apex of the lungs, the concentration of the fine râles to the upper (instead of the lower) parts of the lungs, and, besides, a metallic character of the râles, are in *favour of tuberculosis.* In many cases acute miliary tuberculosis originates in a mild affection of the apex of the lungs, which had not been observed up

till then, or, in the general overwhelming of the body with tubercle bacilli, the chief deposit takes place in the lung. Thus it occurs that *cyanosis* and *dyspnœa* appear as further differential signs and symptoms in acute miliary tuberculosis (as opposed to the condition in enteric fever). These phenomena are not in proportion to the degree of the bronchial catarrh which is noted upon auscultation, as, besides the intensity of the latter, also the direct diminution of the respiratory surface by the accumulation of tubercular nodules in the finest broncholes must be taken into consideration. It is further advisable to observe the *frequency of the pulse* in comparison to the height of the temperature. Relative slowing of the pulse from the onset is in favour of typhoid provided the signs of a meningitis are not simultaneously present, which may be accompanied with a relative slowing of the pulse. Apart from this, the pulse frequency in miliary tuberculosis, in contrast to that in enteric fever, is from the onset a conspicuously high one (120 to 130 beats), which is only present in enteric fever with simultaneous cardiac weakness, especially in the later stages of the disease. Of less importance is the *condition of the spleen;* it is true, it is not so constantly enlarged in acute miliary tuberculosis as in enteric fever; however, the diagnosis of miliary tuberculosis cannot be based upon the absence of enlargement of the spleen, as in rare cases of enteric fever the enlargement cannot always be demonstrated (especially on account of the meteorism). This is also true of the *diazo reaction of the urine* which for a time was looked upon as a characteristic of typhoid; now and then it is absent in typhoid and, on the other hand, is not rare in tuberculosis. A *complicating meningitis* is so rare in enteric fever and relatively so frequent in miliary tuberculosis that the appearance of this condition in the picture of the disease is decidedly more in favour of tuberculosis. This is similarly true of the *eruption,* which, especially if it occurs in successive crops, points unquestionably more to typhoid. But it must never be forgotten that a roseolar eruption has also been noted in rare cases of miliary tuberculosis. Diarrhœa, laryngeal ulcers, etc., are symptoms which may develop upon a tubercular basis, and, therefore, are of no value in the differential diagnosis. However, miliary tuberculosis may be looked upon as absolutely certain if the ophthalmoscopic examination which in a doubtful case must never be neglected and should be frequently repeated, reveals *chorioid tubercles* in the fundus of the eye. Finally, the *bacteriological examination* may be of value in the diagnosis. As has already been mentioned, typhoid bacilli are found occasionally in the blood of enteric-fever patients, and it has also been possible to find tubercle bacilli circulating in the blood in a few cases of miliary tuberculosis. However, a positive finding has only occurred in exceptional cases, and the numbers of bacteria thus found have been very small; besides, the certain proof that we are dealing with typhoid bacilli is often so difficult that a decision upon such theoretical grounds in regard to the specific microbes can rarely be utilized in practice. We are compelled at the bedside to depend upon the diagnostic points which have just been described, in order to make our diagnosis in [illegible] other direction. In by far the majority of cases is it then due consideration of these points to make the correct diagn[illegible]

if the affected patients have been observed for some little time. In such instances it will usually be possible to receive important aid from the special course of the temperature. The course of the fever is generally irregular in miliary tuberculosis, much more irregular than in the case of typhoid, and remissions even occur during the time of the acme of the fever; besides, these remissions may even take place in the evening, a condition not noted in typhoid except in those cases in which certain complications (intestinal hæmorrhage) effectually influence the regular typhoid-fever curve.

Cryptogenetic septicopyæmia in general is easier to differentiate from enteric fever than miliary tuberculosis, although, according to my experience, sepsis assumes some forms in which the diagnosis, for a time at least, is very indefinite. Naturally, those cases of septicopyæmia due to trauma are excepted from this, in which the ætiology, without more ado, guides the diagnosis in the proper direction. More difficult, occasionally, is the diagnosis in the cryptogenetic variety of sepsis which has in common with enteric fever the enlargement of the spleen, the high temperature and the general nervous phenomena, and, under some circumstances, roseolar eruption, (as in one of my cases in which a plentiful roseola recurred for weeks), bronchitis, etc. In favour of the presence of septicopyæmia, in contrast to enteric fever, are: The type of the fever, in the course of which marked falls and rises, accompanied with chills, will not remain absent; further, the very common localization of the infection in the heart as a ("malignant") endocarditis, in the joints in the form of monoarticular or polyarticular, but in general not fleeting, inflammations; the complication with meningitis; the many forms of exanthems which occur; the painfulness of the bones upon pressure; the absence of the relative slowing of the pulse which, on the contrary, is very frequent and irregular; and, above all, when they are present, the changes in the eye-ground (ecchymoses with white centres or without such). In rare cases cocci may be found in the blood in septicopyæmia, especially if they accumulate and increase in the capillaries and grow into the veins, thus reaching the general circulation. It shall again be emphasized here that in rare cases septicopyæmia occurs as a complication in the course of enteric fever, i. e., that, therefore, under some circumstances both affections may occur at the same time in the same patient.

Typhus Fever and Relapsing Fever.—The differentiation of enteric fever from other infectious diseases than the two mentioned, offers but slight difficulties. *Typhus fever* may be differentiated from enteric fever by the rules previously laid down on p. 899. The early appearance of a plentiful, usually petechial, exanthem, the high-pulse frequency, the coryza and the conjunctivitis, the severe cerebral phenomena, and, further, the initial sudden rise and the terminal critical fall of the temperature are the principal differentio-diagnostic points in favour of typhus fever; of course, the presence of an epidemic of typhus fever is also to be taken into consideration in the differential diagnosis. The latter is also a practical point of view in deciding whether enteric fever or relapsing fever is present. The diagnosis of the latter disease, however, aside from the very char-

acteristic temperature course, is never in question since the discovery of the pathognomonic spirilli.

Acute Exanthems.—*Measles, scarlatina and variola* are to be considered in a differentio-diagnostic respect only during the prodromal period, and even here their symptoms (coryza, angina, variolous prodromal exanthem, etc.), and especially also the fever, are of such a nature that the onset of enteric fever can usually be excluded with certainty. After a few days, i. e., after the characteristic exanthem has occurred, a confusion is no longer possible at all. The exanthem itself is like the roseola of enteric fever only in the case of *measles*. In rare cases the eruption of typhoid fever may be as copious as the exanthem of measles; but the latter shows itself as early as the fourth day after a preceding fall of temperature. The eruption in the case of measles usually appears first in the face and then distributes itself over the entire body; a diffuse reddening with small dark spots may be noted upon the mucous membranes of the fauces, cheeks and pharynx even in the prodromal period. In short, a wrong diagnosis, "enteric fever with simultaneous measles," is hardly possible upon a careful examination, and a further discussion of the question is valueless.

A confusion of enteric fever with two other infectious diseases is more possible, which, though usually localized, show themselves by severe organic changes, in rare cases may occur in such a manner that the local phenomena are entirely subjugated by the general infection, or cannot be discerned at all. The affections referred to are the so-called *central pneumonia* and *internal anthrax*.

Internal Anthrax.—Since it was shown by me and others that anthrax may sometimes occur in man, instead of in the usual form of malignant pustules, as a severe infectious disease with intestinal phenomena, it will be well in questionable cases of enteric fever also to think of the possibility of anthrax intestinalis. Fever, enlargement of the spleen, diarrhœa, general cerebral phenomena are common to both affections. However, in the case of anthrax due to infection by nourishment contaminated by anthracic particles, certain symptoms become prominent which deviate from the usual picture of enteric fever: Vomiting, colic, bloody diarrhœa and hæmaturia, marked dyspnœa, and cyanosis which in one of my cases was the most conspicuous, in fact the only, symptom; regularly in my cases very remarkable suffusions and black vesicles filled with blood appeared upon the mucous membrane of the mouth. Naturally, the diagnosis is easy as soon as the possibility of the occurrence of anthrax is at all considered—the microscopical examination of the bloody urine and of the blood taken from the tip of the finger shows, in the case of anthrax, the presence of the very characteristic anthrax bacilli; should the latter not be demonstrated in this manner, the inoculation of guinea-pigs or mice will soon clear the situation. As through the intestine, so may the anthrax bacilli enter into the body also through the lungs (by inhalation) (rag-pickers' disease, *anthrax pneumonia*).

Central Pneumonia.—We naturally now come to the question whether the occurrence of *pneumonia* may eventually resemble the appearance of enteric fever. As has already been mentioned, an infectious fibrinous pneumonia may become associated with enteric fever. These cases, however, are rarely to be considered in a differentio-diagnostic respect, as croupous pneumonia almost never (I know of but one case in my practice) occurs as a complication during the first days of enteric fever. In such exceptional cases "*pneumotyphus*" is spoken of, and it is assumed, without there being a possibility of proof that the typhoid infection did not originate in the intestine but in the lung, and has taken its course from there. In these cases, with the decline of the pneumonic phenomena, the enteric-fever symptoms become unquestionably prominent in the second week, such as diarrhœa, meteorism, eruption, etc. On the other hand, there are cases of genuine pneumonia which are in no relation with enteric fever but which, as their course may be for days without dulness and without bloody sputum and as they show absolutely no objective morbid signs besides the fever, may give the impression of being enteric fever. These are the so-called *central pneumonias*. They are not frequent upon the whole, although the bad custom has arisen, in cases in which after one or two examinations a negative result has been obtained, to diagnosticate "central pneumonia." It is well to be as saving as pos-

sible with such diagnoses and much better to admit that the febrile affection in question is not possible of a certain diagnosis at that time. Against enteric fever and in favour of an affection localized in the depth of the lung, therefore for a pneumonia which runs its course without dulness, are: Onset of the affection with *chill* and a rapid rise of temperature, *stitch in the side in breathing*, difficult respiration, the occurrence of *herpes labialis* and of *jaundice*. *The ensemble* of these phenomena may, in cases in which, besides, the cardinal symptoms of enteric fever are absent (relative slowing of the pulse, enlargement of the spleen, etc.), and in which no positive signs point to the presence of another infectious disease, at least give rise to the suspicion that we are dealing with a latent central pneumonia. This suspicion gains in certainty if an unquestioned *leucocytosis* can be demonstrated which, in the case of pneumonia, belongs to the almost regular phenomena and which, on the other hand, is absent in enteric fever provided no inflammatory complications exist. The diagnosis becomes certain if after a few days either pneumonic sputum appears—the one time expectoration of characteristic tenacious, rusty, glassy sputum suffices—or anywhere in the lung a tympanitic note may be encountered or bronchophony or crepitant râles occur, and the fever disappears by crisis with marked sweating. So long as the appearance of the sputum or a physical sign does not show a change in the lung, the diagnosis of a central pneumonia had better be held in suspense. In from two to four days the situation usually becomes clear; but the determination of a pneumonia which advances from the centre to the periphery under all circumstances requires a very minute examination and great skill in physical diagnosis.

Meningitis, Meningo-Typhoid.—Finally, I wish to remark that occasionally the differentiation between enteric fever and *meningitis* may be very difficult. I exclude those very rare cases in which a meningitis occurs as a complication in the course of enteric fever; in such cases the diagnosis of enteric fever has already been made for some time before the cerebral affection is added. I refer rather to *cases of enteric fever which from the onset appear to resemble meningitis*, in which coma, convulsions, rigidity of the muscles of the neck and back and other symptoms so dominate the picture that we first think of meningitis and not of typhoid (" *cerebral typhoid*," " *meningo-typhoid* "). The relatively slight increase of the pulse and also the eruption may occur in both diseases; but the fever shows a less typical course in the case of meningitis, the belly is usually retracted, whereas in typhoid it is tympanitic, the general hyperæsthesia and especially the headache are much more marked in the case of meningitis than in the case of enteric fever, even if the latter runs its course with these severe cerebral phenomena. Still more in favour of meningitis are the appearance of localized cramps and paralyses and the eruption of herpes (especially upon the extremities); finally, also the absence of eruption, diarrhœa, and enlargement of the spleen, which in such a severe typhoid infection as must always be presupposed in such cases, are always present. Also the inefficacy of all therapeutic measures calculated to reduce the temperature in the individual case may be of value in the diagnosis in meningitis, as antipyretics, and especially the systematic use of cold baths, almost always cause the cerebral phenomena of enteric fever to become markedly milder, whereas these symptoms are not influenced by similar measures in meningitis. Nevertheless, in spite of all these conditions a certain diagnosis in practice is not always possible. I would advise that the diagnosis of meningitis should by no means be definitely made if a certain cause for the origin of the disease cannot be found; as long as this is not possible, the diagnosis of meningitis, no matter if it be ever so well founded, is upon a weak footing. The ophthalmoscopic examination dare never be neglected in a questionable case, as some positive points for the diagnosis are usually offered in the case of meningitis (optic neuritis, retinal hæmorrhages, etc.). (See Suppurative, etc., Meningitis.) On the other hand, the positive result of the Gruber-Widal reaction with consideration of the points that have been previously noted, may make the diagnosis of enteric fever certain.

Gastric Fever; Mucous Fever.—Before we finish the differential diagnosis of enteric fever, we must discuss the question whether the disease may be separated diagnostically from two other affections, which, formerly at least, played an important part in pathology as substantive affections, *gastric fever* and *mucous fever*. Since our methods of examination have become more exact and our diagnoses are

better controlled by pathologic anatomy, these two affections have disappeared more and more from pathology. It is true that there are gastric and intestinal catarrhs which *run their course with fever* and the clinical picture of which may distantly resemble enteric fever. But they are *very rare*, according to my experience, as opposed to the cases of gastro-enteritis which run an afebrile course.

The masses of mucus which are passed in the stool and which are said to characterize "*mucus fever*," are absent in the dejecta of enteric fever which are usually distinguished by the fact that, owing to *absence* of mucus, the well-known layers, i. e., the sharp demarcation between the fluid and compact portions of the stool, is brought about. If, therefore, *much* mucus is found in the fæces, a simple catarrh of the intestines should be thought of primarily. The complication of typhoid with chronic catarrh of the large intestine, which is occasionally noted in the late stages of the enteric-fever course, may in rare cases be the source of the admixture of mucus. If a simple intestinal catarrh is accompanied with fever and, besides, as occurs in very rare instances (I have noted *one* such case), with enlargement of the spleen, the doubt is justified whether enteric fever is present or not. Here the pronounced colic, the irregular course of the fever, as well as the absence of the relative slowing of the pulse and of bronchitis, decide in favour of an enteritis with fever, against the presence of typhoid, and this may be made certain in the further course of the affection by the absence of the positive Gruber-Widal reaction up to the time of convalescence.

The prevalence of dyspeptic phenomena, the pappy taste, the nausea, the eructation and vomiting—are at once against enteric fever and rather in favour of *gastritis*, which, as we have already remarked, in rare cases may run its course with fever. If this is supervened, in the further course, by herpes labialis, the diagnosis becomes almost unquestionable in that these symptoms are quite rare in enteric fever, and even then only are not noted as isolately. Real difficulties in the differential diagnosis cannot, in the long run, be caused by such an afebrile gastritis. According to what has been said, it is advisable to drop the diagnoses "gastric fever" and "mucus fever" entirely, even for the above-described exceptional cases, and to choose in their places the designations: *Febrile gastritis* or *febrile (infectious) enteritis, respectively gastro-enteritis.*

[**Paratyphoid.**—An affection has recently been described in which the symptoms are identical with those of enteric fever, differing in no wise from the well-known classical picture, but in which a different, separate micro-organism has been found as the specific cause. This organism was called by Achard and Bensande, who first described it in 1896, *bacillus paratyphoid*, and the disease to which it gives rise "*paratyphoid.*" Cases in which this bacillus has been found have occurred in different parts of the world. The disease cannot be diagnosticated from its symptoms and signs, a bacteriological investigation being necessary to determine a positive diagnosis.

Diagnosis.—As has already been mentioned, there is no symptom by which this affection can be differentiated from enteric fever. *Paratyphoid* should be suspected in cases in which the complete symptomatology of enteric fever is present, but in which either the Gruber-Widal reaction is conspicuously feeble or is absent altogether during the entire course of the disease. The blood or the excretæ of the affected individual must also contain a bacillus susceptible of cultivation and showing agglutinating properties (1 to 50)].

DYSENTERY—BLOODY FLUX

Ætiology.—Dysentery which is characterized in an anatomical respect by an inflammatory, ulcerative affection of the mucous membrane of the large intestine which may be of a catarrhal, but is most frequently of a diphtheritic, character, unquestionably belongs to the infectious diseases. It is true, the specific infective principle in the shape of a micro-organism has as yet not been definitely determined [except the bacillus of Shiga]. Only for the endemic variety, occurring in tropical and subtropical countries (*"tropical" dysentery*) probably amœbæ, which were first shown by Loesch, in 1875, are to be considered as the generators of the affection, whereas in the epidemic variety occurring in Europe (*dysenteria nostras epidemica*) the pathogenic amœbæ of the tropical form are not found; it is certain that even in *normal* fluid stools frequently amœbæ occur which, however, are benign. As cause of the *non-tropical dysentery* various specific bacteria have been isolated. It appears as if sometimes certain forms of the coli bacillus, at other times varieties of streptococcus, may give rise to the affection, and that the individual epidemics depending upon these causes run a very different course. Clinical experience teaches that the spread of dysentery is principally due to the dejecta of dysenteric patients, i. e., the contagion occurs through privies, bed-pans, clothing, etc.; drinking-water also appears to spread the infectious principle. We must assume in such cases in which the infection does not occur from the rectum but from the stomach, that the poison of dysentery first becomes lodged in the large intestine, as here peristalsis is more sluggish, and its action upon the mucous membrane of the intestine is assisted by the natural decomposition of the excrementitious material. It is found in this connection that individuals who suffer from constipation, for example the insane, are more easily affected by dysentery than others. The poison of dysentery appears to have a long duration as to life, so that it is able to remain for a long time in the earth and cloaca and still retain its contagious property. The observation which was made a long time ago that a hot climate and the hot summer months, especially the change from great heat to marked cold, materially assist the development of dysentery, cannot up to the present be explained satisfactorily, as we know too little as yet regarding the existence and developmental condition of the generators of dysentery. But this much may be said, that dysenteric diphtheria of the bowel, although it does not materially differ from diphtheritic conditions of the intestine due to other causes (although in tropical dysentery the morbid process does not appear to begin in the mucous membrane but in the submucosa), can only be the result of a *specific* cause. It represents an *infectious local disease of the intestine* which, as a rule, is not followed either by diphtheritic affections of other organs or (besides those symptoms which depend upon the local disease) by phenomena of general intoxication (action of dysenteric toxines). *The toxine of dysentery, therefore, on no account must be supposed to be identical with the diphtheria toxine affecting the pharynx,* and dysenteric intestinal diphtheria must not be looked upon as an unusual point of localization of the virus of pharyngeal diphtheria. The period of incubation of dysentery is from a few days to a week. These facts which concern the ætiology of dysentery must always be taken into consideration in the diagnosis of the affection.

In contrast to other infectious diseases, the action of the toxine of dysentery in the body as a rule gives rise to no prodromes, but tenesmus and frequent evacuations of thin stools occur at once. In some rare cases these symptoms are preceded for a few days by dyspepsia, vomiting, moderate abdominal pains, diarrhœa which, however, is not characteristic, mild lassitude, and the affection may now begin with chilliness or with a decided chill.

Fever.—The *fever* which is present in this condition is but moderate, with morning remissions and evening exacerbations (reaching about 102°

or 103° F.). In other cases, especially in the mild ones, there is no rise in temperature; besides, even in the severest cases, thus in gangrenous dysentery, there is no rise in temperature at the time of the acme of the disease.

Fæcal Evacuations.—Now the characteristic *fæcal evacuations* occur which signalize the disease: Accompanied with colic in the vicinity of the umbilicus, and gurgling in the abdomen, marked tendency to stool occur, a painful *tenesmus* which recurs in continually briefer intervals and is accompanied with evacuations of but small quantities of fæces. According to the severity of the case, ten, twenty, yes, even a hundred movements may occur in the course of the day. The tenesmus is due to the inflammation of the mucous membrane of the rectum and to the reflex cramp of the sphincter ani. The patient makes the greatest effort to force out the contents of the rectum; a prolapsus ani is the usual result of this condition of the patient to free himself from the particularly tormenting sensation that a continuously irritating foreign body is in the anus. The *quantity* of the individual evacuations is always slight, amounting to but a few grammes, and in the course of the day all of the evacuations together scarcely amount to one litre!

Appearance of the Dysenteric Stools.—The *appearance* of the stools is at first fæcal, later only masses of pure mucus and blood are evacuated. An admixture of blood may be entirely absent in some cases, not only during certain periods of the disease but also during the entire course of the same. The mucous masses of the stools occasionally resemble yellowish, transparent, bile-like clumps, at other times they represent masses resembling swollen sago and at other times again they are nothing but shreds. If larger quantities of pus and remains of food are admixed with the dysenteric stools, the latter attain a more translucent, chopped-up appearance; in the later stages the dejecta may consist of pure pus. In other cases they are at one time purely hæmorrhagic or, containing but a small amount of blood, show a flesh-water colour. The more the necrotic changes in the mucous membrane of the bowels advance, the more a desquamation of shreds of tissue having a cadaveric odour and being brown or blackish in colour, will take place—the stool of *"gangrenous dysentery."*

Chemical and Microscopic Composition of the Dysenteric Stools.—The *chemical* composition of the dysenteric stools shows large amounts of mucin, *albumin* and *peptones*; the microscopical examination reveals numerous leucocytes, red blood corpuscles, more or less changed intestinal epithelia, triple phosphates, detritus, and countless bacteria; it should be especially observed whether the above-mentioned, probably specific, amœbæ are present.

Other Symptoms.—The belly, as a rule, is not tympanitic but sensitive to pressure, especially in the iliac region, later also in the entire course of the colon. Other symptoms are absent (especially enlargement of the spleen) or are at least of a subordinate nature, thus the results of the fever, and of the marked collapse which develops in the severe cases and which manifests itself by small pulse, cyanosis, weak voice, diminution in the quantity of urine excreted and albuminuria. With this there exist singul-

tus, præcordial anxiety, cramp in the calves, reflex tenesmus, fuliginous coating of the lips and tongue, delirium ("typhoid" dysentery).

Complications of dysentery are unusual. The most frequent of them are those due to the anatomical changes of the wall of the intestine, thus a peritonitis, respectively perforation of the gut, a periproctitis with formation of fistulæ, and especially also *abscess of the liver* due to emboli finding their way into the portal circulation, also splenic and pulmonary infarcts, the origin of which is brought about through the cava system. Further, there may occur in the course of the affection: Septicopyæmia, hæmorrhagic diathesis, and arthritic affections, which have been frequently noted as complications of dysentery. On the part of the nervous system, paraplegia, also hemiplegia with aphasia, have been observed in the course of dysentery. Their occurrence appears to be the result of marantic thromboses which either, starting from the hæmorrhoidal plexus, continue into the anterior sacral plexus and into the veins of the spinal cord and give rise to processes of spinal softening, or arise, remote from the location of the primary affection, in the sinuses of the brain. Peripheral palsies in the course of the brachial plexus have also been noted; whether they may be actually looked upon as the result of the dysenteric toxine appears to be very questionable to me.

Chronic Dysentery and its Results.—More important than these complications which at best are rare and the connection of which with the dysenteric process is rather questionable, are the *sequelæ* of dysentery which, in case the disease becomes *chronic*, remain for a long time after the disease has passed, occasionally becoming permanent. Then, it is true, the severest phenomena of dysentery disappear, but between the dejecta which have again become of a normal colour, the mucous masses and, according to my experience, constantly also bloody masses and eventually pure pus may be evacuated—for months or occasionally even for years. At the same time the general condition may remain undisturbed; as a rule, however, severe marasmus with dropsy and amyloid disease of the abdominal organs, etc., develop. If later a complete cessation of the dysenteric dejecta occurs (which, according to my experience, with the proper therapy is not so rare as is usually supposed), we must be prepared finally to encounter *stenoses of the bowel* of lesser or greater magnitude and, as a result of this, obstinate constipation, meteorism and, occasionally, also of ileus.

Differential Diagnosis.—The clinical picture of dysentery—the tenderness upon pressure over the course of the colon, the tenesmus and, above all, the composition of the stools—is so characteristic that a confusion with other affections is scarcely possible. But a mistake is out of the question only if the picture of dysentery is completely developed, and, what is especially important for the diagnosis, if an epidemic of dysentery exists. If it is a question of a mild sporadic attack of dysentery, the differentiations between dysentery and a non-infectious acute inflammation of the large intestine or rectum cannot be made so long as it does not appear possible to find the specific cause of dysentery.

In certain stages of the disease a carcinoma of the rectum may run its course with similar symptoms as dysentery, i. e., with severe, frequent tenesmus and with the evacuation of small muco-hæmorrhagic masses. But the history will show something of the nature of the rectal affection: Until the symptoms show the phenomena just described in the case of carcinoma of the rectum, months have passed since the onset of the affection; the condition resembling dysentery has developed very gradually after symptoms of constipation, pains in the small of the back, etc., have preceded for a long time. A digital examination or an ocular inspection of the rectum in many cases shows what the true condition is; and, just so, an exact examination of the rectum rarely leaves any doubts as to whether we are dealing with dysentery or a rare form of rectal syphilis.

Chronic dysentery shows solely the picture of a severe catarrh of the large intestine and rectum, with ulcerative changes in the mucous membrane, and may only from the history be correctly diagnosticated as a dysenteric affection.

ASIATIC CHOLERA—CHOLERA INFECTIOSA

Since Europe [and America], in the course of the last sixty years, has frequently shown epidemics, the last in Europe occurring about twelve years ago, and, as a result of this, the clinical phenomena have been studied upon thousands of occasions, especially, however, since R. Koch, in 1883, succeeded in finding the specific cause of the disease, the "comma bacillus," the diagnosis of Asiatic cholera is no longer difficult, on the contrary, it has become one of the most easy and certain of all diagnoses.

After a period of incubation of from one to two, the maximum cases of from four to six days, usually running its course without symptoms, the infected individual is suddenly taken with rumbling in the abdomen and, in the majority of cases, with a painless *diarrhœa*, which is characterized by the fact that conspicuously large masses of fæces are evacuated. These evacuations, which follow one another with increasing frequency, soon become watery, devoid of bile pigment, contain grayish-yellow flocculi (*rice-water stools*) and gradually lose their fæcal smell. This is accompanied with *vomiting* of similar watery masses, and hiccough. The more numerous the quantity of the evacuated material, the quantity in amount by far exceeding the fluids which are swallowed, the less the diuresis, until complete anuria occurs. Symptoms of *collapse* occur at the same time, as may sometimes be the case also, but to a lesser degree, in severe, non-specific catarrhs of the intestine, in which profuse diarrhœas occur as a result of the marked loss of water by way of the intestine. The consequences are extraordinary weakness, thirst that cannot be quenched, and a feeling of oppression, loss of the general turgescence of the body, sunken condition of the eyes, the nose becomes pointed, weakness of the voice (*vox cholerica*), and tonic painful cramps of various muscles. A further result of the blood becoming thick by the enormous loss of fluid is the *impairment of the circulation;* the slowing of the blood stream causes, farther on, insufficient decarbonization, poor nutrition of the heart muscle and weakness of the pulse, which finally can no longer be felt; the skin becomes cold, pale or cyanotic, the respiration increased in frequency and impeded.

Action of the Toxine of Cholera.—Recently, starting from the observation that toxic products of metabolism occur in the cultivation of cholera bacilli, it has been assumed that the general phenomena which have been just described in the picture of cholera primarily are due to the development of toxines. This theory (R. Pfeiffer) rests upon a good experimental foundation; it was shown that the injection of the toxines, respectively of dead cholera vibriones, acts most markedly and quickest from the circulation, and weaker from the peritonæum and the subcutaneous tissue, but that they are not absorbed at all by the intestines; however, it must be mentioned that only then they fail to act when the epithelium of the intestine is intact. In human cholera, however, the epithelium is destroyed by the comma bacillus, the intestinal wall thus becoming capable of absorbing the toxine which produces the general phenomena and especially a paralysis of the centres regulating circulation and heat. I believe, however, that the toxic action of the products of metabolism of the cholera vibrione does not necessarily have to be taken into consideration for the explanation of at least a part of the morbid phenomena. Do we not find, in profuse diarrhœas of a non-specific character, the picture of the general cholera phenomena point for point! Also the anatomical changes in the cholera kidneys, at least in the majority of cases, do not point to non-irritative-inflammatory changes in these organs as a result of the action of the toxine, but solely to a degeneration (coagulation necrosis, after Leyden) of the epithelia which is due to the loss of fluid and the thickened condition of the blood almost preventing the circulation in the kidneys, and which *intra vitam* so frequently shows albuminuria as a result. We may assume, therefore, that the toxine of cholera, because acting in a similar way, aggravates the effects of the loss of water and, farther on, is especially responsible for the development of complications.

Stages of the Course of Cholera.—The usual division of the course of cholera into various stages may be adhered to in a practico-diagnostic respect. The *first stage* would correspond to the usual onset of cholera with the phenomenon described above; the *second stage* (*algid stage, stadium asphycticum*), which only develops in severe cases, shows an increase of the first stage and is especially characterized by unusual cardiac asthenia, a high grade of cyanosis, icy coldness of the skin, dyspnœa, cessation of the excretion of urine and tears, and disappearance of nervous reaction. In the *third stage,* finally, in the *stage of reaction,* a general improvement in the greatly impaired functions of the body occurs, the energy of the heart increasing, the pulse becoming noticeable again and gradually more full, the coldness of the skin disappearing. Vomiting and diarrhœa cease, the stools again contain bile and assume a more fæcal appearance; urine again is secreted, at first but little, containing large quantities of albumin, later more, and then even polyuria occurs. Convalescence, however, is markedly protracted and interrupted by severe symptoms: The patients are delirious or become comatose, convulsions take place, vomiting and diarrhœa recur, exanthems appear (erythema, roseola or urticaria), as well as complications of various kinds, such as diphtheria of the pharynx, of the bladder, etc., gangrene of the skin, of the lungs, parotitis, pneumonia, pleurisy, venous thrombosis, etc. The reason of these "*reaction phenomena*" which, on account of their superficial resemblance to enteric fever, have been designated *cholera typhoid,* evidently varies greatly in the individual cases. In a large part of these, the principal source of the phenomena may be looked for in uræmia which develops subacutely; in other cases, possibly, the greater absorption of the toxines which have been formed during the course of the cholera, may be the cause of the symptoms of cholera typhoid. In other cases, again, the resumption of the functions of the central nervous system, due to the returning circulation, appears to be too stormy, and marked irritative phenomena are the result, which are accounted for by the fact that the nervous system, during the severest time of the disease, has been insufficiently nourished and for this reason has become more irritable. The occurrence of coma in cholera typhoid has recently been in part attributed to a diminution of the alkalinity of the blood —to an acid intoxication—(G. Hoppe-Seyler). The fever which accompanies some of the complications in this stage may also give rise to, or at least may produce, a more pronounced development of, the typhoid-like condition.

As in other infectious diseases, so do abortive cases occur also in cholera, besides

the well-developed forms; the separation of the mildest and mild forms of cholera (the "*cholera diarrhœas*" and the *cholerines*) from the severe forms, the "*genuine cholera attacks*," is decidedly advisable in a practical respect; the proper placing of the individual case into this or that category, however, is not easy and even unnecessary.

Important Individual Symptoms—General Appearance.—Of the symptoms important in the diagnosis of cholera some few require especial discussion.

The entire *appearance* of the patient with cholera is extremely severe, characteristic. The grayish-lead colour of the most peripheral parts of the body, the withered, sticky-moist condition of the skin, and the absence of elasticity of the latter (so that a fold of the skin which is raised between the fingers remains for some time), the icy coldness of the entire cutaneous surface (*cholera algida*), the sunken-in appearance of the face, the falling in of the bulbi into the cavities of the eyes (on account of the absent contents of water in the retrobulbar tissue), impress their stamp upon the external appearance of the cholera that is so characteristic for laymen as well as for physicians. This terror-striking appearance is materially enhanced by another symptom, which is in connection with the increase of general weakness, viz., the inability to close the eyelids between which the dry, lustreless, lower white segment of the apple of the eye appears.

Muscular Cramps, etc.—In the most varied muscles of the body, most frequently in the *muscles of the calves*, least frequently in those of the face, severe painful *tonic cramps* occur, taking place in attacks and undoubtedly due to the dryness of the tissues. As the absence of water, as has been shown experimentally, has as its immediate result an increase in nervous excitability, and as muscular cramps occur in non-specific diarrhœas in a similar manner as in the case of Asiatic cholera, there is no reason, in my opinion, to attribute the cramps to a specific action of the cholera poison alone, as has been attempted recently. This is also true of disturbances of *consciousness and deliria* which, though of rare occurrence, are seen in the course of cholera, and which, in so far as they are not manifestations of an uræmic intoxication, may, in the manner just described, be naturally referred to the dryness and to the disturbance of metabolism. The tendon reflexes appear to be increased in the severe cases.

Symptoms on the Part of Respiration and Circulation.—The *voice* becomes weak in cholera, high and hoarse; this phenomenon is due to the weakness of the laryngeal muscles but is by no means pathognomonic of cholera. *Respiration* becomes difficult, increased in rapidity and deeper, as is self-evident on account of the diminished oxygen supply (not due to the lessened respiratory capacity of the red blood corpuscles, but to the diminished circulation), and of the more marked irritation of the respiratory centre associated with it (*cholera asphyctica*). The *power of the heart* falls rapidly; functional murmurs occur; the pulse is irregular and weak and, finally, is no longer perceptible.

Condition of the Urine.—The circulation in the kidney becomes so deranged under these circumstances that the *excretion of urine* falls considerably or ceases completely. If, with increasing improvement, that is

in the stage of so-called *reaction*, a better circulation occurs in the kidney, the phenomena must now take place with which we are familiar from experiment in the transitory ligation of the renal vessels. As the epithelial cells of the glomeruli and the uriniferous tubules have become markedly damaged in their function and nutrition, owing to the impeded circulation in the attack of cholera, the blood pressure rising again in the kidney will at least in severe cases, not at once cause an increased quantity of urine; on the contrary, anuria may continue in severest cases, and death result from uræmia. In the milder cases, on the other hand, at the onset urine is secreted in small amounts, later in plentiful quantities; this, however, is shown to *contain albumin* and also, just as in the experiment, hyaline casts, which may be mixed with desquamated epithelia or fatty cells. The *amount of urea* in the urine, in keeping with the impeded function of the epithelia of the uriniferous tubules, is at first slight, gradually increases more and more, and finally becomes enormous. An actual *nephritis* which later even would become chronic, does *not* occur in the course of cholera even if leucocytes and some few red blood corpuscles are found in the urinary sediment. In those rare cases in which a typical nephritis follows an attack of cholera, this may be due to an action of the toxine, although the occurrence of nephritis may also be explained from the withdrawal of water and the disturbance of circulation as the result of the attack of cholera; this has become likely from the animal experiments undertaken in my clinic by M. Rothschild regarding nephritis after the artificial production of diarrhœa. It should be mentioned, incidentally, that the urine of cholera patients contains *large quantities of indoxyl and of sulphuric acid which in general combines with aromatic substances* (G. Hoppe-Seyler).

Vomiting, Cholera Stools.—Of greater diagnostic importance among the symptoms that have been mentioned as occurring in cholera are those affecting the *digestive tract*, especially of those of the intestines. *Vomiting* is never absent in the severer cases, especially if the patient, yielding to the great thirst, takes large quantities of fluid. The amount of the vomited material exceeds the quantity of the fluid taken in, so that the secretion of water from the blood must be thought of. The most important diagnostic symptom of cholera is and remains the *condition of the bowel*. The number of stools varies within wide limit; in rare cases diarrhœa may be absent altogether, even while the bowel is filled with fluid masses (*cholera sicca*). This, however, is quite rare; almost always there are unusually frequent diarrhœic stools, ten, twenty and more within the course of a day; in the milder cases the appearance of the diarrhœic stools is not suspicious despite the fact that cholera vibriones are contained in them. In the severe cases the stool of cholera soon loses its fæcal character entirely and takes on the notorious "rice-water-like" appearance, which, however, is not absolutely pathognomonic of the cholera stools. A *chemical* examination shows nothing characteristic; the stools contain much water, sodium chloride and mucin, little serum albumin and skatol; further, according to Kühne, almost constantly a sugar-forming ferment which is probably in connection with a supersecretion on the part of the mucous membrane

of the intestine (perhaps especially of the pancreas). Upon *microscopic* examination of the excrement many leucocytes and intestinal epithelia are found, occasionally massed in actual shreds and—as the most important constituent—the pathognomonic *comma bacilli.*

Cholera Vibriones, Comma Bacilli.—*The vibrio of cholera asiatica forms a constant constituent of cholera stools.* As is well known, R. Koch discovered this fact and has shown further that *cholera vibriones occur in no other disease,* that they are present in the intestines of cholera patients, usually in large amounts, often even in pure cultures and in the shape of short, plump, *bent* staffs (*"comma bacilli"*), which occasionally form in long, corkscrew-like round threads. *The cholera bacilli are only found in the contents of the intestine, not in the blood;* they are, therefore, in contrast to other pathogenic bacteria, *not blood parasites*—cholera is an infectious disease limited to the intestinal canal.

The comma bacilli are actively motile, grow upon gelatine, and liquefy it, so that in cultures in the test-tube, looking down from the surface, a *funnel* is formed with a thin, glassy, shiny thread reaching towards the bottom, in keeping with the point of injection.

If cultures of comma bacilli, which have been grown upon gelatine or bouillon containing peptones, are treated with chemically pure, diluted sulphuric acid, a *purple red colour* occurs in the solution (*"cholera rothreaction"*). It is due, as Salkowski found, to the simultaneous production of indol and of salts derived from potassium nitrate; the nitrous acid which becomes free by the addition of sulphuric acid gives a red colour with indol. This nitroso-indol reaction is not specific of the action of the cholera vibriones, it is also shown by other vibriones; but it is at least of value as a control for the correctness of the proof of comma bacilli.

The cholera vibriones are very susceptible to *drying.* They die exceedingly rapidly in drying, in all stages of their development. Therefore, a production of lasting spores and a distribution of cholera by the air may be excluded. Besides the drying, the *overgrowth of the comma bacilli* by saprophytes is a factor which markedly impairs the life of cholera vibriones, destroying them in a few days. This is the reason that after a few days they can no longer be found in privies, etc.

Diagnostic Value of the Comma Bacilli.—In the *diagnosis of cholera* the proof of the presence of the comma bacillus (the vibrio or spirillum choleræ) in the excrements of patients is of similarly telling importance as the finding of the tubercle bacillus for the diagnosis of tuberculosis. As tubercle bacilli are found exclusively in the last-named affection, *the cholera vibriones are found in but one disease, cholera asiatica,* and their presence in a single case is the most certain criterion of the existence of cholera. If they are absent in spite of *repeated* exact examination in the dejecta of patients suspected of having cholera, epidemic Asiatic cholera may then be excluded. By what method the vibrio choleræ finds its way into the organism, distributes itself and produces epidemics, is of minor importance in the diagnosis of the affection. However, we shall enter briefly into this question, as it must be considered in the understanding of the origin of the individual affection and, with this, indirectly, in the diagnosis.

Ætiology.—Of the requirements which have been designated by R. Koch, and which are necessary to be set up in determining certain bacteria as the specific cause of an infectious disease, the comma bacillus primarily fulfils one of them, namely,

that the comma bacillus has been found in all cases of Asiatic cholera which have been carefully examined, and exclusively so in this one affection. But also the second requirement, that by the transference of the bacterium in question to other organisms an affection resembling the original disease could be produced, may be regarded as having been fulfilled in the main. The circumstances that animals are not susceptible to cholera and that the comma bacillus is destroyed by the acids of the stomach, from the onset did not promise great success in animal experimentation. In spite of this, severe, at least cholera-like, affections in animals were produced in introducing the cholera vibrio directly into the intestine or by administering it *per os*, having previously neutralized the gastric juice by sodium carbonate. The action of the toxine produced by the cholera vibriones and causing severe phenomena of intoxication in animals, has been previously referred to. Also into the *human* subject, partly accidentally and partly by design (by v. Pettenkofer and others), have pure cultures of comma bacilli been introduced, in some instances quite enormous masses, as in the celebrated trials which v. Pettenkofer made upon himself a few years ago in which the above-described neutralization of the gastric juice was carried out. The infected individuals showed well-pronounced cholera diarrhœas in these cases of direct importation of the cholera germ, although the most severe form, which leads to death, did not occur.[1] That the latter, in spite of the enormous intake of living cholera vibriones and in spite of their enormous increase in the intestinal canal, failed to appear, and that, for example, v. Pettenkofer's general health showed no marked signs of disturbance, remains under all circumstances a conspicuous fact. To explain this, we must either assume a weakened virulence of the comma bacilli which were used, and an accidental slighter predisposition of the infected person, or, in the sense of v. Pettenkofer's cholera theory, it must be advanced that the comma bacilli can only then show their full action if a *predisposition regarding time and place* is present. With these two factors are meant, according to v. Pettenkofer's opinion, the physical condition of the earth, the changing contents of water of the same (ground-water) and its impregnation with nourishing substance suitable for lower organisms. With all evidence v. Pettenkofer showed the dependence of the origin of cholera upon the *season* (in general the maximum of cholera occurs from August to October, the minimum from March to May), and especially upon the *dampness of the earth*, so, that the development of cholera epidemics in our climate is hindered by an increase of the dampness of the earth and is furthered by its diminution. That the predisposition regarding place plays a great part is shown from the notorious immunity of certain places and, as I believe, from the experiences of the epidemic in Hamburg, in that cholera, in spite of its being carried to the most various places and even to the largest cities, did not find a firm foothold anywhere except in Hamburg and its surroundings; this may partly be easily explained by the isolation of the cholera patients and by the preventive measures taken, but, at least according to my opinion, it cannot *solely* be ascribed to these grounds. Especially prominent in the Hamburg epidemic became *the significance of the water for the spread of cholera* in that not only the population having to do with shipping along the river, and the harbour workmen were especially exposed to the infection, but also the *drinking*-water proved to be the factor which unquestionably showed the greatest influence upon the spread of cholera in Hamburg. In this regard I desire to emphasize but a few facts. The population of Altona that drank filtered water from the Elbe suffered incomparably less from cholera than the inhabitants of Hamburg who drank unfiltered water from the same river; further, a row of houses in Hamburg which obtained the filtered Altona water, and a garrison which exceptionally obtained its water from good wells, were conspicuously free from the pest. The direct proof of comma bacilli in drinking-water and especially in river water is very difficult; the cholera vibriones naturally, at the time of an occurrence of a cholera epidemic, also reach the water,

[1] An exception occurred in the unfortunate instance of Dr. Oergel, assistant in the Hygienic Institute in Hamburg, two years after the cessation of the epidemic which existed there, who, while working with cholera cultures in the laboratory, was affected by a severe form of cholera which terminated fatally.

but perish in very few days, usually under ordinary conditions. In some rare cases this proof has even been possible, the last time in a case of C. Fränkel who succeeded, in the water of the Duisburg Custom-House port, to identify comma bacilli which were originated from the dejecta of a cholera patient which were thrown there, and to cultivate them.

Differential Diagnosis.—Since the occurrence of comma bacilli in the dejecta of cholera patients has been proven with certainty, the differentiation of cholera asiatica from other affections showing similar phenomena has no great difficulties. It is true, the external picture of the affection in acute gastric and intestinal catarrhs may resemble cholera asiatica, so that such cases have acquired the name of *cholera nostras*. Also curved bacilli, similar to Koch's cholera bacilli, have been found by Finkler and Prior in the stools of patients suffering from cholera nostras. However, the Finkler bacilli differ markedly from the genuine cholera bacilli in that they are plumper and longer than these, liquefying gelatine more rapidly and more in the shape of a sac than in that of a funnel. Besides, the bacilli discovered by Finkler and Prior have lately been shown to be by no means constant constituents of the dejecta of cholera nostras and, as may be assumed to-day, are in no ætiologic connection with this affection. Besides the Finkler-Prior spirilli, quite a number of comma-bacilli-like spirilli have been discovered in the course of the last decade: The *vibrio Metschnikoff, the cheese spirilli, the vibrio Massauah, Danubicus, Berolinensis*, etc. Their differentiation from genuine cholera vibriones may be made by the observation of certain differences in a bacteriologic respect, especially also by the negative result of the specific immunity reaction according to Pfeiffer (see Preliminary Remarks, Infectious Diseases). Other bacteria, bacilli and cocci also appear to give rise to similar clinical pictures as cholera; the bacteriologic examination of the excrements must here, as always, give the decision whether they be cases of actual cholera or not. Cholera may be further confused with *certain cases of poisoning*, especially those of poisoning by arsenic, corrosive sublimate, mushrooms, etc., in that here, as well as there, intense diarrhœa and vomiting with their severe consequences predominate. Nevertheless, the absence of the comma bacilli in the diarrhœic stools which, it is true, may often resemble rice-water, offers the best proof against an attack of cholera; on the other hand, the chemical proof of the poison in the vomited material will be a certain sign of the special form of the poisoning. Finally, it need scarcely be mentioned that in practice the circumstance, above all, that Asiatic cholera is epidemic at the time or not, will be of great assistance in determining the diagnosis in the majority of cases.

MUMPS, EPIDEMIC PAROTITIS

Mumps is a true infectious disease, although opinions still differ as to the nature of the exciting cause.[1]

[1] By the majority of investigators that have lately busied themselves with finding the specific bacteria of mumps (Laveran and Cartin, Busquet, and others, lately also Bein and Michaelis) peculiar *diplococci* have been found in mumps in the exudate of

Mumps characterizes itself as an infectious disease in that it becomes epidemic without a certain external cause, that it is contagious and has a distinct period of incubation and, further, that, besides the organs which are primarily affected, the salivary glands, also certain other organs of the body become affected, and that a recovery from mumps shows a certain immunity against later affection. Regarding the *contagiousness*, this has been proven with certainty in separate cases; it was lately determined by Roth that three individual affections of parotitis occurred one after the other in the same bed or in a bed that was alongside, and, besides, that the disease was carried in one case by a third person that remained unaffected. Children under a year, as with other infectious diseases so also with mumps, are not affected; perhaps this is due to the circumstance that in nurslings the functions of the salivary glands are not yet sufficiently active. The *period of incubation* is a conspicuously long one. In the majority of cases it was over two weeks, most frequently, as it seems, over eighteen days. After eventual prodromes: Lassitude, anorexia, fever, etc., have preceded, dull pains and a sensation of tension occurs in the region of the parotid, soon also a swelling of this gland and its surroundings. As a rule, the parotid of one side is first affected, later, almost in all cases (usually in a diminished degree) also the parotid of the other side. The tumour has a hard, doughy feel and is somewhat sensitive to pressure, the skin covering it, at least usually, is not reddened. Besides enlargement of the parotid, also enlargement of the *submaxillary* and of the *sublingual* glands has been noted in various epidemics. In fact, these glands have been found enlarged in some epidemics of mumps without a swelling of the parotid. Perhaps the pancreas also takes part in the affection; at least upon pressure it was particularly painful in some of the cases. The face becomes distorted by the swelling of the parotid: The expression becomes somewhat stiff, stupid, as the mimical and head movements are particularly painful. To this are added, when opening of the mouth and movement of the jaws become difficult and painful, difficulty of speech and difficulty in chewing; fœtor of the breath also occurs. A frequent accompanying phenomenon is, further, a reddening of the mucous membranes of the cheeks and pharynx, a slight angina; the secretion of saliva is sometimes increased, at other times diminished. On the other hand, in mumps, in contrast to other infectious diseases, there is rarely *enlargement of the spleen* and *albuminuria*; although I have noted both in the course of the disease. The *fever* is also but moderate, about 102°, atypical; exceptionally the temperature may rise to 104° or 105°; in other cases the fever may be absent entirely. The duration of the affection, from the enlargement of the parotid gland to the time of the disappearance of the fever, is from one to one and a half weeks. The swelling of the parotid disappears gradually; abscess formation or even the appearance of gan-

---

the enlarged carotid gland and of the enlarged testicle, in the blood, between and in the cells of the abscess pus. It is characteristic, according to Bein and Michaelis, that the cocci of mumps show individual movements; inoculations into animals have been negative up to now.

grene are rarely present; but, if these occur, septicopyæmia may be the result.

Differential Diagnosis.—Upon noticing the symptoms described, mumps is easy to diagnosticate and can scarcely be mistaken for any other affection. Apart from the epidemic character of the disease, the almost invariable involvement of the parotid upon two sides protects us from confusing the disease with other inflammatory affections of the parotid gland. These are characterized, in contrast to mumps, in that the source of the inflammation can be demonstrated to be in the vicinity of the parotid in the form of disease of the mouth, the maxillæ, etc., or that another severe infectious disease exists, such as typhus fever, variola, measles, etc., the toxine of which as such or in combination with pyogenic bacteria gives rise to a "*metastatic*" inflammation of the gland. In these cases parotitis is a late phenomenon of the special infectious disease, so that the latter is diagnosticated long before inflammation of the parotid glands occurs as a complication. In mumps, on the contrary, the inflammation of the parotid is the first sign of the infection (in rare cases the submaxillary or sublingual glands may be affected before the parotid); the action of the infection remains limited to the parotid, whereas the other organs of the body are usually but slightly affected or not at all. In other cases, however, certain other organs are also implicated in the course of mumps, and this occurs with such conspicuous predilection that the diagnosis of parotitis on this account even gains in certainty.

Orchitis.—The most frequent and most interesting secondary localization of the mumps infection occurs in the *epididymes* and in the *testicles*. The *orchitis* (infectiosa parotidea), as a rule, appears in later stages of the affection (from the third to the fifteenth day); very rarely the opposite may be the case, i. e., parotitis may follow orchitis. In general orchitis is a quite frequent complication, occurring in at least one third of the cases. It only takes place in individuals that have reached sexual puberty and it may be ushered in by a rapidly passing urethritis, similar to a parotitis from an angina; and, as it appears, in rare cases the orchitis may be the primary localization of the mumps infection. The inflammatory swelling of the testicle is accompanied with pain in the spermatic cord, and occasionally with vomiting and exacerbation of the fever. As a rule, the affection is unilateral, rarely bilateral, so that one testicle swells after the other; it lasts about a week and a half, resulting in recovery; but in a by no means small number of the cases (almost in one half if the results of many epidemics are taken together) atrophy of the organ occurs. Besides the orchitis, a serous effusion is noted in the tunica vaginalis of the testicle and œdema of the scrotum. As an analogy to this complication of mumps in men, occasionally in women there are found—but very much less frequently—enlargement of the ovaries, vaginitis, œdema of the labia and swelling of the mammæ.

Rarer Complications.—Besides orchitis, a great number of complications occur, but, differing from the inflammation of the testicle, they are not characteristic of the mumps affection and are, therefore, of a subordinate diagnostic interest. An exception, however, is formed in the case of *affections of the ear* which consist partly

of *middle-ear diseases* (resulting from the inflammation travelling by continuity of structure from the parotid gland to the external opening and to the middle ear or even to the mastoid process), and partly of *affections of the labyrinth* which usually set in suddenly at the end of the first weeks of mumps, with tinnitus aurium and vertigo, representing anatomically serous or hæmorrhagic exudations into the labyrinth or emboli into the labyrinthian vessels, and resulting in complete deafness. Among the rare complications of mumps—pneumonia, bronchitis, endocarditis and pericarditis, peritonitis, cystitis, nephritis, arthritic inflammations, meningeal and eye affections—there should be prominently mentioned: *Meningitis*, which is comparatively most frequently the cause of a fatal outcome of mumps, but this is extremely rare; further, *inflammations of the joints* which are accompanied with pain but without swelling and redness of the joints, and, finally, disturbances of sight due to *pareses of accommodation* after mumps.

Sequelæ.—Of *sequelæ* which are in a causative relation with parotitis epidemica, besides the insufficient resorption of the swelling of the parotid gland and besides the already mentioned atrophy of the testicles, facial paralysis must be enumerated due to the pressure of the parotid tumour; further, ageusia, paralysis of the extremities (apparently of a peripheral nature with loss of electric contractility, of cutaneous and tendon reflexes), and, finally, general disturbances of the cerebral functions, insanity and hysteria.

DIPHTHERIA

Reasons of a differentio-diagnostic-didactic character have prompted me to discuss the diagnosis of diphtheria among the diseases of the pharynx and of the larynx. I refer to the diagnostic details which were there given and shall in this chapter but briefly touch upon pharyngeal and laryngeal diphtherias, in so far as their knowledge is of general importance in the diagnosis of diphtheria, and I shall consider more exhaustively the changes which arise in the different organs in the course of the affection, the complications and sequelæ, as well as the general view-points which are important in the diagnosis.

The Bacillus of Diphtheria.—The diagnosis of diphtheria to-day rests upon a firm basis, as the exciting cause of the affection is known with certainty, being recognised as a specific bacillus, the *bacillus diphtheriæ* Löffler. The bacteria are within the diphtheritic pseudo-membranes; however, they rarely go below the surface and are especially not found in the blood and internal organs of patients with diphtheria. They represent thick wedge-shaped bacilli, with usually butt-like thickened ends, and stain according to Gram. They do not show spore formation, are immotile and grow but poorly at low temperature (under 20° C.). They do not liquefy gelatine in culture; upon glycerine agar the colonies are of a transparent, gray colour, and upon slight magnification are characteristically granular. It is especially easy to grow them upon blood serum, but they also flourish on sterilized milk. Their inoculation is especially successful in birds, guinea-pigs, rabbits, cats, horses, etc. (not in mice). Besides the grayish-white membranes which develop at the point of inoculation, paralyses and disturbances of co-ordination similar to the paralyses which occur in the course of human diphtheria are observed in inoculated animals.

As the bacillus of diphtheria remains principally upon the surface, i. e., does not find its way to the internal structures of the body, it must be assumed at the onset that the *severe general phenomena* occurring in diphtheria are the secondary toxic results of the diphtheria bacilli. In fact, various investigators have succeeded in isolating from bouillon cultures of

diphtheria bacilli chemical products of metabolism which, having been freed from the bacteria by filtration and subcutaneously injected into animals or into the blood vessels, have proven themselves to be highly toxic. As a result of the infection with these toxines there have arisen: Pleurisy, nephritis, fatty degeneration of the liver, but, above all, in the later course, paralysis, and, in subcutaneous injection, brawny œdema of the cutaneous covering; in short, all the various symptoms which occur upon the inoculation of living bacilli, with the single exception of the pseudo-membranes.

Other Cocci Found besides the Bacilli.—Besides the specific bacilli, other pathogenic micro-organisms are found in recent cases of human diphtheria, *streptococci* and *staphylococci*, also *pneumococci*, which, in contrast to the diphtheria bacilli, deeply invade the tissues and by means of the circulation are carried into the most varied internal organs where they form colonies (especially also in the endocardium). Apparently their entrance into the body and their septic-infectious, inflammatory activity is markedly assisted by the preparatorily damaged condition of the tissue, the result of the diphtheria toxine. A part of the symptoms of diphtheria, especially of the malignant forms of the disease, is in the main due to this *mixed infection.*

Cases of diphtheria occur sporadically or, as is most usual, in an epidemic form. The most frequently occurring mode of infection is that particles of the diphtheritic membrane, being expectorated from the mouth, are either directly contagious, or are carried by a third person and by fomites, perhaps also by some food products (especially milk). As a rule, the bacilli primarily lodge upon the *mucous membrane of the pharynx* and here, after a brief incubation, probably in that the poison produced by them affects the vessels and gives rise to exudation, cause the well-known *pseudo-membranes.* Simultaneously a *general infection* shows itself, which may be looked upon as a *secondary* effect of the bacilli, i. e., as the action of the toxine produced by them. In those cases in which the general effect becomes manifest before any changes occur in the mucous membrane of the pharynx, it must be assumed that toxines have been rapidly absorbed, and their action has taken place before the local necrosis of the tissues is noticed. That such cases exceptionally occur, I can say with absolute certainty, having observed them in my own experience.

Diphtheria of the Respiratory Tract.—Whereas, as has already been mentioned, the pharyngeal mucous membrane is almost always the first point of attack for the diphtheria bacilli, and from this point eventually a spreading by continuity to the nasal mucous membrane and, above all, to the laryngeal mucous membrane occurs (*descending laryngeal diphtheria*), finding its way into the trachea and into the bronchi, in rarer cases a *primary* diphtheria of the *nasal mucous membrane* is met with, or, relatively more frequently, of the larynx. The affection may be limited to the larynx (similar to the limitation of pharyngeal diphtheria to the structures of the pharynx) (*laryngeal croup,*[1] *primary laryngeal diph-*

[1] The designation "croup" is an *anatomical* conception; by this term is understood the formation of fibrine exudates upon the free upper surface of the mucous membrane, without extensive damage to the underlying tissue, whereas in diphtheria in the anatomical sense the mucous and eventually the submucous tissue is also

theria), or it may only spread downward to the trachea or, again, as is unquestionably sometimes seen, it may spread upward, affecting the pharynx (*ascending laryngeal diphtheria*) and in such cases the doubt vanishes whether pseudo-croup or genuine laryngeal diphtheria is the disease in question.

Diphtheria of the Digestive Tract.—But very rarely do the diphtheria bacilli find their way into the œsophagus and stomach. It appears that the close approximation of the plate of the cricoid cartilage to the vertebral column is a natural obstacle to the advancement of the bacilli by this road. But primary and secondary diphtheria of the œsophagus is observed exceptionally. *Diphtheria of the stomach* has also occasionally been noted in the form of a flaky white infiltration of the gastric mucous membrane, which, however, up till now is insusceptible to diagnosis during life, as even in the act of vomiting of pseudo-membranes it is impossible to determine whether they originate from the larynx or from the œsophagus.

Individual Varieties of Diphtheria.—After a period of incubation of from two to seven days, rarely longer, *fever* occurs, often accompanied with vomiting and preceded by chilliness or a decided chill; headache, anorexia, etc., are also present. At the same time, occasionally somewhat later, disorders arise pointing to the affected area: Difficulty in deglutition or hoarseness with a barking cough, and pains in the throat. Examination of the pharynx or larynx at this time either shows a simple, apparently unsuspicious catarrh (which in rare cases may not advance beyond this stage in the entire course of the affection), or the characteristic appearance of the *membranes* may already be noticed. If these are desquamated, new masses of membrane form; simultaneously the originally small plaques enlarge rapidly, spreading in breadth and depth. The severest form of pharyngeal diphtheria is the *septic variety*, which is due to the simultaneous action of streptococci and of saprophytes, which colonize with the diphtheria bacilli in the membranes. By their action the *diphtheritic parts* are changed into stinking, pulpous, black, gangrenous masses, and severe cardiac collapse rapidly shows itself. In all three varieties, the *simple diphtheritic pharyngeal catarrh*, the *well-developed fibrinous*, and the *septic pharyngeal diphtherias*, the *submaxillary glands are enlarged and painful*. It can but *rarely be shown that the spleen and the liver are enlarged*. The *temperature* varies considerably in individual cases: at one time it is but scarcely raised, at other times it may be 104° F. and over. There is no proportion between the height of the fever and the severity of the case; as in the severest forms we often note conspicuously low temperatures. More often is the *pulse* an indication of the gravity of the case; a small, weak, or irregular pulse shows a severe infection, abnormal bradycardia (40 beats and less) is also noted in the severer forms of pharyngeal diphtheria.

---

affected and becomes necrotic. Experience has taught that laryngeal croup may also be due to chemical or thermic irritants; but these are clinical rarities, so that it is advisable to designate such forms of laryngitis as "*croupous laryngitis*" and separate them entirely from "*epidemic laryngeal croup.*" More correctly even it appears to me, from a clinical standpoint, to drop the name "epidemic laryngeal croup" entirely and to substitute the designation "*primary laryngeal diphtheria.*"

Laryngeal diphtheria runs its course with the same general phenomena, only that here the well-known symptoms of an increasing stenosis of the glottis, of an essentially *inspiratory stenotic character,* become prominent. The patient makes enormous efforts to overcome the respiratory hindrance, the supraclavicular fossa and the epigastrium sink in during inspiration; the deeper, generally quite toneless, whispering voice, the barking cough, following in short gasps and interrupted by long-drawn, whistling inspirations, and the increasing carbonic-acid intoxication complete the picture. It is but rarely possible to make a laryngoscopic examination; if it is successfully done, the white covering in the larynx and the immobility of the vocal cords are noted. Occasionally the dyspnœa increases to attacks of greatest orthopnœa, especially when the pseudo-membranes became loosened and mechanically obstruct the glottis. It is important both from a diagnostic and prognostic respect always to examine the condition of the lungs in the case of existing laryngeal diphtheria. Acute hæmorrhages occur here and, if the diphtheritic process travels into the bronchi, symptoms of atelectasis or broncho-pneumonia. The origin of pneumonia in diphtheria is partly due to the just-mentioned dissemination of the diphtheritic process to the alveoli, with proliferation of the diphtheria bacilli in the infiltrates, partly also to a damaging of the pulmonary tissue by the diphtheria toxine, which favours the development and activity of pneumococci or streptococci. If the presence of a pneumonia can be determined, i. e., dulness, bronchial breathing, etc., without it being possible, by change of position and deep inspiration, to compensate the physical changes, the prognosis in general and especially the prospect of improvement of the phenomena by a tracheotomy are rendered very unfavourable, and especially for this reason the diagnosis must pay special attention to these conditions. Laryngeal diphtheria may be secondary or primary, and in the latter case, as has already been indicated, may remain limited to the larynx or, by ascending, give rise to a pharyngeal diphtheria. It is, therefore, indicated under all circumstances, according to my experience, at once to isolate children with phenomena of an acute laryngeal stenosis in whom the larynx cannot be examined, and not to wait until the diagnosis becomes certain, unless we wish to risk that the other inhabitants of the room may be infected by diphtheria.

Diphtheria of the Nose and its Adnexa.—A dissemination of the diphtheritic process upward causes *diphtheria of the mucous membrane of the nose.* This is easily diagnosticated: The patients force the air with a noise that may readily be heard, through the narrowed nasal channels, accompanied with the expulsion of brownish-necrotic masses from the nasal openings; inspection shows diphtheritic deposits and necrotic tissue changes in the posterior nares and in the internal parts of the nose; in some cases the process may even reach the skin of the external nasal openings and the upper lip, so that the affected areas appear reddened, ulcerated, and diphtheritic. In other cases the diphtheritic process finds its way into the *Eustachian tube* and into the *tympanic cavity*; the action of streptococci may produce *bone abscesses,* also *meningitis* and *cerebral abscess.* The *conjunctivæ* and *genitalia* or *accidentally existing wounds* may also show a diphtheritic deposit, caused either by a direct propagation of the process or by auto-inoculation with the poison by the patient himself.

theria), or it may only spread downward to the trachea or, again, as is unquestionably sometimes seen, it may spread upward, affecting the pharynx (*ascending laryngeal diphtheria*) and in such cases the doubt vanishes whether pseudo-croup or genuine laryngeal diphtheria is the disease in question.

Diphtheria of the Digestive Tract.—But very rarely do the diphtheria bacilli find their way into the œsophagus and stomach. It appears that the close approximation of the plate of the cricoid cartilage to the vertebral column is a natural obstacle to the advancement of the bacilli by this road. But primary and secondary diphtheria of the œsophagus is observed exceptionally. *Diphtheria of the stomach* has also occasionally been noted in the form of a flaky white infiltration of the gastric mucous membrane, which, however, up till now is insusceptible to diagnosis during life, as even in the act of vomiting of pseudo-membranes it is impossible to determine whether they originate from the larynx or from the œsophagus.

Individual Varieties of Diphtheria.—After a period of incubation of from two to seven days, rarely longer, *fever* occurs, often accompanied with vomiting and preceded by chilliness or a decided chill; headache, anorexia, etc., are also present. At the same time, occasionally somewhat later, disorders arise pointing to the affected area: Difficulty in deglutition or hoarseness with a barking cough, and pains in the throat. Examination of the pharynx or larynx at this time either shows a simple, apparently unsuspicious catarrh (which in rare cases may not advance beyond this stage in the entire course of the affection), or the characteristic appearance of the *membranes* may already be noticed. If these are desquamated, new masses of membrane form; simultaneously the originally small plaques enlarge rapidly, spreading in breadth and depth. The severest form of pharyngeal diphtheria is the *septic variety*, which is due to the simultaneous action of streptococci and of saprophytes, which colonize with the diphtheria bacilli in the membranes. By their action the *diphtheritic parts* are changed into stinking, pulpous, black, gangrenous masses, and severe cardiac collapse rapidly shows itself. In all three varieties, the *simple diphtheritic pharyngeal catarrh*, the *well-developed fibrinous*, and the *septic pharyngeal diphtherias*, the *submaxillary glands are enlarged and painful*. It can but *rarely be shown that the spleen and the liver are enlarged*. The *temperature* varies considerably in individual cases: at one time it is but scarcely raised, at other times it may be 104° F. and over. There is no proportion between the height of the fever and the severity of the case; as in the severest forms we often note conspicuously low temperatures. More often is the *pulse* an indication of the gravity of the case; a small, weak, or irregular pulse shows a severe infection, abnormal bradycardia (40 beats and less) is also noted in the severer forms of pharyngeal diphtheria.

---

affected and becomes necrotic. Experience has taught that laryngeal croup may also be due to chemical or thermic irritants; but these are clinical rarities, so that it is advisable to designate such forms of laryngitis as "*croupous laryngitis*" and separate them entirely from "*epidemic laryngeal croup.*" More correctly even it appears to me, from a clinical standpoint, to drop the name "epidemic laryngeal croup" entirely and to substitute the designation "*primary laryngeal diphtheria.*"

the nuclei of the nerves proved to be normal. We may assume in general that the toxine of diphtheria shows no great affinity for the protoplasm of the nerves and surely acts only very slowly upon it with its toxophorous group, so that in the individual case either no affection of the nervous system occurs at all, or degenerative-neuritic changes develop only late in the course of the disease. The latter are at times localized in the *peripheral* nerves, at other times in the *spinal cord*, and motor centres most frequently in the anterior columns and cells of the anterior horn, from which secondary degenerative changes will then develop in the peripheral nerves. According to the mode of action just described, the paralytic phenomena may occur from step to step. In rare cases a spinal meningitis with its results seems to give rise to the paralyses.

Acute ataxia has occasionally been observed by myself and others after diphtheria, analogous to the disturbances of co-ordination which are produced artificially in animals after the inoculation of diphtheria bacilli as well as after the injection of diphtheria toxines freed from bacilli. The picture of ataxia may completely resemble tabes, although some symptoms, for example the reflex rigidity of the pupils, are absent. Most probably this is due to a toxic change in the posterior roots and in the posterior columns of the spinal cord, as the *sensory sphere* may in general be affected, although frequently to a less marked extent than the motor sphere, after diphtheria. Nervous disturbances originating in the brain, such as hemiplegia, aphasia, epilepsy and mania, have been noted in only very few isolated cases. Characteristic of these diphtheritic paralyses, and on this account also determining the diagnosis, is the *benign course* of all these paralyses which at first sight may appear very severe.

Differential Diagnosis.—It is unnecessary to enter further into the *differential diagnosis*. The differentiation of pharyngeal and laryngeal diphtheria from other pharyngeal and laryngeal affections has been enlarged upon in discussing the diagnosis of the individual affections of the pharynx and larynx. In *questionable* cases the examination of the membranes for the Löffler bacillus and testing their virulence by inoculation into guinea-pigs are imperative in order to reach a certain opinion. Practically it is advisable under such circumstances, even before the diphtheria bacilli have been demonstrated, to isolate the patients; an expectant diagnostic course and too long a wavering in diagnosis often avenges itself in a dreadful manner! The possibility of contagion in diphtheria exists during the entire course of the affection; yes, even for many weeks after the pharyngeal mucous membrane has been completely cleansed, virulent diphtheria bacilli could be demonstrated in the tonsils!

The above-stated characteristics of the diphtheria bacilli: The wedge-shaped form, the immovability of the bacilli, their staining according to Gram, their difficult growth at low temperature and the granular appearance of the cultures, etc., differentiate them easily from other bacteria, but not from *pseudo-diphtheria bacilli* which are frequently found in healthy persons as well as in the sick, especially in the pharynx of the healthy and in diphtheritic membranes, in the nasal cavity and upon the normal and also upon the diseased conjunctivæ ("xerous bacilli"), in abscesses, etc. Sharp morphological differences between both varieties of bacilli have not been determined; the only telling difference is that the *pseudo-diphtheria bacilli do not act pathogenically* in animals (not even in guinea-pigs), i. e., do not give rise to either local or general disturbances. Their biological position to the genuine virul-
theria bacilli has not yet been determined. The possibility that the ps

Complications.—Of the so-called *complications*, the origin of which may for the most part be explained from the fact that the absorbed diphtheria toxine prepares the soil for the simultaneous action of the streptococci as inflammatory or pyogenic agents, the most important are: *Angina Ludovici, endocarditis* and *pericarditis, polyarthritis, parotitis, peritonitis,* etc.; however, these complications are all relatively rare. In some cases the complete symptom-complex of septicopyæmia develops, with chills and multiple metastases.

More important, because more frequent, are certain accompanying phenomena of diphtheria, which are obviously due to a more intense action of the diphtheria toxine which has been absorbed in greater amount. These are the phenomena on the part of the heart, of the kidneys and of the nervous system. By means of the *intoxication of the heart* subacute cardiac asthenia occurs and frequently acute paralysis of the heart with syncope or sudden death, which may even take place during convalescence and, as autopsies teach us, is due to a parenchymatous-interstitial, toxic myocarditis of a high grade. The occurrence of *albuminuria* is very common; in contrast to the nephritis of scarlet fever it may be noticed on the first day of the disease. According to my opinion and experience, provided no very distinct symptoms of engorgement as a result of the poor cardiac activity are simultaneously present, it is to be looked upon solely as a phenomenon of intoxication, the result of irritation of the kidney by the diphtheria toxine. If this irritation increases to a certain height, epithelial casts and blood will be noticed in the urine, besides the albumin; in short, a well-developed picture of an acute *nephritis* appears. Anasarca, according to my experience, is scarcely ever present, and chronic nephritis as a result of acute nephritis is extraordinarily rare; I have never seen it, as frequently even as I have noted a nephritis following diphtheria, but always saw the albumin disappear during convalescence, although often it took months until this was accomplished. The most interesting of all the effects of the toxines of diphtheria are the *paralyses* which present themselves usually only weeks after diphtheria has run its course and which may affect the most various organs. Most frequent is *paralysis of the palate,* showing itself by nasal speech and regurgitation of fluid through the nose upon swallowing. It is easy to determine this upon noting the immobility of the soft palate in phonation. Not rare are. further, *paralyses of the extremities and of the facial muscles, of the muscle of the eye, of the muscles of the apparatus of accommodation, of the musculatures of the larynx and of respiration of the sphincters of the bladder and of the rectum.*

Character of Diphtheritic Paralyses.—The question whether these paralyses are of a peripheral or of a central origin has been variously discussed. According to the clinical material at our command at present, these paralyses, as a rule, are of *peripheral* origin, i. e., flaccid paralyses with a diminution or loss of the tendon reflexes, eventually after a preceding brief period of increase of the same. The affected muscles show inclination to atrophy and, under some circumstances, reaction of degeneration. Post-mortem findings showed in some of the cases changes in the spinal cord, especially atrophic degeneration of the anterior horns and of the fibres of the anterior roots; in other cases exquisite neuritic changes were present, whereas

the nuclei of the nerves proved to be normal. We may assume in general that the toxine of diphtheria shows no great affinity for the protoplasm of the nerves and surely acts only very slowly upon it with its toxophorous group, so that in the individual case either no affection of the nervous system occurs at all, or degenerative-neuritic changes develop only late in the course of the disease. The latter are at times localized in the *peripheral* nerves, at other times in the *spinal cord*, and motor centres most frequently in the anterior columns and cells of the anterior horn, from which secondary degenerative changes will then develop in the peripheral nerves. According to the mode of action just described, the paralytic phenomena may occur from step to step. In rare cases a spinal meningitis with its results seems to give rise to the paralyses.

Acute ataxia has occasionally been observed by myself and others after diphtheria, analogous to the disturbances of co-ordination which are produced artificially in animals after the inoculation of diphtheria bacilli as well as after the injection of diphtheria toxines freed from bacilli. The picture of ataxia may completely resemble tabes, although some symptoms, for example the reflex rigidity of the pupils, are absent. Most probably this is due to a toxic change in the posterior roots and in the posterior columns of the spinal cord, as the *sensory sphere* may in general be affected, although frequently to a less marked extent than the motor sphere, after diphtheria. Nervous disturbances originating in the brain, such as hemiplegia, aphasia, epilepsy and mania, have been noted in only very few isolated cases. Characteristic of these diphtheritic paralyses, and on this account also determining the diagnosis, is the *benign course* of all these paralyses which at first sight may appear very severe.

Differential Diagnosis.—It is unnecessary to enter further into the *differential diagnosis*. The differentiation of pharyngeal and laryngeal diphtheria from other pharyngeal and laryngeal affections has been enlarged upon in discussing the diagnosis of the individual affections of the pharynx and larynx. In *questionable* cases the examination of the membranes for the Löffler bacillus and testing their virulence by inoculation into guinea-pigs are imperative in order to reach a certain opinion. Practically it is advisable under such circumstances, even before the diphtheria bacilli have been demonstrated, to isolate the patients; an expectant diagnostic course and too long a wavering in diagnosis often avenges itself in a dreadful manner! The possibility of contagion in diphtheria exists during the entire course of the affection; yes, even for many weeks after the pharyngeal mucous membrane has been completely cleansed, virulent diphtheria bacilli could be demonstrated in the tonsils!

The above-stated characteristics of the diphtheria bacilli: The wedge-shaped form, the immovability of the bacilli, their staining according to Gram, their difficult growth at low temperature and the granular appearance of the cultures, etc., differentiate them easily from other bacteria, but not from *pseudo-diphtheria bacilli* which are frequently found in healthy persons as well as in the sick, especially in the pharynx of the healthy and in diphtheritic membranes, in the nasal cavity and upon the normal and also upon the diseased conjunctivæ ("xerous bacilli"), in abscesses, etc. Sharp morphological differences between both varieties of bacilli have not been determined; the only telling difference is that the *pseudo-diphtheria bacilli do not act pathogenically* in animals (not even in guinea-pigs), i. e., do not give rise to either local or general disturbances. Their biological position to the genuine virulent diphtheria bacilli has not yet been determined. The possibility that the pseudo-diph-

theria bacillus is a diphtheria bacillus deprived of its virulence, as has been assumed by Roux and others, cannot, in my opinion, be put aside at present, according to the results of investigations reported so far. Between the markedly virulent bacilli in severe diphtheria and the absolutely benign pseudo-diphtheria bacilli, many transitional forms are found, i. e., bacilli of weak virulence which must be looked upon as attenuated diphtheria bacilli. The latter appear to be present in the mild and favourable cases of diphtheria; but, unquestionably, the cases are very rare in which, in apparently true croup, respectively diphtheria membranes, both, the virulent and the non-virulent side by side, cannot be demonstrated, but only the latter, the pseudo-diphtheria bacilli. In general we may still adhere to the postulate: *If in those cases which clinically present themselves as diphtheria, bacilli are found having the appearance of the Löffler bacillus, and if they are present in great amounts, that they are also virulent, i. e., genuine diphtheria is the diagnosis of the case in question.*

PERTUSSIS; TUSSIS CONVULSIVA, WHOOPING-COUGH

The diagnosis of whooping-cough, after the disease has reached its acme, can readily be made with certainty. At the onset of the affection, however, this cannot be done; it remains questionable rather until that stage is reached in which the characteristic attacks of cough occur. The division of the disease into three stages, which has been the custom since ancient times, is valuable in a diagnostic respect, although we must always remember that one stage gradually and imperceptibly merges into the other, and the diagnostic division of these stages is only of practical value.

Catarrhal Stage.—The symptoms of this stage are those of a catarrh of the respiratory mucous membrane and its adnexa. After a period of incubation of about a week, which runs its course without symptoms, there occur at first conjunctivitis, coryza, and possibly also a slight pharyngitis; this is supervened by mild hoarseness and cough, whereas the symptoms of sneezing, difficulty in swallowing and the flow of tears become less marked. After a duration of several weeks (about three), the cough becomes spasmodic; with this the disease reaches its characteristic stage.

Convulsive Stage.—*Stadium convulsivum s. spasmodicum*: The attacks of cough are ushered in by a sensation of tickling in the larynx; the children—who are principally affected, as is well known—feel the attack coming on but are unable to suppress it and show in their entire appearance the expression of great fear on account of the threatening attack. Now the cough occurs; it shows itself in short paroxysms, rapidly succeeding each other, frequently of twenty or more expiratory excursus, which are finally followed by a long-drawn whistling inspiration ("reprise"); this is again succeeded by the expiratory cough paroxysm. These attacks shake the entire body, the face becomes deeply cyanotic ("blue cough"). The bulbi stand out prominently from the cavities of the eyes, the skin becomes covered with sweat; urine, flatus and fæces are voided involuntarily. Usually the attack ceases with the expectoration of a tough, glassy mucus which may often be evacuated with actual vomiting movements. The duration of the individual attack varies greatly as to time—from scarcely a minute to about a quarter of an hour; the number of attacks in twenty-four hours is also variable, twenty to a hundred! The

cause for the production of the attacks may occasionally be plainly noted; they are especially due to mental emotions, to movements of deglutition or laughter, to initial choking movements upon pressing down the posterior part of the tongue with a tongue depressor, etc., above all, however, to accumulations of mucus in the larynx.

Causation of the Attack; Nature of the Disease.—Von Herff, by means of autolaryngoscopy, has observed that upon the mucous membrane of the posterior laryngeal wall, at the level of the glottis, there appears from time to time a small particle of mucus, the formation of which is in direct connection with the cause of the attack. If this was removed by forcible expiration, the attack would stop. At the same time v. Herff, as did also, previous to him, Meyer-Hüni, observed a mild grade of inflammation of the respiratory mucous membrane from the posterior nares down to the bifurcation, which was not diffusely spread in the larynx but was especially concentrated upon the posterior surface of the epiglottis, in the interarytænoid and in the infraglottic regions. Mechanical irritation of these parts in the interior of the larynx regularly produced a typical attack of whooping-cough. Rossbach, in contrast to these observations, did not observe any apparent catarrh of the pharynx, of the larynx and of the trachea, and only admits the existence of a catarrh of the larger bronchial tubes as the essential anatomical substratum of the disease. Formerly an enlargement of the tracheo-bronchial lymph glands was supposed to be an important anatomical change in whooping-cough, in that this would exert a pressure upon the pneumogastric nerve, respectively upon the recurrent laryngeal nerve, and in this way cause the attack. It was found, however, in succeeding times, that the intumescence of the bronchial glands was by no means a constant phenomena of whooping-cough, as it actually occurs frequently without pertussis being present at all. That whooping-cough is a very peculiar affection and cannot be explained as a simple catarrh of the respiratory passages with exaggerated sensitiveness of the nerves of the mucous membrane, is unquestioned for every one that has had an opportunity to observe pertussis frequently and to compare it with other affections of the respiratory organs, which are characterized by a milder or more marked irritability of the mucous membrane. The attacks are the pathognomonic symptom of the disease and occur in no other affection as pronouncedly as they do in the case of pertussis. It is a decided infectious disease which, as has been shown by thousands of observations, is *contagious*, the nature of which, however, can only be determined when we succeed in finding the virus which is at the base of this affection. This is not due to the fact that no investigations have been made in this respect; bacilli, cocci and amœbæ have been supposed to be the specific cause of the disease. However, unquestioned results in the attempt to isolate the toxine have not been obtained, and the bacillus which has been allegedly regularly found by various authors in the glassy expectoration of whooping-cough patients, has by no means been generally accepted. *How* contagion with the toxine of pertussis occurs and how its action in the body may be explained, had better not be analyzed for the present before we have definitely succeeded in finding the specific microorganism of pertussis and have investigated its toxic properties. This much appears to be certain, that under the influence of the infection an increased irritability of the superior laryngeal nerves is brought about and that irritation of certain areas of the respiratory mucous membrane, especially of the interarytænoid region in its lower parts, causes, by mechanical or chemical irritants (accumulation of mucus, ammonia, etc.), the attacks of coughing to appear. That these attacks occur at night in deepest sleep, and are then more marked than in daytime, is a well-known fact which may be explained in that the accumulating mucus under these circumstances requires a longer time to produce the necessary degree of irritation of the centripetally conducting nerves and nerve centres which release cough, their irritability being considerably lessened during sleep. If the paroxysm, however, has once begun, it is more intense and lasts longer, as the virus which has accumulated in greater amounts irritates the nerves much more powerfully upon the awakening of the patient when they again become more irritable.

Results of the Attacks of Cough Paroxysm.—The intense attacks of cough result in some phenomena, the observation of which may materially aid in arriving at a diagnosis if there be no opportunity personally to observe the individual paroxysms of cough. *Vomiting* must be especially mentioned among these symptoms, which follows the paroxysms; this occurs also in other severe coughs, but is decidedly most frequent and most stubborn in whooping-cough. Occasionally alveoli burst, and *interstitial emphysema, pneumothorax* or *cutaneous emphysema* may develop; or—as is quite common—blood-vessels may burst in the body during the attack. This gives rise to ecchymoses of the skin, and, above all, to *subconjunctival hæmorrhages,* rarely retinal hæmorrhages, bleeding from the nose, the external auditory canal, hæmorrhages into the substance of the brain or into the *meninges* with their consequences: Aphasia, hemianæsthesia and hemiplegia; subsequent to the latter condition I saw in a child a state which resembled idiocy but which, after existing for several months, gave place to normal cerebral activity! Herniæ and prolapsus ani may also develop in the course of the paroxysms of cough; further, as a result of the excessive exertion of the abdominal muscles, these may become painful, and dizziness, vertigo and general lassitude may remain. Especially great diagnostic value is placed upon the development of *ulcers under the frenum of the tongue*; not without reason, as these sublingual ulcerations are really only noted in the course of whooping-cough. They are due to *traumatism* in that the incisor teeth, during the paroxysm of cough, rub against the tongue which is forcibly stretched forward and held out; therefore, in toothless individuals and also in mild cases of whooping-cough these ulcers are absent. The general engorgement may give rise, further, to transitory *albuminuria,* to *acute dilatation of the right heart* and to an irregular, small pulse.

Stage of Decline.—With the cessation of the attacks of cough, after the convulsive stage has lasted about six weeks (four to six weeks and longer), the affection passes into the last stage, the *stadium decrementi* (s. criticum): The attacks become less frequent and lose their convulsive character; the sputum becomes thinner—fluid, muco-purulent and looser, till the disease has reached its end after a few more weeks (about three).

Complications.—Whooping-cough does not show many *complications,* as it is an infectious disease which, for the most part, remains local, and complications due to a general infection are, therefore, not present. Thus, for instance, frequently during the entire course of the disease, especially at the time of acme, there are absent: The fever, the infectious nephritis, the irritation of the central nervous system, etc. On the other hand, complications affecting the respiratory tract are comparatively frequent, remaining as sequelæ for some time and in their results may be exceedingly dangerous. The most frequent of these complications of whooping-cough is *bronchopneumonia,* which, to conclude from its intensity and perniciousness in little children (children under one year almost invariably succumb), is of a specific character, in that unquestionably the virus extends into the alveoli; that the bronchitis, by clogging the finer bronchi with mucus, favours the development of pneumonia is self-evident. The occurrence of fever, dyspnœa during the intervals, dulness, etc., scarcely allow us to remain in doubt as to the appearance of this complication. Rarer are complications on the part of the larynx, such as spasm *of the glottis* due to an increase *ad maximum* of the reflex irritability of the laryngeal

nerves. Among the *sequelæ* there must be mentioned: The general languishing condition which in severe cases of whooping-cough is added, produced primarily by the inanition, respectively the frequent vomiting; *pulmonary tuberculosis; emphysema*, which arises as a result of the enormous expiratory efforts with closed glottis; goître, etc. Whether *endocarditis, pericarditis,* and *nephritis,* which are now and then observed in whooping-cough, are in any direct genetic connection with the original affection, is very questionable; this is also true of the alteration of the mental condition of patients with pertussis. A lasting melancholic condition is quite evident in such a stubborn, teasing affection, and in some instances this may increase to actual melancholia.

Differential Diagnosis.—If whooping-cough is typically developed, it cannot be confused with any other disease. However, in the first stage the decision whether whooping-cough or simple bronchitis is present is absolutely impossible; this is also true of the third stage. Provisional diagnoses in both of these stages are based—in the first stage upon the history or upon determining the possibility of contagion, in the third stage upon the description of the attacks of cough which have preceded, upon the residues of ulcers of the frenum, etc.; naturally, these diagnoses are of no great value.

CEREBRO-SPINAL FEVER—EPIDEMIC CEREBRO-SPINAL MENINGITIS

The diagnosis of cerebro-spinal meningitis is an essentially *ætiological* one. The differentiation based solely upon the symptomatology of epidemic cerebro-spinal meningitis from a non-epidemic variety is *impossible,* even if the presence of certain symptoms is in general more in favour of an epidemic cerebro-spinal meningitis. In referring, therefore, to the former discussion of the diagnosis of meningitis (which see), I shall here but briefly recapitulate the phenomena which are most important for the diagnosis and shall mention only those symptoms more exhaustively which are in the main in favour of epidemic cerebro-spinal meningitis in contrast to other forms of meningitis.

Important Diagnostic Symptoms.—The disease begins with rather vague *prodromal phenomena*: General malaise, lassitude, anorexia, slight fever, headache, wandering pains, pharyngitis, etc., or, much more frequently, it sets in at once with severe symptoms with several chills and with signs directly pointing to the seat of the affection being in the central nervous system, i. e., with *severe headache, vertigo* and *vomiting.* To this is added general *hyperæsthesia* of the skin and of the special organs of sense, and usually, from the second day on, the ominous *rigidity of the muscles of the neck.* The patients hold their heads stiff and complain of severe pains upon the slightest attempt to move them. In a part of the cases this tonic muscular spasm extends to the extensors of the vertebral column, so that opisthotonus results; contractures of other muscles, especially trismus, are rarer.

The *reason for the rigidity of the muscles of the neck* is not clear; the simplest and most natural explanation is that it may be looked upon as an irritative symptom of the outpassing nerves. But cases occur in which, in spite of well-developed spinal meningitis, rigidity of the muscles of the neck is absent, and,

may be very pronounced, and yet, as my experience has taught me, no meningitis found post mortem. Upon the whole, however, these are rare exceptions, and rigidity of the muscles of the neck is a most constant symptom of cervical meningitis especially if the lateral movements of the spinal column are sensitive at the same time and if pressure or a slight blow upon the spinous processes produces pain.

Tonic contraction of the muscles of the abdomen is the cause of the very frequently present, diagnostically important, "scaphoid" retraction of the abdomen; but contraction of the intestines as a result of irritation of the pneumogastric nerve may also be concerned in this phenomenon. Besides the scaphoid retraction of the abdomen there is usually constipation. *Excentric pains in the extremities* are now and then markedly developed, as well as chronic cramps of the extremities; these also occur in the region of the facial nerve. Paralyses become prominent in the later course of cerebro-spinal meningitis: Paralysis of the abducens, ptosis, paralysis of the tongue, anarthria, monoplegia, hemiplegia, and paraplegia which are either of central origin (hemiplegia or monoplegia with aphasia) or of a peripheral character, as a result of the pressure of the exudate upon the outpassing nerve trunks; anæsthesia is also occasionally noted.

A greatly overestimated symptom occurs, as in the usual form of meningitis, so also in cerebro-spinal meningitis, the hydrencephalic cry. The patients become confused, somnolent, delirious, or fall into deep coma. involuntary evacuation of urine and fæces takes place or the urine may collect in the bladder as the result of a spasm of the sphincter or due paralysis of the detrusor. As a rule, the *excretion of urine* is scanty; other cases, on the contrary, polyuria exists. This latter symptom, as well as melituria, which has also been noted, are the results of central irritation in the region of the medulla oblongata; albuminuria in some of the cases is undoubtedly also due to the same cause, as its intensity, as I have noted, is in proportion to the severity of the nervous symptoms and independent of the course of the fever. The *pupils* are contracted, as a rule; however this is changeable and very frequently they show normal conditions during the entire course of the affection. It is of diagnostic importance that *they become unequal under the eyes of the physician*; an ophthalmoscopic examination will reveal the changes in the eye-ground which have been previously described (see p. 706), especially optic neuritis, etc. The respiration shows the Cheyne-Stokes type in some few cases but by no means often. *Eruptions* are very common upon the *skin*: Petechiæ, roseola, erythema, etc.; however, none is as frequent as *herpes*—there are epidemics in which almost all cases show herpes. This most often occurs from the second to the sixth day on the face, less frequently on the extremities. It is somewhat characteristic that herpes is not infrequently bilateral, that the entire face appears to be uniformly covered with it, or that corresponding halves of the body are affected by herpes, as, for example, one of my cases in which both thumbs were affected. Without question herpes is a trophoneurotic phenomenon dependent upon an irritation of the exudate of the intervertebral ganglia and of the nerve roots. However, it appears to me that herpes is much more common in *epidemic* cerebro-spinal meningitis than in meningitis due to other causes, and the ve

common occurrence of herpes in epidemic cerebro-spinal fever is for this reason also partly due to the *infection*.

We now come to a series of symptoms in which the question arises *whether they are more the results of the local affections or are in connection with the infection, and which, on the latter account, are of great importance for the differential diagnosis. Fever,* it is true, is constantly present but it does not conform to a distinct type; at one time it may be continuous with slight remissions, at other times it is more intermittent. A certain proportion between the height of the fever and the severity of the clinical phenomena does not exist; in general the range of the temperature is between 101° F. and 104° F. Neither does the *pulse* show a uniform condition; it is conspicuous, however, that its frequency changes very rapidly. A marked diminution in the pulse frequency as a result of the irritation of the pneumogastric nerve is not frequent in cerebro-spinal meningitis, but rather a preagonal rise of the pulse to 100 and over. The quality of the pulse also varies considerably; being at times hard, at other times soft and small, frequently irregular; a marked increase in frequency at the onset of the disease is especially ominous. In keeping with the increase of the pulse as a result of the paralysis of the cardiopneumogastric fibres, paralysis of the pulmonary pneumogastric nerve may cause marked reduction of the respiratory frequency to but a few respiratory movements per minute! Of greater diagnostic importance than the condition of temperature, pulse and respiration, is the *enlargement of the spleen.* However, it is but rarely present, as a rule; but when it can be demonstrated, it is decidedly in favour of the infectious-epidemic character of meningitis. Undoubtedly cerebro-spinal meningitis is due to a specific toxine which acts less generally than specifically *locally,* which may also be noted from the fact that infectious *nephritis* in the course of cerebro-spinal meningitis belongs to the exceptions. Simple albuminuria is not rare, but even in the severest cases it is but a transitory phenomenon the intensity of which in the individual case goes hand in hand with the severity of the nervous symptoms. Frequently, in the course of the affection, *multiple arthritic pains* are observed which are due either to a widespread hyperæsthesia or to a purulent inflammation of the joints. In some cases they may even precede the meningitic symptoms and then must be looked upon as an expression of the general infection.

Complications and Sequelæ.—Of *complications* and *sequelæ* which only indirectly are of importance in the diagnosis we must mention: Catarrhal and croupous pneumonia, of which the latter has a greater pathogenetic significance; further, pleurisy, pericarditis, endocarditis, otitis media, abscess formation in the cochlea, with ensuing deafness, atrophy of the optic nerve and amaurosis, *cerebral abscess,* and *chronic hydrocephalus.* Von Ziemssen has especially called attention to the occurrence of the latter condition: This complication shows itself by headache, arising in paroxysms, by vomiting, convulsions and unconsciousness. Between the attacks occur periods of relative well-being, but general hyperæsthesia, paresis and contractures of the extremities and disturbances of the intelligence may be noted even during these intermissions. Weakness of memory or actual insanity may occasionally persist as sequelæ of cerebro-spinal meningitis. In other cases headache, vertigo, and partial paralysis may persist, for a long time at least.

Microbes of Cerebro-Spinal Meningitis.—The nature of the virus of epidemic cerebro-spinal meningitis has been determined by recent *bacteriologic* investigations. We know to-day that, apart from the *secondary* forms of meningitis in septico-pyæmia, pneumonia, tuberculosis and enteric fever, cerebro-spinal meningitis may occur *primarily*, due to the action of various pathogenetic micro-organisms. These are very probably either streptococci, or—and even frequently—pneumococci, or the intracellular meningococci; should the infection be due to the latter and also in case of the infection of the meninges by pneumococci, the affection shows a tendency to become *epidemic*. It is to be assumed that the invasion of the pathogenic micro-organisms in these cases occurs from the nose and its surrounding cavities. In favour of this is the frequency of the localization of the pneumococci in the middle ear, and the very frequent occurrence of the diplococcus lanceolatus in the nose and its surrounding cavities, and, further, the fact that H. Jäger and Scherer have invariably found Weichselbaum's diplococcus intracellularis, which is usually the cause of cerebro-spinal meningitis, in the nasal secretions of the patients. H. Jäger has lately also succeeded in proving that the *diplococcus*, respectively *tetracoccus intracellularis*, is a special variety of bacteria differing from the pneumococcus that up to this time had been supposed to be the chief cause of epidemic cerebro-spinal meningitis. To the tetracoccus, then, the specific infection of the latter disease may be considered due in the majority of cases.

The diplococcus intracellularis differs from the pneumococcus by its greater breadth (broad bun-shape contrasted with the slender lancet-form of the pneumococcus), by the fact that its capsule is not constantly present, and by the tendency to increase in tetrads. The marked biological difference, however, consists in the action of both bacteria in the animal organism. Whereas pneumococcus cultures, if subcutaneously injected, kill the inoculated animal, the *meningococcus shows itself as non-virulent upon subcutaneous injection*. In an intraperitoneal injection, however, the virulent action of the meningococcus is at one time slightly, at other times more markedly, manifest. A confirmation of Jäger's findings and the first proof of the meningococcus in patients with epidemic cerebro-spinal meningitis was furnished *intra vitam* by Ileubner, who regularly detected the specific tetracocci in the fluid obtained by lumbar puncture. He also succeeded, by an injection of meningococcus cultures into the spinal dural sac in two goats, to produce a well-developed "hæmorrhagic" spinal meningitis, and with this to confirm the positive result of Weichselbaum's injections of meningococcus cultures into the skull cavity of trephined rabbits and dogs. In some few cases of epidemic cerebro-spinal meningitis, *besides* the specific meningococci, pneumococci were found and also streptococci or staphylococci; there was, therefore, a mixed infection which, as it appears, is not very infrequent in cerebro-spinal meningitis. In other cases of unquestioned epidemic meningitis, however, pure cultures of pneumococci were found, so that these, at least in a part of the cases, must be looked upon as the specific cause of epidemic cerebro-spinal meningitis.

Differential Diagnosis.—Epidemics of cerebro-spinal fever occur especially in the winter and spring months. The disease does *not appear to be contagious from person to person;* the mode of infection is probably similar as in the case of diphtheria, i. e., communication of the disease occurring by direct or indirect transference (by means of healthy persons) of material containing cocci, especially from the nose and the pharynx of meningitis patients. A certain *period of incubation* has as yet not been determined; probably it is not very long, the maximum being from three to five days.

In times in which cerebro-spinal meningitis is epidemic we can scarcely go wrong if we diagnosticate an affection with the symptoms that have just been described as cerebro-spinal meningitis. With this, however, it is presupposed that at least certain conditions which favour this diagnosis are taken into consideration, and that affections which may simulate cerebro-spinal meningitis have been excluded. We must first remember

that cerebro-spinal meningitis shows a greatly varying intensity, that severest cases with suddenly occurring chill, unconsciousness and painful rigidity of the muscles of the back of the neck may terminate fatally in a few hours or at least after a day to a day and a half (*m. c. siderans*); others, on the contrary, run an abortive course so that slight rigidity and headache are the only symptoms of which the patient complains. Between these two extremes are found the most varied stages of development of the disease in regard to completeness of the pathological picture and to severity of the symptoms.

Occasionally the patients are taken ill with loss of consciousness and hemiplegia, so that the picture resembles hæmorrhage or embolism of the brain, more than cerebro-spinal meningitis (*apoplectic variety*). In such cases the diagnosis may be extremely difficult; well-developed rigidity of the muscles of the neck, pain along the spinal column on movement, hyperæsthesia, fever and herpes, will eventually put the diagnosis right, especially, however, the circumstance that cerebro-spinal meningitis is *epidemic*. If the latter is not the case, even if the symptoms are not developed in the irregular form just described but are typically pronounced, the diagnosis is enormously difficult. Especially if it concerns the *first case* or one of the first cases in the outbreak of an epidemic of cerebro-spinal meningitis, or if *sporadic cases* of the affection occur. *It is always of primary importance in all cases of cerebro-spinal meningitis, whether they take place in an epidemic or sporadically, to search for some tangible cause (trauma, caries of the bones of the skull, tuberculosis, etc.) for the occurrence of the affection.* Only after it has been absolutely impossible to prove this, dare we think of the existence of a primary cryptogenetic, eventually epidemic, cerebro-spinal meningitis.

Tubercular Meningitis.—As meningitis in general develops most frequently upon the basis of *tuberculosis*, the proof of this condition must be carefully looked for in the individual case. I shall but briefly allude to some few differentio-diagnostic points and shall in the main refer to the discussion of the diagnosis of tubercular meningitis (which see). Usually the course of meningitis is slower, especially does a long prodromal stage precede this form of meningitis, which is very exceptional in the case of the epidemic infection. In tubercular meningitis (in general) the symptoms due to the involvement of the base of the brain are prominent. On the other hand, however, the presence of phenomena which refer to the affection of the spinal meninges is not at all against tubercular meningitis and in favour of a cerebro-spinal meningitis. For the cerebral meninges, under some circumstances, are found exclusively affected in the latter affection, and in other cases, as has already been mentioned, the combination of cerebral and spinal meningitis is quite common in the tubercular form. The enlargement of the spleen is no certain differentio-diagnostic phenomenon either, it occurs occasionally in both forms of meningitis. More important are a *copious crop of herpes and multiple arthritic affections* which are more in favour of epidemic cerebro-spinal meningitis. The diagnosis of a tubercular meningitis is only well founded, however, if tubercular glands and joint inflammations, catarrh of the lungs

with tubercle bacilli in the sputum, intestinal tuberculosis, etc., are demonstrable, and especially if the ophthalmoscopic examination shows the presence of tubercles in the chorioid and if the examination of the fluid taken by lumbar puncture gives a positive result, i. e., the presence of tubercle bacilli.

Meningitis Purulenta—Non Epidemica.—*Purulent cerebro-spinal meningitis* due to other causes than tubercular affection cannot be differentiated by its *symptoms* from the epidemic variety; the most important point for the differential diagnosis is here, too, the ætiology of the individual case, the proof that a primary pus focus existed in the brain or elsewhere in the body, especially, however, in the bones of the skull, before the appearance of meningitis. A plentiful crop of herpes and multiple joint affections, while not exclusively but in general, are against simple purulent meningitis. If an unquestioned epidemic of cerebro-spinal meningitis exists at the time of the observation of the case, the diagnosis is usually very easy; but, inversely, we must then ask ourselves whether there be any reason why the case in question should not be supposed to be one of epidemic cerebro-spinal meningitis. But, as has been previously mentioned, sporadic cases of cerebro-spinal meningitis undoubtedly occur sometimes. If the most exact examination for pus foci, caries, tuberculosis, etc., has shown absolutely nothing which may explain the origin of the (secondary) meningitis, or, particularly, if the proof of specific diplococci in the fluid from lumbar puncture is possible, we are justified in making a diagnosis of primary, *cryptogenetic* cerebro-spinal meningitis. This latter may then be the first case of an epidemic (it was once possible for me by way of exclusion to discover the first case of an epidemic then in the onset) or, what is more frequent, it is a case occurring sporadically.

But the diagnosis of a sporadic cerebro-spinal meningitis is a risky one under all circumstances; we must make it a rule never to make such diagnoses without the most careful reflection if we desire to remain free from gross errors; that is to say, it is necessary for us to exclude affections which appear with a similar pathologic picture and run a course similar to cerebro-spinal meningitis.

Enteric Fever.—It is most easy to confuse the disease with severe forms of *enteric fever,* the picture of which (compare p. 926) frequently resembles meningitis, as insensibility, painful rigidity of the neck and convulsions may dominate the scene (meningo-typhoid). In such cases the irregular course of the fever, the retraction of the abdomen, the absence of enlargement of the spleen, the general cutaneous hyperæsthesia, the intense headache, the appearance of partial spasmodic conditions and paralyses, of herpes, the multiple joint affections, and the constipation are in favour of cerebro-spinal meningitis and absolutely against enteric fever. These points are also conclusive in cases in which, inversely, after a longer duration of cerebro-spinal meningitis, the affection assumes the "typhoid state," i. e., a condition resembling enteric fever occurs with delirium, bed-sores, sordes upon the tongue and lips (*meningitis cerebro-spinalis typhosa*).

Cryptogenetic septicopyæmia may also run a course resembling cerebro-

spinal meningitis, especially if a purulent meningitis develops as the result of a septic infection. We shall later consider the diagnosis of this affection in detail; here it shall only be mentioned that the chills, endocarditis, ecchymoses with a white centre in the eye-ground, which occur with a certain regularity in the course of septicopyæmia, are especially in favour of a septicopyæmic character of meningitis and against epidemic cerebro-spinal meningitis.

Finally, it must also be observed that *croupous pneumonia* in some few cases occurs combined with cerebro-spinal meningitis. Since we know that Fränkel's pneumococcus lanceolatus may disseminate in the body by means of the blood-vessels and lymph vessels and produce secondary cerebro-spinal meningitis, the fact that meningitis may often occur in combination with croupous pneumonia is easily conceivable. It is also worthy of note that the meningococcus, as has been proved upon various occasions, may give rise to a specific pneumonia in the course of cerebro-spinal meningitis.

ACUTE MILIARY TUBERCULOSIS, GENERAL DIFFUSE TUBERCULOSIS

In the diagnosis of acute miliary tuberculosis we must primarily remember that the nature of this affection *consists in an acutely arising dissemination of the organism with tubercle bacilli and in a development of miliary tubercles in the most varied organs of the body.* As is well known, the bacilli, after they have found access into the body, as a rule, produce but a *local* tuberculosis, gradually spread into the neighbouring tissues and organs and lead to caseation of the affected tubercular tissues, as well as to a tubercular infection of the contiguous lymph glands. Should the bacilli proliferate through this barrier and find their way into the lymph channels, a metastatic distribution of tuberculosis in individual organs takes place, which assumes greater dimensions, i. e., leads to a general dissemination over the entire body, if great masses of tubercle bacilli gain entrance into the lymph glands and blood-vessels at one time. The latter mode of dissemination characterizes the nature of the so-called "acute miliary tuberculosis." As is seen, an embolic distribution occurs in the body from a primary focus, and it is plain, therefore, that in the majority of cases of acute miliary tuberculosis we are able to find this primary focus and to determine it directly as such. The latter is the case if acute miliary tuberculosis occurs in the course of pulmonary tuberculosis or of tuberculosis of the urogenital apparatus, and bacilli are passed out in the sputum or urine. If, however, the primary focus of infection is in a cheesy lymph gland, in the pleural or in the peritoneal tissues, in the bones, etc., the direct proof of bacilli is impossible and the diagnosis is deprived of its most certain support. We are then compelled to refer to the analysis of a morbid picture due to a dissemination of the body with the tubercle virus which, upon the whole, is not very characteristic, but, on the contrary, is very ambiguous and which may frequently make the diagnosis extremely difficult.

Diagnostically Important Symptoms.—The general condition points to a severe infection; the *fever* is more or less high, rarely ushered in by a

chill, not characteristic in type, at one time being continuous, at other times remittent; especially does it occasionally show a fall in temperature towards evening [inverse fever type]. In the form of miliary tuberculosis running its course with meningitis these remissions appear to be especially pronounced; in very rare cases the fever may even be entirely absent. In a diagnostic respect, while we have no positive holding regarding the fever course, its *changeable* condition in comparison with the typical fever curve of other diseases, especially in the case of enteric fever, is at least of some importance in the differential diagnosis. The conditions are similar regarding the *pulse*; in contrast to the pulse in enteric fever (at least in the majority of cases), it is comparatively increased, 120 beats and more per minute; a relatively slow pulse only occurs if the miliary tuberculosis is concentrated upon the meninges. A decided loss of weight develops comparatively rapidly; the nervous system also reacts strongly to the affection, giving rise to delirium, somnolence, etc. The *spleen* often shows slight enlargement.

On account of the pronounced predisposition of the *lungs* as a point for tubercular localization, this organ quite usually shows an infection by the miliary tubercles. Accordingly, there develops a conspicuous *dyspnœa* because the development of numerous tubercular nodules causes the respiratory surface to become diminished and also gives rise to irritation of the peripheral ends of the centripetal vagus fibres in the parenchyma of the lung. The *bronchitis* which appears simultaneously also adds to the dyspnœa; everywhere in the lungs fine râles are heard; and these symptoms are supervened by atelectasis of the lung, hypostatic congestions, bronchopneumonia, in short all the resulting manifestations of a capillary bronchitis. The sputum is simply catarrhal, and very rarely does it show an admixture of blood. A symptom which must not be undervalued is the continuous *irritation* causing cough which is present in many cases, due to the development of the tubercles upon the bronchial mucous membrane and the irritation of the sensory vagus fibres. The diminution of the respiratory surface is also the cause of *cyanosis*, which has always been considered a very important symptom of miliary tuberculosis. The degree of cyanosis occasionally is in marked disproportion to the relatively slight bronchitis and pulmonary infiltration. This is of value in a diagnostic respect in so far as a disproportionately marked cyanosis points to the fact that for its production, besides the above-mentioned, demonstrable causes, another must be present which impairs the respiratory surface and the circulation of the lungs (the plentiful development of miliary tubercles in the respiratory organs). If a primary tubercular affection of the lungs is the source of the diffuse affection, as is so frequent, dulness will be found in the upper parts of the lungs and symptoms of cavity must be expected. The latter, however, are rare, as the formation of larger cavities gives rise to a more marked connective-tissue development in the lung, and this, as a rule, prevents the abundant transmission of bacilli from the pulmonary foci.

Miliary tuberculosis in the lungs is almost regularly accompanied with a similar affection of the pleura—a diagnostically important fact, as the occurrence of faint friction sounds is in connection with this. By means

of pleural friction the diagnosis gains a more certain basis in doubtful cases, especially when pericardial friction sounds can be demonstrated besides, as a result of a pericardial miliary tuberculosis. Several times, by the observation of these facts, have I been able to make the correct diagnosis.

The *abdominal organs* are very commonly the seat of miliary tuberculosis, i. e., *the peritonæum, the liver, kidneys, intestines*, are found covered with fresh nodules. This localization of the tubercular poison is of slight importance in the diagnosis, for, as a rule, it does not produce symptoms, or it gives rise to phenomena, such as pain in the abdomen, diarrhœa, albuminuria, etc., which are too ambiguous to be utilized diagnostically, as they exist in so many other conditions. Of very great importance diagnostically, however, is the occurrence of *tubercle bacilli in the urine*, but this is very rare. As has already been mentioned at another place, this proof in general shows the presence of a urogenital tuberculosis. It seems that glomeruli filled with bacilli burst only in the very rarest cases or that the nephritically changed epithelia permit the bacilli to find their way into the urine. In this manner it may be possible in acute miliary tuberculosis that tubercle bacilli occasionally appear in the urine even without a simultaneous nephrophthisis; this, however, as has already been mentioned, is a rarity, so that in general the diagnosis gains but little by an examination of the urine. The same is true of the finding of *bacilli in the blood*. They have on several occasions been detected in the circulating blood of individuals affected by tuberculosis, but the cases in which they have been found are the exceptions compared with the great majority of cases in which the search for bacilli in the blood has proved negative. It is naturally a lucky circumstance if the bacilli which are greatly distributed in the blood are detected in this manner.

In a diagnostic respect it is especially important that miliary tuberculosis frequently localizes in the *meninges*. The occurrence of a *meningitis* in the clinical picture always throws a strong weight into the scale in favour of miliary tuberculosis, although the complication of meningitis also occurs in other infectious diseases, but generally much more rarely than in the case of miliary tuberculosis. The picture of tubercular meningitis is, as is also the picture of meningitis, most ambiguous, the course at times being stormy, at other times, if the base of the brain is especially affected, more protracted. Most frequently, in the tubercular form of meningitis, we are not dealing with an affection particularly limited to the cerebral meninges but with a disease which also affects the coverings and the substance of the spinal cord, therefore with an extended cerebrospinal meningitis. To enter into the symptomatology in more detail is without value, as the symptoms do not vary greatly from those occurring in a meningitis from other causes. Only the finding of tubercle bacilli in the fluid obtained by lumbar puncture and the appearance of tubercles in the chorioid in an ophthalmoscopic examination at once give direction and certainty to the diagnosis. The development of tubercles in the eye-ground also occurs in cases of miliary tuberculosis without accompanying meningitis, and may even be the first sign of a tubercular infection. All the more necessary is it then, in all cases in which a diagnosis of acute miliary tuberculosis comes into question, to examine the eye-ground repeatedly.

Various exanthems are found upon the *skin* (herpes, roseola, erythema nodosum) in the course of acute miliary tuberculosis; also marked sweating may occur—but all these symptoms are without diagnosti

Differential Diagnosis.—If we examine the clinical picture as sketched we will rarely be in doubt in the individual case that we are dealing with an acute infectious disease, but in many instances we will be unable to make a certain diagnosis, as distinct criteria favouring a definite infectious disease are absent. It is rather more often that differentio-diagnostic considerations are necessary for days, yes, even for weeks, to form a certain opinion, and then a great number of diseases must be reviewed. Apart from the rare cases of a diffuse, embolic dissemination of the germs of carcinoma and sarcoma, running a subacute course, we should consider, above all, *septicopyæmia.* This malady, as a result of the general dissemination of the pathogenic material and of its local action, in numerous individual parts of the body develops a clinical picture which very much resembles acute miliary tuberculosis, and which, especially if the affection occurs cryptogenetically, may greatly enhance the differentio-diagnostic difficulties.

The following case observed in my clinic some years ago may illustrate what has been said in this respect: W., aged sixty-nine, workman in a factory, whose work required him to remain for a long time in a dusty atmosphere, was taken ill two months prior to his death with dyspnœa, stitches in the side, and cough. A diagnosis of emphysema, atheroma of the arteries, and cardiac insufficiency was made; the urine was free from albumin. After one week the treatment ceased; and from this time on the patient was no longer entirely well. Three days prior to his death a condition of unconsciousness occurred without apparent cause, and the relatives of the patient brought him to the hospital. The examination revealed coma, twitchings of the right side; *herpes* upon the little finger, and *the epididymes upon both sides nodularly enlarged.* Both apices of the lung showed a raised note upon percussion, breathing, however, vesicular upon both sides; to the left posteriorly below relative dulness, râles; the size of cardiac dulness was unchanged, the heart sounds pure. The urine was cloudy, containing albumin, with numerous (also metamorphosed) casts in the sediment. Highly developed *general hyperæsthesia,* especially on the skin of the abdomen; no paralysis; the left pupil was wider than the right, later both became small. Liver and spleen were not enlarged, temperature 104°, pulse 108, respiratory frequency 38, Cheyne-Stokes type. The ophthalmoscopic examination showed extravasations of the blood in the retina, convulsions occurred in the left arm.—Death. If we examine the morbid picture as sketched there are prominent—on the part of the central nervous system: *General hyperæsthesia, convulsions* on one half of the body (at first on the right, later on the left side), *inequality of the pupils,* later narrowing of the same, *herpes. Cheyne-Stokes respiration*—on the part of the respiratory organs. *Bilateral consolidation of the apices and dyspnœa*—on the part of the urogenital apparatus: *Marked amounts of albumin in the urine,* with casts in the sediment, and *nodular thickening of the epididymes. High fever; sudden onset of the affection* after having been ill for several months.

In the diagnosis there might be considered: Uræmia, as the result of nephritis, or acute miliary tuberculosis. Against the former was, to a certain extent, the temporary localization of the cerebral phenomena to one half of the body, the absence of vomiting, asthma, etc.; in favour of miliary tuberculosis: Bilateral dulness of the apices of the lungs, dyspnœa which was in disproportion to the distribution of the dulness of the lungs, the enlargement of the epididymes, which might have been looked upon as cheesy; the concentration of the infection upon the brain, the affection of which created the impression of an essentially diffuse, most probably of a convexity meningitis. The *autopsy* showed: *Bilateral cheesy epididymitis,* softened thrombus mass in the left epididymis, cheesy degeneration of one renal papilla, *septicometastatic miliary foci up to the size of a pin-head, especially in the kidneys, in the endocardium, in the larynx, and in the mucous membranes of the bronchial tubes, of the large intestine, and in the meninges; bilateral infiltrations of the*

apices of an old date, slight recent enlargement of the spleen (14 :10). It was then not a case of miliary tuberculosis, which might have been expected from the symptoms, but a septic infection in all probability originating in the urogenital system, producing miliary abscesses similar to miliary tubercles in the most various organs!

The confusion of miliary tuberculosis with septic infection which occasionally, as will be seen from the above example, runs a very similar course, may be easiest prevented if some points in the morbid picture of septicopyæmia are noted, which are extremely rare in the case of miliary tuberculosis or do not occur at all. A rapid fulminating course of the affection, quickly terminating in death, in general is more in favour of septicopyæmia than of miliary tuberculosis, in which death only occurs after from four to six weeks, as a rule; in the rarest cases in less than two weeks. High-pulse frequency, enlargement of the spleen, phenomena of cerebral irritation, vomiting and diarrhœa, albuminuria, pleurisy, pericarditis, herpes and roseola occur in both diseases. Pointing more to the development of a miliary tuberculosis are the disseminated bronchitis, the dulness of the apices and, above all, ulcers of the larynx, whereas endocarditis is immeasurably more frequent of septicopyæmic origin. If endocarditis is supervened by severe pains in the bones, a well-developed hæmorrhagic infarct, inflammations of the joints, jaundice, and if the fever shows a tendency to the development of chills and great variations in the temperature, miliary tuberculosis becomes more and more unlikely, especially if petechiæ, pemphigus vesicles or pustules appear upon the skin. The ophthalmoscopic examination may also decide the question if, on the one hand, tubercles are found in the chorioid, and extravasations of blood in the retina with and without white centres, as is the case in septic affections, on the other hand. (Compare also the following chapter.)

Uræmia.—The differentiation of miliary tuberculosis from *uræmia* is less difficult. A confusion of both affections is not easily possible, not even when the cerebral symptoms are markedly developed in miliary tuberculosis. Only in rare cases can real difficulties appear in regard to the diagnosis, namely when nephritis develops upon the basis of a chronic tuberculosis and if to this acute miliary tuberculosis is added. Here the transitory character and the changing intensity of the cerebral phenomena, the prominence of vomiting, the general convulsions and the asthma decide in favour of uræmia, especially if the ophthalmoscopic examination does not show tubercles in the chorioid but a retinitis albuminurica.

Meningitis Non-Tuberculosa.—If the symptoms of *meningitis* are prominent in the picture of miliary tuberculosis, the differentiation of other varieties of inflammation of the meninges may be very difficult, as has already been referred to in the diagnosis of the individual varieties of meningitis. But we are able, in the majority of cases, to make the correct diagnosis, as the finding of tubercle bacilli in the fluid from lumbar puncture and the proof of tubercles in the chorioid furnish us diagnostic aids which not infrequently bring a certain decision in doubtful cases.

Enteric Fever.—The differential diagnosis between miliary tuberculosis and *enteric fever* sometimes gives rise to special difficulties. time this has been looked upon as a most difficult point for

tician and, in fact, every physician has known cases in which it was impossible, at least for a long time, to make a positive diagnosis of the presence of one or another of these affections. This is due to the fact that the symptoms of a severe general affection, the high fever lasting for weeks, with its consequences, the soft pulse, the enlargement of the spleen, bronchitis and bronchopneumonia, as well as the signs of a general irritation of the brain, are uniformly pronounced in both affections! However, there are a number of symptoms which, in a doubtful case, determine the diagnosis in favour of one or the other of the two affections. In referring to what has been said in the discussion of the differential diagnosis between typhoid and miliary tuberculosis (which see), I shall attempt here clearly to arrange the differentio-diagnostic points in order:

IN FAVOUR OF	
MILIARY TUBERCULOSIS	ENTERIC FEVER
Irregular course of the fever; it never occurs for some length of time as a continuous type, but shows, even during the first stages of the affection, remissions which may take place even in the evening.	*Typical course of the fever*, which continues to be high for some length of time; remissions and, later, intermissions in regular order, in keeping with the course of the typhoid process, subsequent to the continuous fever.
Frequency of the pulse relatively high as compared with the temperature and not reduced during the remissions, at most sometimes retarded due to development of tubercular meningitis.	Relative slowing of the *pulse*, especially during the first stages of the disease.
The enlargement of the spleen, if at all demonstrable, not very pronounced and developing gradually.	*Enlargement of the spleen* considerable, almost always demonstrable by percussion and by palpation as early as in the first week.
Roseola, rare and at most occurring isolatedly, does not appear at a certain time during the course of the disease and not in crops.	*Roseola* occurs about the middle of the second week in crops.
Bronchitic phenomena, especially small râles, more marked in the apices of the lungs than in the lower portions of the lungs. *Acceleration of respiration and cyanosis not in proportion to the intensity of the bronchitis.* Dulness eventually over the apices of the lungs.	*Bronchitis*, diffused over the entire lungs, but more marked in the lower portions than in the apices. Dulness eventually in the lower portions.
Pleuritic and pericardial friction sounds.	*Absence* of pericardial friction sounds; pleuritic friction sounds occur also in enteric fever, according to my experience.
Tubercle bacilli in the sputum and in the urine, but rarely demonstrable in general. *Diazo reaction* of the urine eventually absent during the entire course of the affection; however, a positive reaction is not against miliary tuberculosis.	Presence of *typhoid bacilli in the urine* may be expected with constant albuminuria. *Sputum* free from tubercle bacilli. *Diazo reaction* of the urine well developed, sometimes not occurring until late.
Diarrhœas not especially frequent, fæces may eventually contain tubercle bacilli.	*Diarrhœas* frequent, fæces may eventually contain typhoid bacilli.

MILIARY TUBERCULOSIS	ENTERIC FEVER
Appearance of the *tongue* not characteristic.	The *tongue*, during the first week, shows red borders and, during the second week, is of a uniformly diffused, red colour.
Meningitis very common, eventually chorioid tubercles.	*Meningitis* very rarely supervening upon enteric fever.
Gruber-Widal reaction, see p. 962.	

In individual cases, however, even with complete consideration of the differentio-diagnostic points referred to above, the differentiation of both diseases is difficult, and even impossible upon brief observation.

Thus Senator observed a case of miliary tuberculosis in which enlargement of the spleen, well-developed roseola, dicrotic pulse, epistaxis and purulent parotitis, in short the entire picture of enteric fever, was developed and the chorioid free from tubercles. Only the fever from the onset showed marked remissions, amounting to 2° F. A similar case occurred recently in our clinic:

Case of Miliary Tuberculosis resembling Enteric Fever.—Patient, aged forty-nine, has been coughing for one year. Eight days prior to his admission to the Julius Hospital the affection in question commenced with lassitude, headache, diarrhœa, cough, and disturbance of sleep. The examination showed *very marked development of the body. Colour of the face* pale-*cyanotic; increase of the respiratory rate*, now and then râles at the right apex of the lung, over other parts of the entire lung no sign of catarrh or dulness. *On the left posteriorly below fine pleuritic friction*; the heart sounds were also partly accompanied with friction, cardiac dulness normal, impulse very weak. Soft, relatively strong, *frequent* pulse. The sputum was sparse. Delirium.

The skin of the abdomen showed several unquestioned *roseolæ; the spleen was plainly enlarged, the hard point being palpable; the tongue was coated only in the centre; diarrhœa; fever about 104° F. with partly decided morning remissions, to about 101° F. Urine was albuminous, also containing epithelial casts; the eye-grounds were normal.*

The last-mentioned symptoms are all directly in favour of typhoid. Nevertheless, a diagnosis of acute miliary tuberculosis was made, and for the following reasons: The respiration was markedly accelerated (it is true, without catarrh of the fine bronchi), cyanosis was well-developed—a decrease in the respiratory surface was, therefore, unquestionably present. The sparse sputum contained tubercle bacilli in quantities; therefore, a source for an acute miliary tuberculosis was unquestionably present. The pulse was constantly disproportionately frequent and not in keeping with the temperature, whereas it should be relatively slow in enteric fever. On the other hand, the roseolæ were but few, the spleen was too hard for typhoid (we assumed that this was due to former causes of enlargement, perhaps to malaria), the fever was too markedly remittent for a *severe* typhoid which was the only form of the affection to be considered. The presence of diarrhœa, on account of being so variable a symptom and occurring in so many different diseases, was not utilized in the differential diagnosis, nor the intact condition of the eye-ground, as the latter may also remain normal in acute miliary tuberculosis. More in favour of the diagnosis of miliary tuberculosis was the fine pleuropericardial friction which pointed to the development of tubercles in the pleura in the neighbourhood of the heart. The *autopsy*, after death had occurred upon the seventeenth day of the disease, confirmed our opinion. A small cavity was found in the right apex, disseminated acute miliary tuberculosis, tubercles of the pleura, with beginning inflammation of the pericardial pleura, splenic tumour of hard consistence, miliary tuberculosis of the kidneys, slight nephritis.

In contrast to this is a second case from the Würzburg clinic, in which just the opposite occurred, all characteristic symptoms of enteric fever being absent and still

typhoid being the correct diagnosis, not miliary tuberculosis which was the only affection to be taken into consideration.

Case of Enteric Fever occurring under a Picture resembling Miliary Tuberculosis.—A girl, aged twenty-five, previously allegedly healthy, was taken ill with pains in the small of the back and *chills*, lassitude, headache, and *vomiting*. She continued her work for three days longer and only then came under clinical observation. The examination revealed: Well-developed body, *fever* very high, 107° F., *with marked morning remissions down to 103° F. The pulse markedly accelerated, between* 100 and 120, later even 150; *two questionable roseolar spots, spleen not enlarged; constipation; a disseminated catarrh of both lungs*, i. e., therefore, all symptoms more in favour of acute miliary tuberculosis than of enteric fever. Nevertheless, the latter was more likely than miliary tuberculosis, as there was no increase in the respiratory rate nor cyanosis, nor were pleural or pericardial friction sounds present, and, later, the bronchial catarrh, which was well spread through both lungs, first disappeared in the apices; the pulse frequency was, it is true, unusually high, but just in the first week it had been relatively low in comparison with the fever. Therefore, in spite of the fact that eruption, enlargement of the spleen, and diarrhœa were absent from the beginning to the end of the affection, a diagnosis of enteric fever as the more probable was made. In fact, this diagnosis showed itself to be correct in so far as the patient, after a very protracted disease, showing many complications, left the hospital well after an illness of four months.

Fortunately, by means of the Gruber-Widal reaction we have lately obtained a further, quite important, differentio-diagnostic aid which will allow us to determine a class of cases of enteric fever with certainty, which, formerly, we were unable to diagnosticate. In referring to what I have previously said in reference to the diagnosis of enteric fever (which see), I should like to emphasize here once more that neither the positive result nor the absence of the reaction *without* further consideration is in favour or against the presence of enteric fever, but that, on the other hand, *in cases in which the reaction is negative for several days, yes, even for weeks, and only very gradually becomes positive, the diagnosis of enteric fever may be made with absolute certainty, and other diseases, especially miliary tuberculosis, may be excluded.*

Confusing miliary tuberculosis with *malaria, pneumonia,* and *various forms of poisoning,* or even (in exceptional afebrile cases) with insanity can only result from a very superficial examination or brief observation.

Capillary Bronchitis.—However, occasionally an acute capillary *bronchitis*, especially in children, may resemble miliary tuberculosis. In such cases the hereditary conditions, the severity of the morbid picture, the rapid loss of strength, a possible enlargement of the spleen, the predominance of the râles at the apex of the lungs, the pleuritic friction sounds, etc., very frequently direct the diagnosis into the proper channel. But so long as a meningitis does not supervene or the ophthalmoscopic finding shows pathognomonic results, the diagnosis had better be left in suspense in many cases, as nothing but a probable diagnosis is possible. It will be well in such doubtful cases to search the blood for tubercle bacilli; but, as has already been mentioned, a negative finding is not at all against the existence of miliary tuberculosis.

CRYPTOGENETIC SEPTICOPYÆMIA

Up to within recent times the septic diseases were grouped among the so-called "accidental wound diseases," i. e., they belonged to the domain of surgery. Since the year 1878 in which, by reason of several observations, I called attention to the frequent occurrence of septic affections *without a proof of a traumatic infection being possible,* and proposed for such cases the designation *"cryptogenetic septicopyæmia,"* numerous cases have been reported of this affection from medical clinics—isolated cases are also found in the earlier literature, and especially Wunderlich determined and described correctly the clinical picture of "spontaneous pyæmia." The affection is much more frequent than is usually supposed, especially if those cases are included in which a careful examination and differential diagnosis leads to the positive assumption of a septicopyæmia, in spite of death exceptionally not occurring but recovery taking place. We are not only justified but absolutely compelled to do so after the possibility of cure of the most marked cases of septicopyæmia, in which an ætiologic basis could be demonstrated, has been repeatedly observed; in fact, after it has been proven that even recovery may take place in cases of cryptogenetic septicopyæmia in which it is possible to demonstrate, as in one of my cases, cocci in the blood of the patient and to produce a pure culture.

Pathogenesis of Septicopyæmia.—Before we describe the diagnostically important symptoms, it is necessary briefly to enter into the nature of septicæmia and pyæmia in general and of cryptogenetic septicopyæmia in particular, so that we may be able to differentiate the affection with which we are concerned, from other diseases. By *pyæmia* we understand diseases that are characterized in that a primary focus, containing staphylococci, streptococci, or pneumococci, has, *by means of the lymph channels or of the blood current, disseminated the exciting cause of the disease in the body.* The pathogenic bacteria which thus colonize in various parts of the body cause here, in keeping with their action upon the tissues, i. e., with the action of the inflammatory and pyogenic "proteins" produced by them, *localized "metastatic" suppurations.* Besides, on account of the absorption of the toxine substances produced by these metastasing bacteria, an intoxication manifests itself which occurs in separate stages and markedly assists the fatal termination of the affection. The characteristic condition in pyæmia is, and this must be remembered, the *disseminated metastatic suppurations* arising from a primary focus. These primary foci of the pus cocci are abscesses, phlegmons, wounds, etc. The micro-organisms which are most frequently found are staphylococci, especially the staphylococcus aureus (which is also the usual pathogenic agent in acute osteomyelitis), rarely streptococci and pneumococci.

Septicæmia differs principally from pyæmia in that in this affection the *bacteria spread from the primary focus over the entire vascular system, accumulate especially in the capillaries, and grow.* This course of distribution is typical of certain bacteria: The spirilli of recurrent relapsing fever, the bacilli of anthrax and, under certain conditions, streptococci and pneumococci, and to a certain degree also of staphylococci. According to this, relapsing fever and anthrax are typical examples of true septicæmia. This general conception of septicæmia which is precisely defined by bacteriologists is not generally accepted in human pathology. We understand for the present by "septicæmia sensu strictiori" those forms of septicæmia which are produced by the so-called pyogenic organisms κατ' ἐξοχήν, as a result of their dissemination in the blood from a primary focus. I do not see why this specialization, provided an agreement can be had as to the clinical conception of s ou

strictiori, shall not conform to the bacteriologic, general conception of septicæmia. After the streptococci or pneumococci, by means of the lymph channels or directly, have found their way into the blood from a primary focus (which may have been a phlegmon, a diphtheria, a pneumonic area of infiltration, or a local puerperal affection), they *increase* in the vascular system, especially in the capillaries and gradually grow into the veins, from whence they are carried into the general circulation. This is rarely the case in man, as death occurs before this takes place, and this explains that the specific bacteria are relatively rarely found in the blood. The action of bacterial toxines is to be considered the most important factor of the affection also in the pathological picture of septicæmia, besides the mechanical effect due to the obstruction of numerous capillaries.

A strict differentiation of pyæmia and septicæmia, as much as we might wish for it from a scientific standpoint, is not possible at the bedside, as, according to the amount of bacteria which find their way into the blood, at one time metastases result, at other times septicæmia. Theoretically, we may, therefore, expect transitional forms between pyæmia and septicæmia, and this actually occurs in fact, and so, in a practical respect, it is certainly correct in the majority of cases to speak of "*septicopyæmia.*" Only in extreme cases is it of value to adhere to the names pyæmia and septicæmia, i. e., to designate as cases of *pyæmia* those in which demonstrable pus foci and phlebitico-thrombotic areas are the starting-points of secondary metastatic pus formations at various parts of the body, and in which the general phenomena are more of a secondary nature; but to use the designation septicæmia for those cases in which from a primary focus, especially from phlegmons, local puerperal affections or pneumonic infiltrations of the lungs, severe general affections are brought about without producing demonstrable purulent metastases.

Special Mode of Infection.—Why in individual cases a primary purulent focus runs its course without producing reaction or gives rise to metastases or to septicæmia, is due to various factors. Obviously, under ordinary circumstances, the organism is able to overcome these deleterious effects and that by means of numerous protective agencies over which the normal body has power. To these belong, in the first place, the local reaction of the organism to bacterial invasion in the form of inflammation; the leucocyte wall which is formed thereby, is able, very probably by the protection of *alexines*, to weaken or entirely to destroy the bacteria and thus prevent their entrance into the circulation. If this defensive measure does not act or is insufficient, and if the bacteria reach the circulation, the infection of the organism is dependent upon the amount and virulence of the bacteria which have invaded it. Bacteria that are few in number or not highly virulent are easily disposed of by the organism with the assistance of its protective measures, so that demonstrable inflammatory phenomena or multiplication of the bacteria do not occur, but the latter rather disappear from the body in a very brief period. The case is different, however, if some parts of the body are seriously damaged in their circulation or nutrition, for example, if a bone crush, etc., preceded the invasion of bacteria, or if great quantities of virulent bacteria disseminate in the blood. Here the protective power is no longer sufficient to destroy the bacteria: They increase in the capillaries and eventually produce non-demarcated inflammations in their neighbourhood; the bacterial toxines are more or less completely absorbed and the organism, in the majority of cases, finally succumbs to the infection. This occurs, as experience has taught, still more easily if the protective power of the body against the bacteria has been weakened by other factors acting at the same time. To conclude from the results of experiments, such factors seem to be exhaustion, hunger, anæmia, diminished alkalinity and watery condition of the blood, and, finally, also a flooding of the body with putrid substances. It may possibly be due to the suspension of the expulsion of putrid substances from the body that, upon stagnation of the intestinal contents or upon deficient function of the kidneys, septic infection generally occurs more readily. But, otherwise, clinical manifestations which are produced by intoxication with putrid substances, i. e., with the products of saprophytic micro-organisms (as we observe after the ingestion of decaying food and in putrefactive decompositions of portions of our own body), have nothing to do with septicæmia and pyæmia.

Ætiological Points of Support.—The place of entrance of the bacteria into wounds, abscesses, etc., is quite evident in some cases of septicopyæmia, but in other cases the source of septic infection is not demonstrable during the life of the patient, in spite of the minutest investigation (*cryptogenetic form of septicopyæmia*). In such cases we do not obtain the key to explain the occurrence of septicopyæmia until at the autopsy, in that ulcerated bronchial and mesenteric glands, old abscesses in internal organs, residues of puerperal or perityphlitic processes, etc., are found post mortem and can be brought into relation with the septic infection. But sometimes even the autopsy does not give any information, i. e., no such area can be demonstrated as the starting-point of the general infection. Nothing remains in such cases but to assume that bacteria of an extremely great virulence entered in large quantities by smallest wounds which cannot be demonstrated post mortem, or exceptionally also by the uninjured surface of the skin and mucous membranes (lungs, intestinal mucous membrane), passed into the blood current, and attained their pernicious action under the influence of factors which debilitate the defensive power of the organism. In the discussion of the diagnosis of septicopyæmia we shall only consider the cryptogenetic form of the disease.

Symptoms which are to be considered Diagnostically.—The patients, in the full enjoyment of health, or in some cases after slight vague pathological symptoms have preceded, such as lassitude, pains of the limbs, loss of appetite, eventually vomiting and headache, are attacked by a more pronounced fever; they create the impression of being prostrated, very sick, even if the course is not a very stormy one or if the case terminates in recovery.

Condition of the Fever and of the Pulse.—The *fever* is of an extremely varying type; generally *a tendency to a very abruptly changing course* cannot be mistaken, although in some cases continuously high, and in others intermittently continued low, temperatures are observed, and this fact is in connection with the irregular intercurrence of *chills*. The *pulse*, at least in the severe cases, is disproportionately accelerated (120 to 150), soft, dicrotic and sometimes irregular. These alterations of the condition of the pulse are especially marked in cases in which the heart post mortem shows changes of the myocardium (punctiform, mostly hæmorrhagic, areas).

"Malignant" Septic Endocarditis.—Percussion of the *heart* reveals sometimes a diffusion of cardiac dulness, either as the expression of the decreased energy of the heart, of the acute dilatation of the organ, or as a sign of an *endocarditis* which gradually becomes more manifest and the onset of which represents a diagnostically important, frequent symptom of cryptogenetic septicopyæmia. There are cases in which, at the beginning of the disease, nothing else can be demonstrated objectively but an endocarditis with its subsequent symptoms (a loud systolic or systolico-diastolic murmur, accentuation of the second pulmonary sound, etc.), in which the affection of the heart predominates, and in the further course of which emboli take place, especially into the spleen and kidneys, and metastatic abscesses develop. This form of endocarditis was until recently designated as "malignant endocarditis," and is still considered a substantive disease. But for theoretical reasons this assumption cannot be upheld, and should be rejected also in a practical respect, because, with such a conception of the disease, the complications which appear in the train of those "malignant endocarditides" cannot be anticipated to occur and their

importance is misjudged. *Malignant endocarditis, according to my opinion, is nothing but a cryptogenetic septicopyæmia in which the localization of the septic poison has taken place primarily in the endocardium and is restricted for some length of time to the heart.* However, such an exclusive septic affection of the endocardium is by no means frequent; in *by far the majority of cases endocarditis rather forms only a link of the large chain of multiple localizations of the toxine*—it is true a very important localization of cryptogenetic septicopyæmia, because a bacterially inflamed endocardium opens the gate to an embolic dissemination of the toxine into the various organs of the body.

Inflammations of the Joints and Bones.—The greatest diagnostic significance, second to endocarditis, is due to *inflammations of the joints.* In some cases several joints are affected at the same time, and the inflammatory manifestations disappear and recur, in a similar manner as in an acute articular rheumatism. But septic arthritic inflammations show in general a more *steady* character; the process is frequently concentrated upon only one joint, and it is especially suspicious if the joint which is alone affected is a large one. The same as endocarditis so can also an articular affection predominate as the sole localization of the infection, at least for a short time; then the differential diagnosis offers considerable difficulties. In such cases we generally find, besides the articular inflammations or even without them, an *affection of the bones,* especially of the long tubular bones. They are painful upon pressure and also upon movements or upon concussion of the body. The metastatic bacterial inflammation of the periosteum and bone marrow—it is a question of such a disease according to the results of autopsies—is often restricted to a small area of the bone, with sometimes temporary painfulness of this one area or at other times with a permanent pain in this locality. Metastatic inflammations of the *muscles* are less frequent.

Changes of the Skin.—Of diagnostic importance are, furthermore, *changes of the skin* which almost constantly occur in the course of cryptogenetic septicopyæmia. The most varied manifestations of septic infection have been observed and described in such cases: Hyperæmias of the skin in the form of roseolæ and erythemata, urticaria-like exanthemata which cover the skin like a map; furthermore, purpuric areas, hæmorrhagic pemphigus vesicles, variola-like pustules, etc.; herpetical eruptions are also seen at times. In the further course it may occur that the subcutaneous cellular tissue becomes affected by inflammatory-œdematous and hæmorrhagico-purulent infiltrations. Although cutaneous affections, especially hæmorrhages, are frequently (surely in three quarters of the cases) found in cryptogenetic septicopyæmia, yet the ambiguity of the eruptions alone does not permit us to draw conclusions regarding the presence of septicopyæmia, but, nevertheless, they form a support for the diagnosis so that we should be careful in assuming septicopyæmia if no eruptions are present.

Nervous Symptoms.—Diagnostically to be considered of other symptoms of cryptogenetic septicopyæmia are, above all, *manifestations on the part of the nervous system.* They are almost constantly present, sometimes very distinctly pronounced, at others at least indicated. These are some-

times slight symptoms: Headache, vertigo, psychical excitement, insomnia; at other times most severe disturbances of consciousness, and convulsions, and also paralyses of some nerve districts. The clinical picture of these nervous disturbances depends upon the manner in which the toxine acts upon the central nervous system, i. e., whether only a general intoxication becomes manifest or whether anatomical changes develop by way of bacterial metastasis, such as purulent meningitis, multiple hæmorrhages, emboli and consecutive, purulent areas of softening in the substance of the brain, with their respective clinical sequelæ. Septicopyæmia may also exert a toxic effect upon the peripheral nervous system, and the individual nerves may become irritated and neuritic, respectively neuralgically affected.

In connection with nervous manifestations we may mention *changes in the eye* which have been studied particularly by Litten, but which had already been demonstrated and correctly explained in my first cases by J. Michel. Above all, it is a question of retinal hæmorrhages which develop in the course of the disease and which, though they are not strictly pathognomonic, yet may materially facilitate the determination of the diagnosis.

Ophthalmoscopic Findings.—These hæmorrhages are sometimes rounded, at other times of an irregular shape and of varying sizes, but they may also be so massive that they can be designated as regular patches of blood; sometimes they show a whitish coloration in their centre. The retina is generally slightly cloudy. In such cases we are dealing with embolic obstructions of smaller vessels of the retina and of the optic nerve. If emboli occur into the larger vessels of the retina and of the eye in general, symptoms of phlegmonous inflammation of the eyeball, a so-called panophthalmia, become manifest.

Minor Symptoms.—Other symptoms of cryptogenetic septicopyæmia which are also observed in the course of the affection, are of minor diagnostic importance in comparison to the manifestations of septic infection mentioned so far, but they supplement the clinical picture in the given case and in such a way are worthy of careful consideration. Owing to localization of the septic process upon the *pericardium, pleura and peritonæum*, there arise either the smallest areas of inflammation or serous and purulent exudations; we find, accordingly, in these locations friction, dulness, etc. The *lungs* are sometimes the scene of miliary abscesses, at other times of large infarcts, abscesses and of lobular pneumonias. Of especial frequency is a diffused bronchitis the origin of which should probably be considered a consequence of the action of the toxine. These morbid changes in the respiratory tract and the cardiac weakness cause the high frequency of respiration and the cyanosis which are observed in patients with cryptogenetic septicopyæmia.

Although *swelling of the spleen* should also be counted among the regular sequences of septicopyæmia, yet the symptom is of little value diagnostically; on the one hand, because splenic tumours are quite as frequent in other infectious diseases as in the affection we are dealing with, and, on the other hand, because a septically swollen spleen is so slightly enlarged and so soft that it cannot be felt during life. But it will be of greater diagnostic importance if the spleen is the seat of a large embolic infarct which originates in the heart, and if the organ thus becomes acutely enlarged, with pains, and easily palpable. The cloudy swelling of the *liver* and also the occurrence of metastases in this organ cannot be diagnosticated unless larger abscesses of the liver develop. The symptoms on the part of the *digestive organs*: Dyspepsia, vomiting, diarrhœa, are neither constant nor characteristic; more attention should be paid to (septic) *jaundice*, which, however, is of rare occurrence. But disturbances of the *renal* function can be constantly observed, and we find especially albuminuria, undoubtedly caused by irritation of the kidneys in consequence of the septic intoxication. If the irritation increases to parenchymatous inflamma-

tion of the organ, we will observe urinary changes characteristic of acute nephritis, such as the occurrence of blood and casts in the urine, etc. If large abscesses form in the renal tissue, the pus may perforate into the renal pelvis and be voided with the urine; but usually it is a question of multiple miliary, hæmorrhagico-purulent areas of the renal tissue which are without clinical importance.

From the above statements we will note that those parts of the clinical picture of cryptogenetic septicopyæmia which are to be considered diagnostically, are composed of general symptoms: Irregular, usually considerable, fever, disproportionately high frequency of the pulse, great debility, more or less severe affection of the central nervous system, enlargement of the spleen; and of special symptoms caused by the localization of the septic toxine in the various organs of the body. The latter, the peculiar forms of eruptions, articular inflammations, pains in the muscles and bones, endocarditis, emboli in the spleen and liver, lungs and brain, nephritis, inflammations of the serous membranes, and retinal hæmorrhages, are of far greater importance diagnostically than the general manifestations caused by the action of the toxine. However, the diagnosis is by no means easy; on the contrary, it requires, under all circumstances, an exact consideration of the possibility that another affection be present the cause of which is accompanied with similar symptoms.

Differential Diagnosis.—The differential diagnosis is especially difficult when the localization of the toxine is a limited one, when one or the other organ is separately affected, thus simulating the presence of a local affection or of another infectious disease which especially attacks the organ in question. In this respect we must consider principally the following diseases:

Acute articular rheumatism is most apt to take a course similar to septicopyæmia. Articular pains, endocarditis and the general symptoms are common to both diseases. However, the transient nature of the articular affection and also profuse perspiration are decidedly more characteristic of articular rheumatism, whereas the painfulness of the bones, the eruptions and the inflammations of the serous membranes are more in favour of septicopyæmia. If, besides, intermittent fever sets in interrupted by chills, this fact is decidedly in favour of septicopyæmia, the diagnosis of which becomes more certain if retinal hæmorrhages are also present. But there are cases of septicopyæmia in which the majority of the above-mentioned differentio-diagnostical characteristics are absent, and in which the articular inflammations and endocarditis are so isolated in the clinical picture that the diagnosis remains doubtful for quite a while. On the other hand, according to my experience there are also cases of articular rheumatism in which even the autopsy does not permit of a positive opinion whether one or the other disease, or a combination of both, was the cause of the fatal termination. The diagnostic doubts are sometimes removed by a rapid, permanent success of sodium salicylate, of antipyrine and of other specifics for articular rheumatism.

Intermittent Fever.—If a decidedly intermittent fever, interrupted by chills, is manifest, the question arises whether *intermittent fever* be present. However, the paroxysms of fever are much more regular in the latter

affection, the swelling of the spleen is more pronounced, and metastases, which are so characteristic of septicopyæmia, are absent. The fact that quinine does not exert so lasting an effect and never aborts the disease in a septic infection, can also be applied to the diagnosis. Of course, in doubtful cases it becomes necessary to examine the blood for the plasmodia malariæ. (Compare the case of intermittent fever in the chapter on Malaria.)

Acute Miliary Tuberculosis.—Much more difficult is the differentiation of cryptogenetic septicopyæmia from *acute miliary tuberculosis*. We have, when discussing the diagnosis of the latter disease (p. 958), reported a case of septic infection which clearly illustrates the differentio-diagnostic difficulties in the given case. Common to both affections are the acute flooding of the body with pathogenic microbes, the severity of the clinical picture, the rapid loss of strength, the splenic enlargement, giddiness, the high frequency of the pulse, inflammations of the pleuræ, pericardium, peritonæum, meninges. Acute miliary tuberculosis is generally more favoured by cyanosis, dyspnœa, apical catarrh. But if no tubercle bacilli can be demonstrated in the sputum, urine, or blood of the patients (and this demonstration is impossible in by far the majority of cases), the diagnosis must sometimes be left in suspense until violent, localized pains of the bones, intercurrent articular inflammations, endocarditis, chills, jaundice, pemphigus-like or variola-like eruptions and the ophthalmoscopical finding (retinal hæmorrhages may also occur upon the supervention of meningitis upon miliary tuberculosis), will definitely lead the diagnosis in the direction of septicopyæmia.

Enteric Fever.—The last-mentioned symptoms which are more in favour of septic infection also enable us to make the diagnosis between septicopyæmia and *enteric fever*. At the beginning, as long as nothing but enlarged spleen, bronchitis, diarrhœa, continuous fever and roseola are demonstrable, the diagnosis may remain very doubtful, until the irregularity of the fever curve, the inflammations of the joints, jaundice, osteomyelitic and endocarditic manifestations, become more prominent, and the retinal hæmorrhages secure the diagnosis of septicopyæmia. For, although retinal hæmorrhages are also found in typhoid fever, i. e., when meningitis complicates the latter affection, it is such a rare occurrence in the course of typhoid that *retinal hæmorrhages* ceteris paribus are almost positively determining against typhoid and in favour of septicopyæmia. Finally, the occurrence of the Gruber-Widal reaction in the course of the disease offers the most important support of a definite diagnosis of enteric fever.

The differential diagnosis at the bedside wavers comparatively often between the above-named diseases: Acute articular rheumatism, intermittent fever, acute miliary tuberculosis, and especially the last two, on the one hand, and septicopyæmia, on the other. Rarely, that is, only in septic cases which are accompanied with albuminuria or nephritis and rapid onset of coma, are we confronted by the question whether we are dealing with cryptogenetic septicopyæmia or with simple *uræmia*.

Uræmia.—This question is the more justified because other symptoms besides coma (vomiting, headache, diarrhœa, etc.) which are characteri-

of uræmia are also occurring in septicopyæmia and, besides, owing t nephritis which associates itself with sepsis, a retention of excrementiti substances and an admixture of uræmic symptoms to the septic ones n occur. For septicopyæmia and against a uræmia on the basis of a n septic acute nephritis are the recession or absence of convulsions, com cating endocarditis, and articular inflammations, but, above all, the h fever with chills, and a certain stability of the morbid picture, whereas uræmic attack may disappear suddenly and be replaced by thorough w being as soon as the discharge of the urinary substances has improved.

It is only exceptionally that we are compelled to consider other dise in the differential diagnosis. Thus, with an early, marked predomina of an eruption it may be a question of *scarlatina* or, as in one of my ca of *variola*; furthermore, if the septic infection localizes preferably in t lungs, of lobular *pneumonia*; with septic infection of the brain, of a *plexy*. However, such cases will not present very serious diagnostic di culties for any length of time, as cryptogenetic septicopyæmia alm always, although not at the onset but still in the course of the dise furnishes a symptom-complex which is especially characteristic and adm of a positive diagnosis. The latter becomes almost certain if we succ in demonstrating and cultivating in the *blood* during life such bacteria are in genetic connection with septicopyæmia. This demonstration is, t fortunately, rarely successful, owing to the reason that we have sta above.

ACUTE ARTICULAR RHEUMATISM—ACUTE POLYARTHRITIS

Acute Rheumatic Fever

There can be no doubt of the infectious nature of acute polyarthritis. The mu plicity and, above all, the transitory appearance of the inflammatory sympto which distinguish polyarthritis from every other kind of articular inflammation, fact that certain specifics, primarily salicylic acid, cause immediate cure in so cases even with very extensive local manifestations, force the conclusion upon that we are dealing with an infectious disease when confronted with acute artic rheumatism. Less determining of the infectious character is the occurrence of affection in epidemics, the inverse ratio of its frequency to the atmospheric preci tations, etc., because this condition might also be in direct connection with clima circumstances.

A distinct micro-organism as the specific generator of the disease is not gen ally acknowledged as yet for acute articular rheumatism. Various investigato (Petrone, Tizzioni, Guttmann, Sahli, etc.) have found micro-organisms in the e date of the joints: Streptococci, pneumococci, and, most frequently, staphyloco By reason of these findings an ætiological *rôle* in the origin of polyarthritis has b ascribed to the "pus cocci," and it was assumed that this affection is the prod of an infection of the body with attenuated pus cocci. The fact that angina is f quently associated with acute articular rheumatism was explained in such a man that the tonsils might be the portal of entrance for the respective micro-organisms many cases of polyarthritis.

In fact, Fr. Meyer recently succeeded in the clinic of Leyden in cultivating up blood agar from the tonsillar mucus of rheumatic patients *delicate streptococ diplococci* which, inoculated into animals, produced, after one week, fever, se purulent articular inflammations, serous exudates free from bacteria in the pe cardium and peritonæum and in the pleural cavity, as well as endocarditis of eith a verrucose or of an ulcerative nature, but no sepsis. There can be no doubt, the

fore, as I believe, that the diplococcus which was cultivated by Meyer is in a direct ætiological relation with acute rheumatism in man. But whether this organism is the sole generator of the disease, or whether, similarly as in cerebro-spinal meningitis, other micro-organisms might not, at the same time, be able to produce polyarthritis, must be left undecided for the present.

The onset of a pronounced affection is rarely preceded by prodromes of a vague nature, such as lassitude and sharp pains, and relatively often, as it appears, by *angina*; usually *fever* appears at once and very soon afterward *articular pains.*

Affections of the Joints.—The latter in most cases localize at first in the joints of the lower extremities, especially in the *joints of the foot and knee*; the joints of the hip are more rarely, those of the hand and finger more often, affected. It is of importance diagnostically that the *vertebræ* are also affected and, according to my experience, not rarely and even quite isolatedly, thus causing the simulation of other clinical pictures such as caput obstipum and spinal meningitis; the sternoclavicular joints are sometimes also the seat of the articular affection, rarely the joints of the lower maxillary bone, the symphysis pubis and sacro-iliaca. The region of the affected joints is swollen, the skin over them appears tense, reddened, glistening, sometimes slightly œdematous. The slightest touch of the joints causes severe pains; passive and active movements increase them excessively; the patients, therefore, assume at once a slightly flexed posture and an absolutely quiet position. I must leave it undecided whether the decrease of electro-cutaneous sensitiveness to pain over the affected joints, which has been frequently observed, is of pathognomonic significance. As a rule, the inflammation remains only a few (three to four) days in one joint; the affected joint becomes smaller, painless, and, instead, other joints now become affected. It is particularly this rapid change in the localization of the affection which is characteristic of polyarthritis, also the dissemination to many joints. It is true, it also happens that individual joints remain isolatedly affected for some time, and also that only *one* joint becomes affected, but these occurrences are always exceptions to the rule.

Fever.—The course of the *fever* does not adhere to a certain type. It usually commences with chilliness, rarely with a chill, and the temperature rises to 102.5° F. to 104° F., to remain at this height, with morning remissions, for some time. The fever is less in mild cases, but it is never absent, although periods free from fever may occur in the course of the affection. The decrease of the temperature is by lysis, rarely by crisis unless specifics are administered. Hyperpyretic temperatures occur (109.4° to 111.2° F.) in remarkably severe, but fortunately very isolated, cases. These rises of temperatures are the highest which have ever been observed at the bedside [in an infectious disease]. Death occurs in such cases in connection with these rapid, enormous rises of temperature, with severe cerebral symptoms, which will be referred to later on. The *pulse* is always accelerated, sometimes disproportionately so; its frequency and other conditions are essentially influenced by the prevailing activity of the heart.

Nervous System.—The *central nervous system* does not present any disturbances in by far the majority of cases; in some severe cases, he

Symptoms on the Part of the Skin.—A symptom of polyarthritis has always been observed by physicians and was considered to be cha istic, is *excessive sweating*; since the introduction of treatment wit cylic acid this symptom is noted to be less constant. The sweat, i trast to normal sweat, shows an acid reaction; *sudamina* often occu result of the marked diaphoresis. Other exanthems: Urticaria, r herpes, purpura are rare in the course of the affection and witho nostic significance.

Condition of the Urine.—The *urine* shows but few changes wh important in the diagnosis. The amount is often considerably redu account of the great sweating; the reaction is markedly acid, the for of urate sediments being pronounced. But rarely does a decided irr of the kidney occur in acute rheumatic fever due to the infectious p and with this the development of a marked *nephritis* (compare th history just described); I have seen this condition in but few cases.

Endocarditis and Pericarditis.—Of great diagnostic importance frequent occurrence in acute rheumatic fever (in Würzburg in a one half of all cases) is a "complication" the observation of whic greatest importance on account of the prognosis, viz., an inflam affection of the heart in the form of rheumatic *endocarditis* and *p ditis*. Of these two processes *endocarditis* is conspicuously muc frequent (at least three times) than pericarditis; isolatedly, i. e., simultaneous endocarditis, the latter only occurs exceptionally in rheumatic fever. The symptoms of myocarditis also occur occasion the course of this disease. In the diagnosis of an endocarditis we n careful not to confuse endocardial murmurs with functional acc murmurs. In general, however, functional murmurs are rare in arthritis, according to my experience, and even very faint murm usually of an endocardial nature, i. e., they remain, after the rheu has run its course, as an expression of a usually lasting valve lesion origin of chronic valve lesions is, as is well known, at least in one the cases, due to a polyarthritis which has occurred during a former

According to my experience there are cases in which the *rhe poison first attacks the endocardium and only later affects the join* fact, any one who has seen many cases of recurring endocarditis, time with and at other times *without* simultaneous arthritic infl tions, will incline to the view (even if this cannot be proven with po ness up till now) *that such acute relapses of endocarditis are prin the result of a rheumatic infection.*

Complications and Sequelæ.—Compared with endocarditis, all the other cations occurring in acute rheumatic fever are rare or less important—fr previously described meningitis up to the *inflammations of the muscles an dons* which represent another localization of the rheumatic infection and oc ally, as I have seen, may precede the joint affections or entirely take their Besides the already mentioned complications, respectively localizations, the fol conditions are also noticed, arranged in the order of their frequency: *Bron pleurisy, pneumonia, peritonitis, thyreoiditis, nephritis* (see above), *cystitis thritis* (without gonococci); also *enteritis* and *dyspepsia, neuralgia* and tra *paralyses* (similar to diphtheritic paralysis) which may be distributed ov

May 28.—Since the previous afternoon *slight ptosis of the left eyelid*; patella tendon reflex markedly diminished. But the mind was clear. Patient asked for something to read to pass the time.

May 30.—Shoulder joints free from pain, and the tenderness in the cervical vertebræ had almost disappeared. However, since this day the *right elbow joint became painful.* Temperature, normal for the last three days, rose again to over 100.4° F., to fall again to normal within two days, remaining so for five days.

June 2.—Patient said that during the night he had stiffness in his extremities. During the day vomiting occurred again, the vomited material showing no HCl reaction.

June 3.—Drawing pains and light contractions in the right arm and in the finger. No headache, but vomiting, with good appetite.

June 4.—Marked delirium during the night. Patient jumped out of bed. Pains in the small of the back and in the neck. Pupils reacted promptly, but nystagmus and strabismus divergence. *The entire skull sensitive to slight taps, painful rigidity of the muscles of the back of the neck.* Decided redness of the cheeks. *Spasmodic contractions* of the fingers of the left hand; the legs were movable but more marked tonus and spasm could be noted in them. At times *uncalled-for, deep respiration, urine voided into bed.*

June 6.—Inequality of the pupils, contraction of the muscles of the lower extremities. *Abdomen retracted and hard.*

There could no longer be any doubt as to the existence of a *cerebro-spinal meningitis* (hyperæsthesia, spasmodic rigidity of the muscles of the back of the neck, strabismus, twitchings, contractures of the muscles of the extremities and of the abdomen, diminution of the tendon reflexes, delirium, headache, vomiting, involuntary evacuation of urine, etc.). *Acute articular rheumatism* is assumed as the cause of this condition, on account of the multiplicity and transitory character of the arthritic inflammations. Sepsis and tuberculosis are excluded owing to the absence of any positive point of support (negative findings in the lungs and absence of tubercle bacilli in the sputum in numerous preparations).

The diagnosis of cerebro-spinal meningitis became even more complete in the next few days by the appearance of wild delirium and psychical alienation, by the rapid loss of flesh and by collapse with strong pulse, by a slight right-sided paralysis of the facial nerve and by an excessive hyperæsthesia which again became prominent. The *pulse* which was slow until then *became extraordinarily rapid,* 160. The *temperature,* after for some days slight elevations to 100.4° F.–102.2° F. had occurred, remained continuously *normal.* The ophthalmoscopic examination showed normal conditions during the entire course of the affection.

Improvement occurred towards the end of the month of June in the cerebral condition, the patient taking more nourishment and the rigidity of the muscles of the back of the neck relaxing; ptosis and hyperæsthesia also disappeared. The urine, which at the onset contained albumin, during the middle of June showed from time to time the character of nephritic urine (epithelial casts), then became free from albumin, but during one day, July 14, it again contained great quantities of albumin; from July 15, however, it become permanently free from albumin. From the middle of July on, while the patient was up and about, and besides a slight melancholic condition showed no disturbances of the cerebral function, an exceedingly interesting agraphia and *alexia* occurred (the patient could not write or read, for example, the word Maulkorb [muzzle], although he spelled it correctly; the word Sakristei he could write without making a mistake, but he could not read it); aphasia was merely indicated in the patient. The above-described cerebral phenomena gradually disappeared in the course of three weeks; the condition of strength, which was reduced to the utmost, increased more and more, and all the symptoms which reminded us of the recovery from meningitis, disappeared rapidly so that, upon August 6, the patient left the hospital *cured.* Only the pulse remained accelerated, conspicuously labile in its frequency, so that even in the last week during the stay of the patient in the hospital variations between 100 and 135 occurred; the heart sounds remained clear from the beginning to the end of the affection.

Symptoms on the Part of the Skin.—A symptom of polyarthritis which has always been observed by physicians and was considered to be characteristic, is *excessive sweating*; since the introduction of treatment with salicylic acid this symptom is noted to be less constant. The sweat, in contrast to normal sweat, shows an acid reaction; *sudamina* often occurs as a result of the marked diaphoresis. Other exanthems: Urticaria, roseola, herpes, purpura are rare in the course of the affection and without diagnostic significance.

Condition of the Urine.—The *urine* shows but few changes which are important in the diagnosis. The amount is often considerably reduced on account of the great sweating; the reaction is markedly acid, the formation of urate sediments being pronounced. But rarely does a decided irritation of the kidney occur in acute rheumatic fever due to the infectious product, and with this the development of a marked *nephritis* (compare the case-history just described); I have seen this condition in but few cases.

Endocarditis and Pericarditis.—Of great diagnostic importance and of frequent occurrence in acute rheumatic fever (in Würzburg in at least one half of all cases) is a "complication" the observation of which is of greatest importance on account of the prognosis, viz., an inflammatory affection of the heart in the form of rheumatic *endocarditis* and *pericarditis*. Of these two processes *endocarditis* is conspicuously much more frequent (at least three times) than pericarditis; isolatedly, i. e., without simultaneous endocarditis, the latter only occurs exceptionally in acute rheumatic fever. The symptoms of myocarditis also occur occasionally in the course of this disease. In the diagnosis of an endocarditis we must be careful not to confuse endocardial murmurs with functional accidental murmurs. In general, however, functional murmurs are rare in polyarthritis, according to my experience, and even very faint murmurs are usually of an endocardial nature, i. e., they remain, after the rheumatism has run its course, as an expression of a usually lasting valve lesion. The origin of chronic valve lesions is, as is well known, at least in one half of the cases, due to a polyarthritis which has occurred during a former period.

According to my experience there are cases in which the *rheumatic poison first attacks the endocardium and only later affects the joints.* In fact, any one who has seen many cases of recurring endocarditis, at one time with and at other times *without* simultaneous arthritic inflammations, will incline to the view (even if this cannot be proven with positiveness up till now) *that such acute relapses of endocarditis are principally the result of a rheumatic infection.*

Complications and Sequelæ.—Compared with endocarditis, all the other complications occurring in acute rheumatic fever are rare or less important—from the previously described meningitis up to the *inflammations of the muscles and tendons* which represent another localization of the rheumatic infection and occasionally, as I have seen, may precede the joint affections or entirely take their place. Besides the already mentioned complications, respectively localizations, the following conditions are also noticed, arranged in the order of their frequency: *Bronchitis, pleurisy, pneumonia, peritonitis, thyreoiditis, nephritis* (see above), *cystitis, urethritis* (without gonococci); also *enteritis* and *dyspepsia, neuralgia* and transitory *paralyses* (similar to diphtheritic paralysis) which may be distributed over the

entire body. It is especially interesting that the rheumatic virus, as has already been indicated above, not rarely gives rise to insanity and that, in cases that have been absolutely determined, the origin of *chorea* is in connection with polyarthritis: I must refer in this respect to what I have said, when discussing the diagnosis of chorea, in reference to the comparative frequency of these two affections. *Septico-pyæmia* is not a very infrequent complication of rheumatic fever (see p. 968). The joint inflammations in such cases are of a purulent and of a less transitory character, the endocarditis shows the severe malignant type, and the picture of cryptogenetic septicopyæmia then develops step by step (pain in the bones, retinal hæmorrhages, etc.).

Besides the valvular lesions, the insanity and the chorea, there remain occasionally but rarely as *sequelæ*, ankylosis or *dropsy of the joints* which I have seen especially in the sternoclavicular joints. Weakness and atrophy of the muscles (probably as a result of rheumatic myositis) and the development of nodules, varying in size from that of a pin-head to that of an almond, in the subcutaneous cellular tissue, in the tendons or in the periosteum, are sometimes noted in connection with acute rheumatic fever or in the later stages of the disease. (*Rheumatismus nodosus.*)

Differential Diagnosis.—If acute polyarthritis is observed for some little time, it cannot be confounded with any other affection. That the differentiation of cryptogenetic septicopyæmia from acute rheumatic fever may occasionally be difficult, was thoroughly shown in the preceding chapter.

Gout.—Less difficulty is encountered in the differential diagnosis from *gout*, especially so if not the first arthritic attack must be judged. The concentration of the affection to *one* joint and that almost always to the metatarso-phalangeal joint of the big toe, the much severer and the more obvious phenomena of inflammation of the joint and of the periarticular tissues, as well as the ætiology scarcely allow a doubt as to the existence of this affection (compare also the Diagnosis of Gout).

Gonorrhœal Arthritis.—Mistaking a *gonorrhœal arthritis* for acute rheumatic fever, we must admit, may occur at first glance. In gonorrhœal arthritis, however, most frequently only the knee joints are affected. It is good practice, if an acute arthritic inflammation occurs with inflammation of the knee joint, at once to examine the genitalia for the presence of a gonorrhœa; in this manner we may avoid unpleasant diagnostic errors.

Peliosis rheumatica.—The arthritic affection arising in the course of a hæmorrhagic diathesis (*"peliosis rheumatica"*) may, as I know sufficiently from my own observation, under some circumstances so closely predominate in the clinical picture that for some time we may be in great doubt whether we are dealing with acute rheumatic polyarthritis, especially as purpura rheumatica may run its course with fever and endocarditis. Decisive, however, in such cases is the simultaneous occurrence of hæmorrhages of the skin (purpura occurs in acute rheumatic fever only in the rarest instances, and it is still undecided even whether such cases should not be counted as belonging to peliosis), of hæmorrhages in the mucous membranes and of internal hæmorrhages. The confusion of polyarthritis acuta with *hysteric arthritic neuroses* is impossible on account of the afebrile course and the absence of inflammatory phenomena in the case of hysteria.

Meningitis.—As has already been mentioned, acute articular rheuma-

fine as a hair. Segmentation occurs, similar to the tertian form, into from 15 young parasites which, however, under all circumstances are smaller. The cour development of the individual parasites is usually not uniform in tropical feve that small and large tropical rings are found side by side. The gamete forms themselves as "*crescents*," i. e., crescent-like formations which almost compl fill the blood corpuscles and in their further development become spindle-shaped later, egg-shaped ("*tropical spheres*")

In the Romanowsky double stain with a methylene-blue-eosin solution plasma of the parasites is coloured blue, the nuclear substance (the chromatin) In the ring the button-shaped enlargement consists of chromatin and the segm tion forms later develop from this part. In the gametes and spheres and in crescents of tropical fever a differentiation is noted between male (weakly stained protoplasm, many-threaded chromatin) and female parasites (chrom sparingly and markedly blue-stained protoplasm). From the chromatin of male parasite the flagellæ are formed, which, as we have seen, externally to human body (in the stomach of the mosquito) as spermatozoa find their way the female parasites and impregnate them, whereas in the body of the "bet host" (of man) the condition for copulation of the sexually developed indivi appears to be absent.

Development and Transmission of Malaria.—As the preceding shows, the c of the development of the malarial parasite is very completely known to-day in of its divisions, and the malarial parasite may justly be looked upon as the cause of the affection, even if the direct proof, i. e., the artificial developmen attacks of intermittent fever by inoculation of the plasmodia, has been impossibl to now, probably due to our inability to produce pure culture. On the other h the indirect proofs are even more in favour of this view. Neither in the blood o healthy nor in human beings suffering from other diseases (than malaria) has parasite in question ever been found. If the blood contains malaria parasites is injected into healthy individuals (Gerhardt, and others), the inoculated perso taken ill, after a *period of incubation* of from one to one and a half weeks, typical malaria, which is cured by quinine, identically to malaria occurrin its usual course. That the poison is transmitted by the air is hardly serio believed any longer, and, just so, the assumption that drinking-water introduces germ into the human body is no longer tenable, after the experiment to produce disease by taking water from malarial districts and giving it to normal individ in districts that are free from malaria, has absolutely failed despite the fact that individuals experimented upon drank the questionable water by the litre for w Inversely, all the experiments attempted lately force us to believe in the correct of the assumption *of an exclusive transmission of the malarial parasite, and wit of the disease, by the bites of mosquitoes.* In this respect but two facts shal mentioned: The marked success of the use of mosquito-nets in malarial regions, w protect the individuals from the bites of the anopheles and effectively prevent larial disease in this way, as well as the well-known experiment of Manson, Jr the spring of 1900, upon himself. He allowed some female anopheles which he sent to him from Italy and which had sucked the blood of Italian malarial pati to bite him in London, and in this way acquired a typical tertian attack!

Varieties of Fever.—The *clinical picture* of malarial disease is do nated by the *fever*; the onset of the same, after one to two weeks of cubation and unsteady, vague prodromes, shows the beginning of the d nosticable affection; its type, i. e., the course of the fever in distinct atta characteristically stamps the affection ("intermittent"), and determi at the same time the diagnosis of the individual malarial variety. In regard we differentiate, according to the fever occurring every day, every other day, every fourth day, etc., from ancient times an intermitt fever of a *quotidian, tertian, quartan type*; intermissions which are longer duration (from five to twelve days) have also been observed

competent physicians. *The fever constantly begins with the division of the parasite* (the sporulation). The cause of the fever in this course is unknown (destruction of the red blood corpuscles or other causes).

Quotidian fever to-day no longer is looked upon as a special type due to a separate parasite developing in one day, but it is considered as a *double tertian* or a *triple quartan form.* In the first case we are dealing with two generations of tertian parasites, in which the one sporulates twenty-four hours before the other. If in quotidian fever only quartan parasites are found in the blood, the quotidian type is to be explained by the fact that three generations are found simultaneously in the blood, which sporulate regularly, each in twenty-four hours. Much rarer is a double quartan, i. e., a quartan with two generations of parasites, which sporulate upon two succeeding days, whereas the third day remains free from fever. The *tropical fevers* appear to be true tertian forms; only the attacks are protracted, the temperature remaining high for from twenty to thirty hours, the intermissions being short, so that finally only remissions occur. If the new attack of fever sets in somewhat sooner than the last preceding one, and if this recurs regularly, we speak of a *febris intermittens anteponens,* in an opposite condition of a febris *postponens.*

Stages of the Fever.—In some paroxysms of fever we are able to distinguish several *stages,* it is true, not strictly separated from each other and not even especially characteristic of intermittent fever: *Stages of chill, of fever, and of sweating.*

The *cold stage* is characterized by a chill, by pallor and cyanosis of the skin, acceleration of the pulse and of the respiration, by nervous phenomena, such as anxiety, palpitation of the heart, dizziness, vertigo; in children, convulsions, etc. Of diagnostic importance is only the fact that during the cold stage, which usually lasts from one to two hours (rarely shorter or longer, up to six hours), the temperature of the body rises suddenly and even with the end of the cold stage or at the beginning of the *hot stage* the acme of the fever is reached (105.8° F. and over). In this second stage the temperature of the external skin, which was also low during the cold stage, rises considerably; the skin appearing turgescent, burning hot, red, but dry. The increased rates of the pulse and of respiration, as well as the nervous phenomena (headache, vertigo, etc.), remain, or increase in severity; over the lungs bronchial râles are noted, in the heart blowing systolic murmurs, as well as occasionally over the enlarged spleen; the pulse is soft, dicrotic. The urine, excreted in larger amounts during the cold stage, becomes less in quantity during the hot stage; the increased amount of urea excreted may now reach its maximum. After the hot stage has lasted about double as long as the cold stage, the *sweating stage* sets in, with a powerful diaphoresis and a moist condition of the dry mucous membranes, with a continuous fall of temperature or with very slight interruption by slight rises (to normal or to subnormal ranges), and with decrease in the size of the spleen. The duration of the sweating stage varies, lasting usually several hours, so that the entire attack of malaria lasts, as a rule, from six to ten hours. In general little of importance can be determined for the diagnosis from the peculiarities in the course of these fever stages.

Time of the Onset of the Fever.—The attacks of fever usually occur in the time between morning and midnight; they are rare during the time of sleep, therefore between nine o'clock in the evening and five o'clock in the morning. Such exceptional cases in which this occurs under some circumstances may give rise to great diagnostic difficulties in that, as occurred in one of my cases, the morning and evening temperatures show nor conditions, whereas temperatures taken during the night exhibi attacks of fever.

Enlargement of the Spleen.—Besides the course of the fever, as a tom of malaria second in importance, ***enlargement of the spleen*** is nificance in the diagnosis. The organ is almost constantly enlarge enlargement being easily demonstrated by percussion and palpation. during the prodromal period, but certainly in the cold and hot stag spleen enlarges, to return to its normal volume during the sweating and during the period of apyrexia. After frequent attacks, howeve increased size of the spleen remains, and the enlarged spleen compa

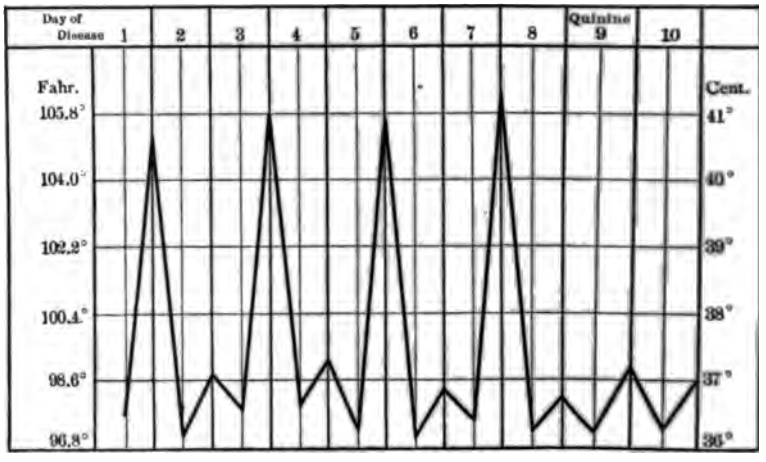


FIG. 79.—AVERAGE CURVE IN INTERMITTENT FEVER.

the splenic tumour in other infectious diseases is characterized by i treme *hardness*. The *liver* may also be enlarged, in a long-lasting ma infection it may assume very marked dimensions, and may remain c ically enlarged.

Composition of the Urine.—Less constant is the action of the malarial t upon the kidneys. As interesting as the excretory conditions of individual co ent parts of the urine are in a theoretical respect during the attacks of the and during the period of apyrexia,[1] nevertheless, they are neither constan pathognomonic, so that they play but a very subordinate part in the diagno the disease. Especially worthy of mention is the fact that, similar to other tious diseases, so also in malaria albumin is frequently found in the urine. Th tation of the kidney by the malarial poison is so marked in some instances besides albumin, especially immediately after the attacks, blood and casts a creted; occasionally accompanied with pains in the renal regions. Glycosuria i now and then noted in the course of the attacks.

Skin.—A frequent appearance in malaria is the occurrence of ***herpes*** upo face, rarely on other parts of the body. This may be of value in the diagnosis far as no other infectious diseases, excepting pneumonia and, perhaps, cerebro- meningitis, so frequently show an eruption of herpes as malaria. Roseola, pu and urticaria are also noted, but very much less frequently, in patients af with malarial fever.

[1] Regarding the details, compare my findings in "Lehre vom Harn" by Sa ski-Leube, p. 539.

Differential Diagnosis.—The diagnosis of a simple intermittent fever, with well-developed febrile attack and enlargement of the spleen, is easy and may be made with certainty, even without the proof of parasites in the blood; their determination, it is true, only giving the diagnosis absolute surety. We must now state in which direction difficulties may arise for the *differential diagnosis.* First, we must emphasize that the febrile conditions do not always show the well-developed type which has just been described. This is not only the case in persons who have suffered from malaria upon more than one occasion and in whom the later febrile attacks become less regular (*febris intermittens erratica*), but, according to my experience, also in patients who are suffering from the first attack of malaria. Frequently it may take some time, occasionally a week or longer, in such cases until the paroxysms of fever develop the typical text-book picture. The diagnosis of malaria cannot even be made with probability during the time of these irregular, varying febrile attacks; but it must rather be left undecided whether another infectious disease with enlargement of the spleen, which may be demonstrable even in this stage—typhoid, variola, etc.—or malaria is present. As soon as well-developed chills occur, and the time in which the characteristic skin phenomena take place in the acute exanthemata, therefore the first four days, has passed, the circle of diagnostic possibilities becomes smaller. Now the question to decide is usually whether we are dealing with septicopyæmia or with malarial intermittent fever.

Septicopyæmia.—But the diagnosis, even here, does not remain in the balance for a long time, as in the case of septicopyæmia, although the fever paroxysms which arise during chills also alternate with afebrile periods, yet the latter, as a rule, do not represent complete apyrexia and the onset of the chill occurs more irregularly, i. e., does not adhere to certain days or even certain hours, as in the case of malaria; nor is the enlargement of the spleen so marked and so constant in septicopyæmia as in malaria. Should inflammations of the joints, endocarditis, osteomyelitic and other metastatic phenomena take place which, it is true, may be absent from the picture of septic infection for quite a time, the confusion of this infection with malaria is no longer possible, especially as the trial administration of quinine in intermittent fever will almost always break up the attack, whereas in septicopyæmia the disease is not influenced by this drug. Naturally, in cases that are difficult of diagnosis the examination of the blood for malarial parasites should always be undertaken; the positive demonstration of the parasites always determines the diagnosis. The following case observed by me some time ago will show the great difficulties of the differential diagnosis and the importance of the blood examinations.

Case of Malaria running an Irregular Course.—An engineer, aged nineteen, never having been sick previously, coming from a healthy family, was taken ill on October 31, 1891, with nausea, vomiting, severe headache and vertigo; at the same time fever occurred, ushered in by a chill and great thirst. The patient was unable to define a cause for the affection.

Examination on November 2 showed a severe morbid picture. Evening fever 105.4° F., morning 101.4° F.; absence of patellar tendon reflex; pains in the right elbow joint; normal conditions regarding lung and heart; pulse accelerated, soft

and regular; tongue coated; no membrane in the throat. *Liver and spleen were not enlarged.* The urine, free from albumin and sugar, showed a weak diazo reaction.

November 3.—Increase of the pains in the right elbow joint which also appears to be somewhat swollen. Repeated vomiting, three diarrhœic stools. A probable diagnosis of acute rheumatic fever was made. Specific treatment unsuccessful. Temperature the same as on the previous day.

November 4.—Spleen distinctly palpable, diarrhœic stool, diazo reaction marked. This naturally turned the diagnosis to enteric fever, but upon the following day this could be definitely dropped, as there appeared:

November 6.—Well-developed herpes nasalis, and a spontaneous fall in the temperature, to 98.2° *F.* Evenings, with chilliness (with a distinct chill), rise of temperature to 104° F.; no changes in the organs were demonstrable until then, only the right shoulder joint sensitive to pressure, also the femurs. Now *cryptogenetic septicopyæmia* became probable.

November 8.—After the temperature had remained at 104° F. on November 7 for the *entire* day, it fell on the 8th to 100.4° F., rising again in the evening to 105.8° F., to fall to 99.5° F. on the 9th. Joint and bone pains had then *disappeared*; with this the diagnosis of sepsis must be abandoned, especially as no sign of endocarditis nor any other localization of a possible septic infection was present.

November 11, 12.—The headache recurred, but even now, besides the enlargement of the spleen, no organic changes were noticeable. After a spontaneous fall of the temperature occurred on the 11th to 98.2° F., and a few hours afterward the fever again rose to 104.4° F., the question of an *atypical malaria* was taken into consideration. As the patient belonged to this absolutely malarial-free neighbourhood, this assumption was improbable at the outset. In answer to the question regarding his history, where the patient had been previously, he stated, only in the neighbouring city Ansbach; after inquiring further and more exactly, it was shown that the patient had made a pilgrimage to Rome and only eight days prior to his present affection did he return to Ansbach and thence to Würzburg. *The examination of the blood for malarial parasites* which was now undertaken *gave an indubitably positive result.*

This determined the diagnosis of malaria, in spite of *stiffness of the muscles of the neck* and slight *spasms* in the hand and the ophthalmoscopic examination (Professor Michel), which made it more than probable that a beginning tubercular meningitis was present. As soon as quinine was given in decided doses, two to three times daily, 0.5 gm., the temperature steadily and definitely returned to normal inside of four days and remained so for fourteen days. During this period of fourteen days of apyrexia the patient showed marked pallor, great lassitude, somnolence and conspicuous weakness of memory.

November 27.—In the midst of relatively good conditions headache appeared with chill and a rise of temperature to 104.4° F., followed by decline to 99° F.; then again exacerbation of the fever on the same day, a rise (without chill) to 106° F. with an ensuing fall to 100.8° F. Quinine now administered again produced an afebrile period lasting fourteen days. On December 19 again a recurrence of the fever set in for two days, then *permanent* apyrexia. Patient left the hospital on January 14, 1892, with a spleen which is still distinctly palpable.

As may be seen from this clinical history, the examination of the blood for plasmodia made the diagnosis possible, which, on account of the atypical fever course which did not show a pure intermittent type, the marked pain in the bones, the plainly pronounced symptoms of meningeal irritation, would not have been looked upon by me formerly as due to malaria but probably erroneously as due to cryptogenetic septicopyæmia! But it must be mentioned that the examination of the blood only in the first febrile period revealed malarial parasites, not, however, in the time of the relapses.

Tuberculosis, etc.—Confounding malaria with cases of *tuberculosis* that run their course with hectic, rhythmical fever may be conceived, but can only occur in practice upon a very superficial examination. Regular

attacks of chills, rhythmic-fever paroxysms alternating with apyrexia are absent in uncomplicated cases of tuberculosis, and palpable enlargement of the spleen, excepting rare cases, does not occur. Above all, however, the exact examination of the individual organs, especially of the lungs, the larynx, etc., is the surest method to avoid gross diagnostic errors in this respect. A certain similarity may exist between attacks of malaria and the febrile attacks occurring in *cholelithiasis* and *abscess of the liver*; but by a careful examination lasting some little time these errors may be easily omitted.

Varieties of Malarial Fever.—Malaria does not always run the typical course which has been described, showing the febrile attacks and enlargement of the spleen. But the organism rather reacts occasionally from the onset with a non-intermittent fever and with other irregularities in its course (*febris remittens et continua*), in other cases with an excessive development of the usual intermittent symptoms and of especially severe accompanying phenomena (*febris intermittens perniciosa et comitata*). Frequently, finally, the infection with malaria does not show itself at all by fever symptoms, enlargement of the spleen, etc., but only by the occurrence *of nervous phenomena*, neuralgias, etc., showing a certain periodicity, the connection of which with malaria can only be demonstrated by the fact that these phenomena occur in malarial districts alongside of the usual cases of intermittent fever and promptly disappear under quinine treatment (*febris intermittens larvata*). We shall devote a short space to the description of the diagnosis of these forms of malaria.

Malaria Larvata.—To begin with the last-mentioned malaria larvata, this shows itself most frequently in the form of *neuralgias*. The supraorbital nerve is most frequently affected, less frequently the intercostal nerves, the sciatic nerve, etc. These neuralgic attacks due to malarial affection occur most frequently in the spring; according to my experience they show, similar to the fever paroxysms, quite a distinct type of appearance, usually occurring with great regularity certain hours of the day. This, however, by no means determines the malarial nature of the neuralgia, as neuralgias of other origin also occur intermittently. Not even the disappearance after the administration of quinine is an absolute proof of the malarial character of the neuralgia, as quinine also occasionally shows an almost specific effect in other forms of neuralgia (although rarely very markedly).

A case in my practice may illustrate this. The patient, living in a district entirely free from malaria, was attacked by a very stubborn form of supraorbital neuralgia. As the neuralgia recurred in distinct intervals, the spleen was enlarged and the patient during a long journey subjected herself to a malarial infection; a diagnosis of intermittens larvata was made and quinine ordered. Apparently brilliant success; but after a little while the quinine was no longer effective in combating the neuralgia. In her anxiety the patient went to a dentist; after the removal of a carious tooth the neuralgia disappeared spontaneously and never recurred!

It is more important if enlargement of the spleen, chills and rise of temperature occur simultaneously with the neuralgia. Unfortunately, however, the latter symptoms are by no means constant accompaniments of malarial neuralgia, so that, in a not small number of cases, it is true, the physician must content himself with a probable diagnosis. Usually the diagnosis of masked malaria is looked upon as correct if the doses of quinine that have been administered abort the malaria and the affected patient had previously exposed himself to a malarial infection. That this diagnostico-therapeutic method does not always show the most certain results, the history quoted above proves; but in practice we must content ourselves with this method for the present, all the more as the result of the examination of the blood for parasites *is negative as a rule* in these masked malarial forms.

The same diagnostic criteria which are determining for the diagnosis of masked malaria appearing as neuralgia, are also necessary in those cases i
diseases of the nervous system arise in an intermittent manner and n

in connection with the malarial infection. An innumerable number of morbid c ditions has been described as masked malaria: Paralyses, cramps, hysterical attac vaso-motor disturbances of all kinds (erythema, cutaneous œdema, dropsies of joints), asthma, nervous dyspepsia, diarrhœa, etc. Whether we are justified referring one half of pathology to this ætiological basis, I cannot decide; bu should like to be permitted to doubt whether the great mass of these morbid co tions which are described as varieties of malaria are actually the result of a mala infection, and I should like to advise that care be taken in making such a sweep diagnosis. I have noted, besides a neuralgia, although I have paid special atten to it, but very little of these masked forms of malaria in malarial districts.

Pernicious Intermittent Malarial Fever.—The diagnosis of *pernicious inter tent fever* is identical with the diagnosis of the simple form and does not give to difficulties provided we remember that, in a conspicuously severe infection or i very debilitated condition of the individual, certain symptoms occur in the mo picture with pernicious intensity, or that severe local affections arise under influence of the malarial toxine and give rise to a fatal issue. Such severe phen ena are: Deep syncope during the fever paroxysms, coma, apoplectic and epile conditions, an icy coldness in the stage of chill, colliquative sweats in the th stage, cardiac asthenia, severe collapse, intermittent pneumonias and pleurisy, Some few of these symptoms, especially the severe cerebral phenomena, are undo edly due to the changes of hæmoglobin into melanin by the parasites and to subsequent disturbance of function of the red blood corpuscles which are necess to life, so common in these pernicious malarial forms.

Remittent Fever, Tropical Fever.—The *remittent malarial forms* can very ea be confounded with other affections. They only occur in severe malarial epide and are not common in the temperate zone. The remittent curve is especially ch acteristic of *tropical fever*. As has already been remarked, the course of the fe is more protracted than the intermittens produced by the large parasites; the de vescence of the fever is of but brief duration or incomplete, so that intermissi no longer occur but only remissions. The chill which is so characteristic of simple forms of intermittent fever, is absent in tropical fever, as a rule, and the enlargement of the spleen is not always pronounced. On the other hand, general phenomena are the more severe: Unbearable headache, delirium, etc. diagnosis of tropical fever may be made with certainty to-day, provided we succ (which, of course, is not always the case) in finding the previously described, spec small parasites, the tropical rings, and, above all, the "crescents" which are s to belong alone to the tropical varieties of malaria in the blood of patients.

Black-Water Fever.—In some cases of malaria of tropical and subtropical co tries fevers are noted running their course with severe nervous phenomena, jaund and, above all, with hæmoglobinuria (*febris biliosa-hæmoglobinurica, black-wa fever*). It may be subject to discussion whether the wholesale destruction of the blood corpuscles under such circumstances is brought about by the malarial infect itself, or whether it is the result of a quinine intoxication (R. Koch) if quinine h been administered to individuals whose power of resistance was debilitated by t tropical climate and by a previous malarial infection. In all cases, however, quini plays the most important ætiological *rôle* in the causation of hæmoglobinuria.

Malarial Cachexia.—Under the influence of long-continued attacks of mala there develops in some individuals in malarial regions a severe cachexia which h been designated malarial cachexia. The patients suffer from the symptoms of seve anæmia: A waxy pale colour of the skin, dyspnœa, palpitation and dilatation of t heart, anæmic heart murmurs, œdema and hæmorrhages. These latter, as well intercurrent dyspepsias and diarrhœas, make the condition worse and worse, so th such persons finally succumb to general weakness or to tuberculosis, gangre amyloid degeneration of the organs, etc. The differentiation of this malarial cache from other forms of cachexia and anæmia is done principally by the *blood findi* This may be positive, i. e., there may be found parasitic forms capable of segment tion, or crescents; under other conditions it may be entirely negative. In such cas by paying attention to the ætiology and by the determination of a chronical enlarged spleen and liver which cannot be attributed to any other cause than to

chronic malarial affection, we shall be able at least to make a probable diagnosis of a cachexia due to a chronic malarial affection.

INFLUENZA, LA GRIPPE

On account of the pandemic of influenza which travelled over the world in the winter of 1889-90, this disease has again become the subject of universal interest. Upon the basis of my own experience I must designate influenza as a *contagious* disease, a fact which finds its confirmation in the biological qualities of the influenza virus, and which must occasionally be taken into consideration in the diagnosis.

The *specific cause of the infection is considered to be the bacillus influenzæ, discovered by R. Pfeiffer, in 1891, in the sputum of influenza patients*; it is very small, not staining according to Gram, but may be easily stained with a diluted carbo-fuchsin solution, and it may be cultivated upon blood-containing agar, in this manner forming very small, not confluent colonies. The bacillus of influenza possesses quite marked properties in producing inflammation and pus formation, and in the human organism gives rise to purulent pneumonias, pleurisies, etc.; as a rule, the inflammatory-purulent affections occurring in the course of influenza are due to *mixed infections* with pneumococci and streptococci. Influenza bacilli have often been found in the blood also. In a dried condition they rapidly perish, so that a transmission to great distances by means of the air is not possible.

Symptoms of Value in Diagnosis.—After an always *very brief period of incubation* (the maximum being from one to three days) and vague, frequently unnoticed, prodromal phenomena (muscular and arthritic pains and frequently also pains in the small of the back, lassitude, and debility), the affection begins with *fever*, setting in with chilliness or with a *decided chill*; only in exceptional cases does the disease run its course without fever. *The course of the fever is by no means typical*; the temperature is usually between 102° F. and 104° F., more rarely between 104° F. and 106° F.; *the rise in temperature* occurs abruptly, i. e., reaching its acme upon the first day, or it may rise step-like with morning remissions of about one to two degrees. The *defervescence* also occurs in various ways, sometimes by lysis, occasionally by crisis: In some cases, a few days after the fever has fallen, a rise in temperature of brief duration in the form of a *sequel fever* occurs. The duration of the febrile period averages about three to four days, but rarely over a week.

Influenza is characterized in general as an infectious inflammation of the various organs, especially of the mucous membranes of the respiratory and of the digestive tracts, with simultaneous toxic disturbance of the nervous system, thus causing at one time one group, at other times another, of morbid phenomena, most frequently catarrh of the upper parts of the respiratory tract, to become prominent and to give rise to great variations in the morbid picture in individual cases; hence, various forms of influenza have been differentiated: A (respiratory) *catarrhal*, a *gastro-intestinal* and a *nervous, form*. The physician must be familiar with these chief forms of influenza, but he must especially understand *that there are also cases of influenza which run their course without catarrhal and inflamma-*

tory pnenomena, in which the influenza toxine may either give rise to only a mild general disturbance in keeping with the fever ("pure influenza fever"), or to a more marked irritation of the cerebro-spinal nervous system (producing the previously mentioned nervous variety).

General Morbid Picture.—The general appearance of the patient usually shows the picture of a mild, rarely that of a severe, affection. In the *eyes* a *moist glance* is noted, the conjunctivæ are inflamed, the lids are occasionally œdematously swollen, the movement of the bulbi is sensitive; herpes is sometimes noted upon the cornea. *Exanthems* are not very frequent in general, according to my observations in about one sixth of the cases, showing themselves as erythema, roseola, urticaria and herpes. Comparatively frequently (in about one third of the cases) *angina* of varying intensity occurs, also laryngitis, coryza and, above all, *bronchitis* (in over one half of all cases), with a wide dissemination of numerous râles and with an expectoration which, however, is by no means in keeping with this condition. This is sometimes accompanied with a conspicuous dyspnœa. Less frequent, aside from the constant loss of appetite, are phenomena on the part of the *digestive tract:* Vomiting and diarrhœa, severe enteritis with muco-hæmorrhagic dejecta, and eventually also peritonitis. The *spleen,* in the majority of cases, is *not enlarged*; but it would be wrong to consider that this condition is at all characteristic of influenza; for an *enlargement of the spleen* could be noted in the course of the affection *in about one sixth of the cases in my clinic,* and a rapid diminution in the size of the organ during convalescence *could be certainly demonstrated by means of palpation.* In a small percentage of the cases (not even ten per cent) albumin occurs in the urine, probably as a result of the action of the infectious products in the kidneys, which in some few cases, acting more vigorously, gives rise to *nephritis,* with the excretion of blood casts and epithelial casts in the urine; influenza bacilli were found in the kidneys, and glomerulo-nephritis, similar to the condition after scarlatina, was observed at autopsies. The action of the infection upon the *nervous system* is especially marked in most cases, in the form of the almost never absent pains in the head, extremities and joints; further, by vertigo, tinnitus aurium, muscæ volitantes, somnolence, or also by agrypnia, delirium, convulsions and maniacal attacks. The *heart* is also affected by the toxine of influenza, at one time the disturbances being of a nervous character, at other times the result of an intoxication of the heart muscle; tachycardia, bradycardia, arrhythmia, angina pectoris, and cardiac asthenia were observed. The latter most certainly is not due to the fever alone, as cases are noted in which, in spite of very marked fever, the cardiac activity remains intact, whereas systolic murmurs, etc., occur in other cases with slight fever. Especially patients affected by other chronic diseases are those who perish under the influence of la grippe, due to increasing cardiac weakness, most likely on account of their cardiac energy having suffered from the underlying affection. Endocarditis, which may give rise to permanent valve lesions, has also been noted. Numerous observations regarding arterial and venous thromboses in the course of influenza have been reported, also hæmorrhages from the most varied parts of the body (epi-

staxis, metrorrhagia, intestinal hæmorrhages, purpura, etc.), probably due to a toxic fatty degeneration of the walls of the vessels.

Complications.—The usual picture of influenza just described is subject to many changes by the supervention of so-called *complications*. Especially are some organs affected in which not only the action of the influenza bacilli, but also (due to a mixed infection) the action of streptococci and pneumococci gives rise to severe diseases; according to the experiences of the last epidemic, the pulmonary complications seem to be most marked. *Pneumonias* are relatively frequent in the course of influenza (from 5 to 8 per cent with a mortality of about 20 per cent). Not infrequently do these arise in the form of the usual *croupous* pneumonia, with an initial chill and rusty sputum. In the majority of cases, however, it is a question of atypical pneumonia with moderate fever, small frequent pulse, dyspnœa, cyanosis, and severe general phenomena of a so-called "typhoid" character, with but slight clinical local phenomena on the part of the lungs (indistinct dulness, crepitant râles, slightly rubiginous or non-hæmorrhagic, purulent sputum), which post mortem are in keeping with lobular, widely distributed areas of infiltration, most frequently in several lobes of the lung. After the experience gained in the last years, there can no longer be a doubt that the pulmonary inflammations observed in the course of influenza are of the most varied forms of pneumonia: Pure catarrhal pneumonia due to the specific influenza bacillus, streptococcus and pneumococcus pneumonias, but, above all, mixed infections involving co-operation of the various generators of pneumonia. The differential diagnosis can only waver between the assumption of the above-described forms of pneumonia and that of a miliary tuberculosis which may have occurred in connection with the disease (compare the diagnosis of the latter). In other cases, as noted by myself and others, *abscess formation may develop in the lungs*; the pus of the abscess showed pneumococci, streptococci, and staphylococci, and also the specific influenza bacilli, after an atonic pneumonia had previously affected the pulmonary tissue. To this a *purulent pleurisy* may be added, which was comparatively frequent in the last epidemic both due to pneumonia and as an independent sequelæ of influenza. A second organ in which complicating purulent infections were comparatively often noted, is the *ear*. Here are found partly simple swellings of the mucous membranes of the tubes and of the tympanic cavity, partly exudations into the tympanic cavity with subsequent perforation of the drum. Rarely does this otitis media run its course without pus formation. It is usual for the pus to collect in the middle ear in which, by means of bacteriological examination, pure cultures of the streptococcus pyogenes or of pneumococci may be obtained. The inflammation may affect the bones and give rise to a purulent sinus phlebitis and meningitis.

Sequelæ.—The majority of the numerous *sequelæ* observed in the last influenza epidemic belong to the *nervous system*. These are especially, similar to what occurs in other infectious diseases, debilitating and paralytic conditions, paralyses of the muscles of the eye, etc.; further, cerebral affections (meningitis and especially encephalitis), diseases of the spinal cord (myelitis, tabes, etc.), neuritis (with muscular atrophies), neuralgias, psychoses, epilepsy, hysteria, etc., which all have been noted in connection with influenza and due to this cause. A part of these nervous affections is undoubtedly due to the influenza bacilli and their toxines, the spe Pfeiffer bacilli upon several occasions having been found in the brain and cord! But whether the entire large series of nervous disturbances and also wh else (tuberculosis, diabetes, etc.) has been explained as a sequelæ of influe is daily so explained, actually is in a genetic connection with influenza is able, all the more so as the diagnoses "influenza" are frequently superficia an uncertain nature.

During the occurrence of an epidemic the diagnosis is very easy cially in cases in which a certain complex of the phenomena occurs taneously: Catarrh of the nose and bronchial tubes, accompanie marked implication of the general condition, headache and pains

extremities, etc. The diagnosis becomes more difficult when the usual ac mulation of symptoms is absent and only nervous or gastric phenomena pear. Certainly every one who is at all strict with his diagnosis has had same opinion as I have that, during an influenza epidemic, many diagn which were attributed to influenza rested upon a very weak foundation even later, after the affection had run its course, remained questionable were proven to be erroneous. The control of the diagnosis by bacte logical examination is, as need scarcely be mentioned, especially in a ease like this which is of such short duration, occurring epidemically pandemically, only possible in the rarest cases in practice.

Differential Diagnosis.—From a *differentio-diagnostic standpoint* th must be considered, principally, enteric fever, measles and scarlet fe simple coryza, or bronchitis and miliary tuberculosis. A beginning att of *enteric fever* can sometimes only be excluded after a few days, i. after the temperature has fallen, especially if influenza has set in witl step-like rise in temperature and with diarrhœa, roseola, and enlargem of the spleen. To think of enteric fever is all the more justified as e staxis is also not infrequent in influenza, and the initial nervous gene symptoms are common to both affections. The diagnosis is usually aright in such cases by the appearance of coryza and conjunctivitis wh occur in influenza, as well as by the absence of a relative decrease in t pulse rate, which is characteristic of enteric fever. Influenza differs fr *simple coryza and bronchitis* by the suddenness of its onset, its rapid cou and the intensity of its nervous disturbances, eventually by the enlar ment of the spleen and the appearance of eruptions. The differential dia nosis between *measles* and influenza may become very difficult. Co junctivitis, laryngobronchitis, angina and frontal headache, etc., are co mon to both affections; the absolute diagnosis can only be made upon t fourth day, as a rule, if, after a fall in temperature, a new rise takes pla and the eruption of the exanthem shows the presence of morbilli. In t first days, above all, the reddening of the mucous membranes of the pala and pharynx is to be noted, which in the prodromal stage of measles n infrequently appears as small patches and allows the swollen follicles become prominent as minute nodules. Finally, also the question wheth acute *miliary tuberculosis* or influenza is present must be seriously tak into consideration, especially when tubercular patients are affected by i fluenza. Principally the observation of the prominence of conjunctivit and coryza makes the diagnosis certain in such cases. It should also mentioned that, when phthisical patients are affected by influenza, t tubercular process very frequently is unfavourably influenced and brought to a more rapid development.

TETANUS—LOCKJAW

Tetanus is a genuine infectious disease, the causation of which is abs lutely known to-day. This may be looked for in the tetanus bacillus di covered by Nicolaier in the dirt, and pure cultures of which were obtain by Kitasato, and the transmission of which upon animals results in

inoculated tetanus. The latter is also successfully accomplished by inoculating the chemical toxines obtained from cultures of tetanus bacilli. By immunizing the animal first with a weakened and later with a gradually increasing toxic dose, it is possible to produce in animals an "immunity to the poison," i. e., to make them insusceptible to the injection of the tetanus toxine and great quantities of cultures of tetanus bacilli.

Tetanus Bacillus.—The tetanus bacilli represent large and movable staffs presenting a "button-like" thickening at one end and spore formations. The bacillus is anaerobic, flourishing upon various culture media; the spores are found, as has been demonstrated, in the superficial layers of garden earth, in dung heaps, etc. *In the internal organs* of animals that have been inoculated with tetanus bacilli and that have perished under tonic convulsions, not only all gross pathological changes are *absent* but, *above all, also the bacilli themselves.* It must be assumed, therefore, that the bacilli remain at the point of invasion and that from here, by extraordinarily toxic products of metabolism, invade the body.

The importation of tetanus bacilli into the body occurs, as a rule, by skin wounds (frequently small ones) becoming contaminated by earth, particles of brick, cement, dung, by dirty fingers, etc. Tetanus, therefore, must be designated in general as a disease of exquisite wound infection (*tetanus traumaticus*). The same as in the case of surgical erysipelas and in septicopyæmia, there are also cases of tetanus in which the place of the invasion of the bacilli cannot be determined, the disease therefore apparently arising spontaneously (*cryptogenetic, idiopathic, rheumatic tetanus*). Perhaps the infection occurs by means of the mucous membrane of the respiratory tract, in the secretion of which virulent tetanus bacilli have been found in rheumatic tetanus.

The designation of these cryptogenetic forms of tetanus as "*rheumatic*" is justified in so far as it has been determined for some time that atmospheric influences unquestionably have something to do with the production of the affection (probably a predisposing influence). Psychical factors, injuries to nerves, and marked cerebral disturbances may also predispose to an outbreak, or at least increase the intensity, of the affection, in so far as the reaction of the nervous system to the tetanus toxine, which acts especially upon the central apparatus similar to strychnine, may on this account be more pronounced. Opportunity for infection, besides accidental skin wounds, is given by lesions occurring during the puerperal period (*puerperal tetanus*) and, further, in the case of the new-born by the umbilical stump (*tetanus neonatorum*), etc. All these ætiological conditions must be taken into consideration in the diagnosis of tetanus in the given case.

Diagnostic Value of the Tetanus Bacilli.—The bacilli, as has already been stated, are never found in the internal parts of the bodies of inoculated animals and they are only occasionally met with post mortem at the point of infection; it is obvious, therefore, that the demonstration of the specific tetanus bacilli can only be utilized to a limited extent in the diagnosis. As important, and as the last resort determining, as the *positive* proof of tetanus bacilli in the secretion of wounds may be for the diagnosis, it is nevertheless positive, according to the above statement, that the *clinical picture* of the disease in some cases is sufficient to determin
nosis. But this is also indirectly of decisive importance in
of those cases in which the finding of the tetanus bacilli is

far as only after the characteristic phenomena of the affection have occurred the physician is stimulated to look for tetanus bacilli.

Diagnostic Symptoms.—The phenomenon which dominates the entire picture of tetanus are the *tonic muscular spasms,* which, beginning with slight, drawing pains and occasionally with stiffness of the muscles, soon increase to a frightful extent and may affect the greater part of the muscles of the body in succession. First the muscles of the jaw, the muscles of deglutition and of the tongue, then the muscles of the neck and of the body, and, finally, although more rarely, the muscles of the extremities; especially rarely do the cramps extend to the lower part of the arms and to the hands. Usually tetanus sets in with a spasm of the bones of the jaw (*trismus*) and in some few cases, especially in the new-born, it remains confined to the muscles of deglutition. Due to the tension of the muscles of the face the whole expression changes and this in a highly marked manner (facies tetanica): The corners of the mouth are drawn downward and outward, the openings of the nose are widened, the palpebral fissure is narrowed, the forehead wrinkled, the borders of the masseter muscles stand out as stiff bands. This gives the expression a somewhat tense, rigid appearance; under some conditions the face of the patient affected by tetanus shows the terrible picture of the "risus sardonicus." Due to the impossibility of separating the tightly closed jaw, the result of spasm of the masseter muscle, the speech becomes unintelligible, the taking of food (even sucking) difficult; it is absolutely impossible, finally, to administer nourishment if to the trismus is added a spasm of the muscles of deglutition.

Especially characteristic is the position of the body caused by spasm of the extensors of the back (*opisthotonus*), the muscles of the abdomen are board-like in their tension, the head is drawn backward. Directly dangerous to life are the consequences of spasm of the muscles of respiration in that the patients show violent sensations of oppression, becoming cyanotic and finally choke; death by suffocation is threatened especially if the muscles of the larynx take part in the tetanic spasm and spastic closure of the glottis occurs.

The spasms are usually tonic, continuous, only ceasing during sleep; in other cases intermissions occur also during the day, at least a relative cessation of the tension, which, however, rapidly gives place to new spasms (partly also clonic), as soon as only the slightest sensory irritation takes place. The reason for this may be looked for in the more marked irritability of the sensory nerves; however, this is not the case, according to the result of newer investigations, in that inoculation tetanus could also be produced after cutting of the posterior roots. We must assume rather *that the tetanus toxine possesses a specific affinity for the cells of the anterior horns and produces increased irritability in them.* The tetanic contraction of the muscles gives rise to pains which may also be produced, respectively increased, by pressure upon the tense muscles; but, on the other hand, the sensations of touch and of temperature appear diminished rather than increased; the mind usually remains clear up to the time of death.

The *temperature of the body* shows a very varying course, occasionally

being normal, at other times, especially in the premortal stage of the disease, markedly raised (even up to 111.2° F.); it is not possible up till now to give a satisfactory explanation of this. The *pulse* is usually small and, especially during the paroxysms, frequent; occasionally slowing of the pulse is also noted. Special attention has been paid to the excretion and composition of the *urine* in tetanus. In general the patients void a sparse, highly concentrated urine which contains occasionally, probably due to the specific irritation of the medulla oblongata, albumin, rarely sugar. The excretion of urea occurs independently of the spasmodic attacks, and is not increased in amount (in keeping with the physiological fact that muscular activity does not go hand in hand with a more marked decomposition of albumin); neither is the excretion of creatinine and of phosphates increased. According to this, nothing positive is to be gained for the diagnosis by the chemical condition of the urine. On the other hand, the tetanus toxine appears to be excreted by the kidneys, as Bruschettini was successful in producing tetanus in animals into which he injected the urine of human beings affected by tetanus. The passage of urine may become very difficult owing to spasm of the sphincter, and for the same reason and especially on account of spasm of the abdominal muscles, the excretion of fæces may become impossible. The *secretion of sweat* is usually permanently and markedly increased.

Local Tetanus.—Whereas tetanus more or less involves the entire muscular structure in the majority of cases, there are also cases in which only those muscles are affected by spasm that are in the neighbourhood of the point at which the toxine was introduced into the body, a fact which is in keeping with the spasms occurring in experimental inoculation tetanus. As a relatively frequent form of this "*local tetanus*," we observe "*head tetanus*." This occurs after head injuries and is distinguished by spasmodic contractions of the muscles of the face on the side of the trauma, by paralysis of the facial nerve, by trismus and spasm of the muscles of deglutition. Unilateral affections of muscular groups of the body and extremities, arising from the point of infection, have also been noticed, yes, in rare cases even a unilateral spasm of the muscles affecting one side of the body (*hemitetanus*). The occurrence of local tetanus is explained with difficulty. Perhaps the tetanus toxine, as Stinzing has lately made probable, is carried to the spinal cord along the nerves which are adjacent to the point of infection, and there sets up an irritation at first only of those cells of the anterior horn which are nearest the point of entrance of the poison. In human beings, however, the toxine appears to diffuse rapidly so that, as a rule, not local but general tetanus is the result of an intoxication of the spinal column.

Differential Diagnosis.—The tetanic spasms are so typical in tetanus that a *differentio-diagnostic consideration* is necessary only of those diseases in which well-developed spasms are also noted.

Meningitis.—The confusion of tetanus with *spinal meningitis and cerebro-spinal meningitis* is possible as the rigidity of the muscles of the back of the neck and the spasmodic contraction of the extensor muscles of the back, etc., which are accompanied with pains, are common to both affections. However, the diagnosis will not remain long in the balance—in favour of tetanus, in contrast to meningitis, are the absence of fever, the prominence of trismus, and the greatly increased reflex irritability, the absence of hyperæsthesia, of paralyses and of cutaneous eruptions. If it

be a question of the cerebro-spinal form of meningitis, it is difficult to confuse the two affections, in that headache, vomiting, paralysis of the muscles of the face and eyes (in so-called "head tetanus," as a rule, also a paralysis of the facial nerve would occur on the side of the lesion), involuntary evacuation of fæces and urine, inequality of the pupils, disturbance of consciousness, and, finally, the characteristic ophthalmoscopic findings, are directly in favour of meningitis.

Muscular Rheumatism.—A confusion of tetanus with *rheumatism of the muscles of the neck and back* is scarcely possible. The absence of increased cutaneous reflexes, of trismus, of facies tetanica, of cyanosis, and of the more marked secretion of sweat; further, the exquisite painfulness of movements in muscular rheumatism, the limitation of the affection to some few muscles, which is but extremely rare in tetanus, etc., guard against errors in diagnosis. Besides, the stiff attitude of the neck and back in muscular rheumatism is forced by the pains which every movement produces, and is not caused by spasm.

Hysterical Spasm.—More readily may a *hysterical spasmodic condition* at first sight resemble tetanus. Upon closer investigation, however, a diagnostic error can be easily avoided. In tetanus muscle after muscle is affected by spasm, the spasmodic contractions of the extensors developing in a uniform succession almost conforming to a law; in hysteria, on the other hand, a certain irregularity, a polymorphic condition, appears in the morbid picture, something explosive in the appearance of the spasms, and certain nervous symptoms are intermingled with the spasms: Sobbing, singultus, etc., which are foreign to tetanus. But, above all, psychical phenomena do not occur in the latter condition, whereas in hysteria there is always a more or less altered psychical condition which is susceptible to suggestion. Further, fever and accelerated pulse are absent in the case of hysteria, whereas the former may be absent in tetanus, but in other cases reaches the highest grades. Disturbances of sensibility are nearly always well pronounced in severe hysteria; they are but scarcely indicated in tetanus. It is not necessary to enter into further differential points; any one who has had the slightest experience in the observation of the morbid picture of hysteria will note slight signs here and there which this picture possesses, and will rarely confuse it, or if at all but for a very brief period, with the typical severe morbid picture of tetanus.

Spasm of the Muscles of Mastication—Trismus.—The same is true of the rare substantive affection known as *tonic spasm of the muscle of mastication*. It is true, trismus is occasionally prominent in tetanus, but other muscles are always affected at the same time, especially the muscles of the neck and face and the muscles of deglutition.

Strychnine Poisoning.—Of practical importance is the differentiation between infectious and toxic tetanus, especially that due to strychnine poisoning. Primarily the ætiology is important; farther on, certain differences in individual phases of the affection. In favour of strychnine poisoning are in general the prominence of spasms in the extremities, especially also in the hands, the greater distinctness of the intermissions, the unquestioned dependence of the spasm upon an increase of the reflex

irritability from the onset, and, finally, the brief duration, persisting usually not longer than a few hours, of the severe phenomena, whereas infectious tetanus frequently lasts for a week, on an average from five to ten days, before the fatal termination occurs. As the tetanus toxine, as has been positively proven by experiment, circulates in the blood of tetanus patients, this fact may be utilized in questionable cases of tetanus, according to the process of Kartuli. A small quantity of blood is drawn from the patient, great care being taken to obtain it as sterile as possible. From the serum white mice receive from 0.3 to 1.0 cc. by intraperitoneal injection. If tetanus occurs in these animals in from twenty-four to forty-eight hours after the injection, the diagnosis of infectious tetanus in the human being may be considered as determined.

Occasionally the spasms of deglutition are more markedly developed in tetanus patients; then the question may arise whether we are dealing with tetanus or hydrophobia. The differentiation of the latter from tetanus, however, does not show any real difficulty, as will be seen from the following description of rabies.

HYDROPHOBIA, RABIES, LYSSA HUMANA

Hydrophobia is also unquestionably a genuine infectious disease, its specific micro-organisms having probably been discovered recently by Bruschettini, who has found a bacillus which was susceptible of cultivation and inoculation. This disease never occurs spontaneously, is not transmissible from person to person but only by the bite of rabid animals (of dogs, cats, wolves, foxes, more rarely from herbivorous animals—cattle, etc.), or that the blood or *saliva* of a rabid animal in which the toxine is contained in large amounts, is brought in contact with a wound of an individual. The lyssa toxine is found especially, according to Pasteur's investigations, also in the central nervous system of the infected animal, a fact which gave rise to the celebrated Pasteur method of preventive inoculation against hydrophobia with emulsions of the medulla of animals that had died of hydrophobia. Whether hydrophobia occurs after an infection with the lyssa toxine, depends upon the individual predisposition of the infected individual; further, upon the circumstance whether the poison has directly entered the wound or has become attached to the clothes, etc. The affection is characterized by *a long period of incubation* (averaging seventy days, in rare cases even a year or more).

Clinical Picture.—The diagnosis must always take these ætiological points into account, and must not only consider the so very characteristic *morbid picture.* As a rule, the onset of the affection is noted by pain and swelling at the point of lesion, and drawing pains or paræsthesia, by arthritic pains, lassitude, eventually by frequent sneezing (provided the affection is due to a bite in the face), and, occasionally, by mild fever; at the same time there is general unrest and fear of the occurrence of the disease. Soon afterward (one to three days) the *hydrophobic or convulsive stage* sets in, with *spasms of inspiration and deglutition.* As soon as the patient makes an effort to swallow fluids or solids, a deglutition or inspiratory spasm of brief duration occurs, with a marked sinking of the diaphragm and a high grade of dyspnœa. At the same time a deathly fear is noted in the features of the unfortunate patients; they throw the head and shoulders back until an expiration produces a relaxation of the spasm. The experience that the latter condition occurs with every effort to drink, gives rise to the "water fear," causes the patient to avoid swallowing the saliva and forces him to expectorate it in brief intervals. Later the mere appearance of a glass, of any shining object, a breath of air, bright light, touching the skin, breathing upon the skin, etc., are sufficient to produce *reflex spasms.* Besides the inspiratory and deglutition spasms, tonic contractions of the muscles of the back and chronic spasms in various parts of the body occ

This is supervened, finally, by characteristic signs of morbid *psychical excitation*: Delirium, hallucination, and actual *attacks of fury*, in which the patients struggle with their hands and feet, attempt to injure those standing around and with this display snapping, biting movements. In these attacks, which may last for half an hour or longer, death may occur from suffocation; usually however the patient collapses in the course of a few days and death occurs under the picture of general exhaustion and paralysis. The *temperature*, especially towards the end, is *raised*, the pulse is small and irregular; the urine contains albumin, and frequently sugar (which is important on account of the relation that hydrophobia bears to the nervous system, especially to the medulla oblongata).

Differential Diagnosis.—If this typical clinical picture is borne in mind and if the history of the case is known, the diagnosis presents no difficulties. In a differentio-diagnostic respect only *tetanus* is to be considered in which spasms of the muscles of deglutition occur also, whereas, on the other hand, opisthotonus and other tetanus symptoms may also be present in the course of hydrophobia. The prominence of the spasms of the muscles of deglutition and respiration, varying with intermissions in which the spasm is entirely absent, as well as the conspicuous presence of psychical phenomena in hydrophobia, quite apart from the ætiology of the individual case which, as a rule, is clear, permit us to differentiate between rabies and tetanus without much hesitation. In a questionable case the examination of the blood for the presence of the tetanus poison, as previously mentioned, will show whether tetanus is present or not. If a person is bitten by an animal in which hydrophobia is suspected to exist, it is very questionable, as has already been indicated, even if the presence of hydrophobia be later demonstrated in the animal, whether the affected person will develop hydrophobia at all. But as rabies, as is well known, is an affection which, prior to the introduction to the Pasteur immunization method, showed an almost absolutely lethal prognosis, it is self-evident that the affected individual, expecting the outbreak of the disease, will be in mortal fear, and that, if it should occur in people with an easily irritated nervous system, a condition of most marked psychical excitation, accompanied with hysterical spasms, may arise, which may closely resemble an attack of hydrophobia (*lyssophobia*). The long duration of such periods of fear and spasms, the observation that no rise in temperature occurs for weeks, and that the expected dangerous collapse does not take place, further, the perception that these hydrophoboid symptoms are intermingled with true hysterical phenomena and that the entire clinical picture shows marked exaggeration, etc., will prevent confusing a pseudo lyssa with true hydrophobia.

In those rare cases of *atypical* hydrophobia (terminating in death) in which, for example, the increased reflex irritability, the fear of water, etc., are absent, the diagnosis of hydrophobia can only be made by a certain determination of the ætiology.

ANTHRAX

Similar to hydrophobia, anthrax is also an infectious disease which is transmitted from animal to man ("Zoonose"). The specific virus of anthrax has been known for a long time (since 1849), discovered by Pollender and Brauell, being a variety of bacillus the nature of which for the last thirty years has been the subject of exact studies by the most various investigators. The determination and the culture of the bacillus of anthrax and the observation of its properties of life have been the corner-stone of the modern developments of the doctrine regarding the infectious diseases! The proof of the presence of the anthrax bacillus has gained an eminently diagnostic significance and, as we shall see later, has been the means of the discovery that certain mysterious affections which until then had been incorrectly explained, are in connection with anthrax and are caused by the toxine of the latter.

Anthrax Bacilli.—The bacilli of anthrax are *large, clear, immotile rods, very susceptible to tinction, also according to Gram*; they divide at a higher temperature and grow out into mycelium which may accumulate into bundles. In the centre of the rods, or outgrowths, the clear-shining *spores* develop; this, however, only occurs at a higher temperature, i. e., not under 25° C. and best at 37° C., but only under a *plentiful supply of oxygen*. This last circumstance explains the fact that in the living animal body and in the corpse the bacilli of anthrax will not produce spores. Whereas the bacilli are very easily influenced by external circumstances, especially by drying, the spores, on the other hand, show themselves to be capable of long existence; the influence of sunlight, however, cannot be withstood by the spores. The micro-organism giving rise to anthrax accordingly thrives especially saprophytically, i. e., outside of the organism, but it is only facultatively saprophytic, in that, finding its way into the living organism, it becomes exceedingly dangerous as a parasite and gives rise to a high grade of pathogenic, infectious action. It may enter the organism by the most various routes and, according to its method of invasion, produce different clinical pictures. The poison finding its way into wounds of the skin shows the most frequent picture of the affection, *wound anthrax*; by the importation of anthrax spores with nourishment by way of the digestive tract *intestinal anthrax* is developed, "mycosis intestinalis"; finally, by inhalation of dust from wool, etc., containing the spores, *pulmonary anthrax* occurs, giving rise to pneumonic phenomena and a severe septic general infection. The way and method by which the toxine of anthrax, after having gained access to the susceptible organism, displays its action is, generally, that the bacilli, respectively the bacilli that germinated from the spores, multiply enormously and are distributed in the body by the blood and lymph streams, thus, as *truly septicæmic* noxa, overwhelm the entire organism.

The marked infectious action of the anthrax bacilli unquestionably depends upon a *chemical* poison produced by the metabolism of the bacilli, the nature of which, however, has as yet not been definitely determined.

Ætiology.—Certain ætiological conditions must constantly be taken into consideration in the diagnosis of anthrax. The transmission of the poison to man almost always results from the fact that the affected person has come in contact with living animals affected by anthrax (cattle, sheep, horses, goats, pigs, etc.), or with substances which originate from animals affected by anthrax. Therefore, certain individuals whose occupations cause them to come in contact with living or dead animals or their skins are especially predisposed, such as hostlers, herdsmen, farmers, dealers in skins, tanners, hat makers, furriers, etc. Insects that have been feeding on animals suffering from anthrax may also transmit the poison to man. The cases in which contagion takes place from person to person are very rare, but I have had an opportunity of seeing such an undoubted instance. The time from the invasion of the toxine of anthrax to the development of the morbid phenomena is usually very short; in general the period of incubation is but a few days (averaging about three days), rarely a week and more.

The diagnosis is certain only in those cases in which it is possible to demonstrate the bacilli in the affected organism. This proof, however, will not always succeed *intra vitam*, and especially not in cases of internal anthrax, as the circulating blood does not always contain anthrax bacilli. We are impelled to make a bacteriological investigation only by some distinct morbid phenomena, the knowledge of which is necessary, therefore, to set us upon the right track in making a diagnosis in the individual case. The symptoms are either uniformly developed in all severer cases of anthrax, or they may vary greatly in individual cases, according to the particular variety of anthrax which has appeared.

General symptoms of anthrax, i. e., of the infection, are: Fever (eventually also being absent), with increased pulse frequency, pains in the extremities, enlargement of the spleen and liver, cerebral confusion, collapse,

increasing cyanosis and dyspnœa, hæmorrhages from the most varied parts of the body, especially from the gums upon which suggilations form (in my cases these were particularly dark-blue, pock-like swellings), inflammation of internal organs (also endocarditis in which anthrax bacilli could be found in the valves). In the main, therefore, the signs of a severe infection of a septicæmic nature are pronounced.

Local Symptoms—Wound Anthrax.—If the bacillus finds it way into the organism through wounds of the skin, there will be, at the point of invasion, pain, reddening and infiltration of the skin; in the centre of the papulous, itching elevation of the skin a vesicle will form, which is filled with a serohæmorrhagic fluid, bursts and changes into a brownish-red gangrenous mass (*pustula maligna*). The skin of the surrounding areas swells more markedly, becomes bluish-red by infiltration, and hard, and eventually at the periphery shows a circle of small vesicles the size of a millet-seed. In contrast to the usual carbuncle, the infiltration in anthrax is not very painful but rapidly increases in circumference, showing œdema, inflammation of the lymph vessels and painful enlargement of the lymph glands; simultaneously the gangrenous scab in the centre also increases markedly in size.

Anthrax Œdema.—In some cases the development of the cutaneous œdema is more marked than that of the carbuncle. The *œdema* does not then limit itself to the originally infected area and its surroundings, but occurs all over the body, especially around the eyes, showing a pale-red, yellowish swelling. In the anthrax tumour, in the cloudy, gelatinous fluid of the œdema and in the contents of the vesicle, more or less bacilli may be found.

Intestinal Anthrax—Pulmonary Anthrax.—The picture of *intestinal anthrax* differs! Diarrhœa, eventually of a hæmorrhagic nature, vomiting, tympanites and painfulness upon pressure of the abdomen, cyanosis, dyspnœa, fever and collapse characterize this unusual fulminant form of anthrax. The infection in this case may have originated by means of food (primary intestinal anthrax) or from another area of invasion, anthrax bacilli, etc., may have found their way into the tissues of the intestines (secondary intestinal anthrax).

If the infection occurs in the tissue of the respiratory organs, anthrax runs its course by developing inflammation of the bronchi, pneumonic infiltration of the lungs, pleurisy and severe general phenomena (*pulmonary anthrax*).

Differential Diagnosis.—The diagnosis of anthrax and of its various forms is easy as soon as external phenomena of anthrax, which is usually the case, are well developed alone or with symptoms on the part of the internal organs. The appearance of a carbuncle or of inflammatory œdema with redness and gangrene of the skin and the formation of vesicles under all circumstances invites the examination of the fluid of the gangrenous parts or of the contents of the pustules for anthrax bacilli, especially if the history, the occupation of the patient, etc., make it at all possible or likely that an infection of anthrax may have occurred. Much more difficult is the diagnosis of pure *internal anthrax*. Bloody diarrhœa

is so frequently noticed from the most various causes that only exceptionally it occurs to the physician to search for anthrax bacilli, all the more as *intestinal anthrax* is a very rare affection. Nevertheless, the circumstance that in anthrax intestinalis dyspnœa and cyanosis appear rapidly, and eventually enlargement of the liver and spleen is demonstrable, may give us the correct clew; further, simultaneous epistaxis, hæmaturia, petechiæ and suggilations of the gums—phenomena which I have seen upon several occasions in internal anthrax and which closely resemble the picture of morbus maculosus Werlhofii—are certainly suspicious. More necessary does the examination of the blood become, which, it is true, does not always result positively even if anthrax be present, if the ætiology of the individual case directly shows infection with anthrax, if, for instance, it can be determined that the patient in question has taken food in a raw condition, or perhaps also water from a notorious anthrax district, shortly before he was taken sick, or if, during the time of summer, meat which was taken from an animal affected with anthrax and exposed to the air, has been eaten in an uncooked condition, and in this manner spores found their way into the stomach and intestine in which they could continue to develop. In general, the infection by anthrax evidently does not easily arise from the digestive tract, as it is very difficult to produce anthrax artificially in susceptible animals by feeding with spores or anthrax bacilli. As in the diagnosis of intestinal anthrax, the observation of the ætiology is also the most important element in *pulmonary anthrax* which is even more difficult to diagnosticate. If a pneumonia takes place in persons whose occupation consists in the sorting of rags, in the picking of sheeps' wool, in removing hairs from skins, or working in horse-hair, etc., and, therefore, involves the inhalation of animal dust, the inhalation of anthrax toxine should be thought of as a cause of the severe pneumonia arising with enlargement of the spleen, prostration, headache, vertigo, and chill; and in all cases the blood should be examined for the presence of anthrax bacilli. If we succeed in finding bacilli, the diagnosis of pulmonary anthrax ("rag-pickers' disease") becomes certain. In these cases the infection is due to the inhalation of *anthrax spores,* which cling to the pulmonary tissue and develop into bacilli, then find their way into the lymph channels and bronchial glands, thence to be distributed through the body by means of the blood stream and to produce a general septicæmic infection.

GLANDERS—MALLEUS HUMIDUS

Bacillus of Glanders.—The bacillus of *glanders* was discovered much later than that of anthrax, by Löffler and Schütz, in 1882, in the form of small, immovable rods, which are similar to the tubercle bacillus but smaller and shorter; they are stained with difficulty, not according to Gram. They are cultivated only in higher temperature upon agar, in blood serum and especially upon potatoes, and here in the shape of a yellowish-red sod. Inoculation upon animals (horses, donkeys, cats, guinea-pigs and field mice) at first causes only a local reaction, followed only after weeks by a general action. The former manifests itself in the shape of nodule-like new formations (similar to tubercular nodules) which show great tendency to break up and lead to cheesy degeneration, forming ulcers with an infiltrated base and hard edges; with this the lymph glands enlarge. The bacilli perish in the ulc

formation, they are found with difficulty, therefore, or not at all in the pus of the ulcer. The invasion of the glanders bacillus in the *human organism* occurs through small wounds of the skin or mucous membranes, also, as it appears, with an intact surface by taking up the toxine from animals affected by glanders into the conjunctivæ and mucous membranes of the nose and mouth in persons that frequently come in contact with horses: Coachmen, farmers, soldiers, horse butchers, etc. It appears that infection may also occur by way of the respiratory tract and that contagion may take place from person to person.

Diagnostic Symptoms.—After a period of incubation lasting from four to five days, *glanderous nodules* and *ulcers* appear at the area of invasion, which show no tendency to heal and present the appearance previously described. The surrounding parts become affected with inflammatory œdema and lymphangitis. As a rule, however not always, the nasal cavity is the place which primarily shows the eruption of glanders, in which case torpid ulcers arise upon the nasal sæptum and upon the mucous membranes of the muscles with a very fluid, purulent or bloody, occasionally also a foul-smelling, secretion. At the same time the external nose increases in size and becomes red, with band-like enlargement of the lymph channels and swelling of the neighbouring lymph glands. A further development of the process causes ulcerations in the mucous membrane of the mouth and of the pharynx, infiltrations in the larynx, in the bronchi and lungs, accompanied with hoarseness, cough, and expectoration, but rarely affecting the liver, spleen and other organs. As a rule, but later, in acute glanders at once, by means of the gradual increase of the process in the body, symptoms of general infection become manifest: Lassitude, headache, delirium, somnolence, pains in the muscles and joints, fever with chills, dyspepsia, swelling of the joints (due to a purulent effusion), pustules upon the skin, and abscesses, and the characteristic affection of the testicle which occurs in inoculated animals is also found in man.

Differential Diagnosis.—The course of the affection is sometimes acute, lasting from two to three weeks, at other times chronic, lasting from a quarter of a year to a year and longer; in the latter case the ulcers of glanders may be easily confused with tubercular and syphilitic ulcers. We are able to assume with a certain degree of probability the existence of glanders, as against tuberculosis, if a more marked reaction is present in the neighbourhood of the ulcers; if erythemata and numerous pustules or abscesses of the skin develop, and the proof of a primary tuberculosis in the lungs or in the urogenital system is negative in spite of repeated examinations of the urine and sputum for tubercle bacilli. The ulcers of glanders may also be differentiated with difficulty from syphilitic ulcers. Here principally the multiformity of the morbid phenomena and the simultaneous presence of bone affections show the syphilitic nature of the disease; the appearance of wide-spread band-like inflammations of the lymphatic vessels and the severe implication of the general organism, on the other hand, point to an affection by glanders. A *certain basis*, however, is only obtained for the diagnosis by taking into consideration the *history* and, finally, the *bacteriological examination* which, at least in the majority of cases, will clear the situation in that, in the morbidly changed tissues, glanders bacilli are found which may be cultivated and, when inoculated into animals, especially into guinea-pigs, give rise to the disease. At the same time cheesy nodes and ulcers form at the place of inoculation, also nodules in the spleen and lungs, and, what is especially conspicuous, soon after the inoculation inflammation and caseation of the testicles.

TRICHINOSIS

Trichinosis naturally no longer belongs in the group of infectious diseases, but is best treated of in connection with the previously described zoonosis.

Mode of Infection.—If a healthy person partakes of meat containing trichinæ, especially the meat of the domestic pig and of the wild pig in a raw or half-raw condition, the mode of infection is that the trichinæ contained in the meat (the intes-

tinal trichinæ vary in size according to sex, the females being from 3 to 4 mm., the males about 1.5 mm. in length) by a dissolution of the muscle fibres (and eventually of the capsules) become free in the stomach. The trichinæ which have become free develop in the upper parts of the intestine in the course of a few days into sexually mature animals, "intestinal trichinæ," mate, and in the course of a week after their introduction into the stomach bring forth living young. According to the newest investigations of Askanazy, this occurs to a great extent *inside* of the intestinal mucous membrane, in which the mature females have found safe lodgment, and only to a small extent in the lumen of the intestine. Those young trichinæ which have been born in the intestinal wall, especially in the lymph spaces, wander from there, at first by means of the lymph channels, to the muscles. Having reached this place, they increase in size (they are then about 1 mm. long) in the course of a further two weeks, roll themselves up and embed in a fine granular mass. Only after some time does a deposition of calcium salts occur in the capsule. The male trichinæ, and probably also the female trichinæ which have deposited their young in the lumen of the intestine, find exit with the dejecta.

The time from the entrance of the trichinæ into the intestine until the migration of the young trichinæ occurs into the muscles and their encapsulation in the latter, embraces the complex of phenomena which is called "trichinosis."

The knowledge of the existence of a *disease due to trichinæ* in man we owe to Zenker, who, in the beginning of the year 1860, noticed, at the autopsy of a girl, numerous freshly immigrated, youthful trichinæ in the muscles, and numerous sexually ripe trichinæ in the intestine, the affection having run its course with severe muscular symptoms and markedly disseminated œdema. Zenker proved at the same time that the importation of trichinæ in this case was due to pork containing trichinæ.

Morbid Picture.—The symptoms of the disease which point to the existence of trichinosis and which permit the diagnosis to be made, are the following: Primarily there are phenomena on the part of the digestive organs, which greatly vary in intensity, such as a sensation of fulness, nausea, loss of appetite, vomiting and diarrhœa; in other cases there is scarcely an indication of these symptoms, at least not in the first few days.

In the course of the first week, however, rarely later, marked attacks of fever occur (up to 104° and over), increased diarrhœa and collapse-like conditions, and, what is most important, great *lassitude and painfulness of the muscles.* The latter increase in size and, on account of the acute myositis producing a condition of irritation, various contractures and disturbances of the movability of the musculature arise. In the latter respect there must be particularly emphasized: The painful and difficult movement of the bulbi, mydriasis, weakness of the muscles of the larynx, dyspnœa as a result of the affection of the muscles of respiration (especially of the diaphragm); further, difficulties of chewing and swallowing, occasionally accompanied with trismus. The electric irritability of the muscles is occasionally diminished; the patella tendon reflex was temporarily noted to be absent in some cases. Of symptoms belonging especially to the nervous system, neuralgia has frequently been noted (neuralgia cœliaca), also delirium, hyperæsthesia and anæsthesia of the skin, and, occasionally, exanthems: Urticaria, herpes, petechiæ.

More important than the last-named phenomena, besides the muscular pains, is the almost never missing *cutaneous œdema.* The inflammatory œdema of the eyelids and face is supposed to be especially charact

it usually disappears rapidly, whereas the œdema occurring later on in the lower extremities is less transitory. According to my experience in polymyositis infectiosa, this œdema may partly be due to venous thromboses. Plentiful sweats have also been noted by all observers as conspicuous symptoms in the clinical picture of trichinosis; they occur early and last through the entire course of the affection; their genesis is not clear. The *blood* of patients affected by trichinæ shows, as it seems, a constant increase of the leucocytes, especially of the eosinophiles (Brown, and others), which may be of use in the diagnosis. The changes in the *urine* observed by some have attained no diagnostic importance; in severe cases albuminuria has been noted, which was independent of the fever. The almost constantly present *bronchitis* is more important in a diagnostic respect. The secretion is expectorated with difficulty, on account of the insufficiency of the expiratory muscles; its accumulation favours the development of *catarrhal pneumonia.*

Differential Diagnosis.—The diagnosis of trichinosis is only easy provided a case occurs at the time of an epidemic. But if we have to do with the diagnosis of an individual affection or with the first cases of an endemic, the diagnostic difficulties will be manifold. The onset of the disease may resemble an acute attack of gastroenteritis; in fact, in this stage the diagnosis cannot be made. Only from the end of the first week on is the morbid picture such that it is susceptible of diagnosis, as now *muscular pains* and *œdema* occur. The latter are certainly diagnostically important if the eyelids and the face are affected in the first place and if nephritis, circulatory disturbances leading to general stasis, or cachexia of a marked grade can be excluded. Usually the affections mentioned give rise to marked excretion of albumin in the urine, whereas trichinosis only exceptionally causes albuminuria. Frequent examination of the urine, therefore, is necessary under all circumstances.

The pains and swelling in the course of the muscles, besides the œdema, point directly to a local affection of the muscles, and to a *widely distributed* one at that, so that diseases implicating only some muscles, therefore the usual forms of myositis, muscular rheumatism, etc., need not be considered, and the differential diagnosis really only lies between *infectious polymyositis* and trichinosis. A differentiation, according to the experience of myself and others, is not easily possible by reason of the morbid picture, as all symptoms of one affection occur also in the other. Only the prominence of the gastro-intestinal symptoms and the initial œdema of the lids and of the face, provided they are present—and this is almost exclusively the case in trichinosis—are decidedly in favour of the existence of the last-named affection. But a certain diagnosis can only be made from the result *of the microscopic examination of the fæces for intestinal trichinæ, and of excised parts of muscles for muscle trichinæ.* Usually the former will be sufficient, especially if at the onset of the disease a purgative is given in order to dislodge the trichinæ more easily in this way. Positive results, however, can only be expected if the contents of the intestines are examined in the first five weeks. Nor must it be forgotten that plainly demonstrable intestinal trichinæ are rare in

the fæces, and, therefore, a negative finding by no means excludes the second method, the excision and microscopic examination of a part of the muscle. With a limited distribution of trichinæ in the muscular system, the result of this examination may also be negative, so that it is not allowable upon the basis of a negative finding at once to exclude the existence of trichinosis.

Naturally, the diagnosis is markedly assisted by the ætiology, i. e., by the fact of the patient telling us that he has partaken of raw pork or similar substances and, moreover, by the proof of muscle trichinæ in the meat of which the affected patient has eaten. As a rule, then, the case in question does not remain isolated, and a number of affections of a similar kind will occur in the same place or in the same district, and this in itself is in favour of the presence of trichinosis.

YELLOW FEVER

[Yellow fever is an acute specific, endemic and epidemic disease of brief duration, occurring in tropical and subtropical countries. It is characterized by abdominal pain, albuminuria, vomiting of black, altered blood, and by a yellow discoloration of the skin, from which the disease receives its name.

Ætiology.—The disease occurs particularly in the populous centres of travel along the seacoast, rarely reaching regions of decided elevation. Humidity and heat favour the prevalence of the disease. The affection occurs at all ages, children being particularly liable to it; the white race is more frequently attacked than the negro race. One attack, as a rule, confers immunity. The disease is rare above the 40th parallel. The *period of incubation* varies from a few days to a week or more.

Mode of Transmission of the Disease.—The disease is most probably transmitted by the mosquito; this view is favoured by the following facts: 1. The direct inoculation experiments have proven successful. 2. The prevalence of epidemics, as pointed out by Reed, shows that the disease occurs during the hot and moist season (the mosquito season), and disappears with the appearance of frost. The exciting cause is probably a protozoon conveyed by the mosquito; this has, however, not been definitely determined as yet.

Pathology.—Marked jaundice is present. The blood is altered. Hæmorrhages are noted in the skin and mucous membranes. Fatty degeneration of the liver and cloudy swelling of the kidneys are common findings. A black fluid is found in the stomach; the walls of this organ show areas of hæmorrhage, microscopic examination revealing some fatty degeneration. The heart is flabby and may also show fatty degeneration.

The Clinical Picture.—The onset is sudden and without prodromes, as a rule, the fever rising rapidly. The tongue is coated and moist, the bowels are constipated. The temperature is not typical, varying between 100° and 106° F.; as a rule, however, yellow fever is not a disease of high temperature. Remissions occur in the course of the fever and to
morning of the third day, and in mild cases there is a marked

temperature. In severe cases the temperature is more or less subconti ous, even becoming higher than the initial fever. The pulse is betw 100 and 110, but as the disease advances, even if the temperature tinues high, the pulse rate falls so that in the defervescence it may be tween 40 and 50 or even lower than this. Albuminuria is quite a cons phenomenon of the affection, there being some correspondence between amount of albumin in the urine and the severity of the affection, i. e mild cases the albuminuria is slight, in severe cases gradually acute phritis appears, in which uræmic convulsions may take place. The gas intestinal phenomena are prominent. There is marked irritability of stomach from the onset, nausea and vomiting being quite common. black vomit (altered blood) usually occurs in the final stage. Hæ rhages in other parts of the body also take place, thus petechiæ in skin; there may be bleeding from the gums and other mucous membra Constipation is the rule, except late in the affection, when diarrhœa occur. As a rule, the mind is clear, but active delirium may be pre from the beginning of the affection.

For convenience of description the disease may be divided into t stages, the period of onset, the calm, and the collapse. The first st the period of onset, lasts about three days; calm, usually twenty-four h or less, the mild cases never going beyond this stage. The duration of period of collapse varies, and in this stage the peculiar yellow discol tion of the skin appears, from which the disease receives its name, as also the black vomit. All the initial symptoms return with greater seve in the stage of collapse. The mind, which up to this time has remai clear, becomes clouded, and the patient may die in coma.

Types of the Disease.—Mild, severe or grave (hæmorrhagic) c occur during an epidemic. The mortality in this affection is high, in s epidemics being about 50 per cent.

Diagnosis.—The diagnosis depends upon the presence of an epide the disproportion of pulse to temperature, the early albuminuria, the low discoloration of the skin, and the black vomit; the agglutination may be useful.

Differential Diagnosis.—The affection is readily differentiated f *malaria* by the presence of the plasmodium in the blood in the latter af tion. The table on p. 1003 will serve to present the differential poi between yellow fever, influenza and dengue.]

DENGUE

[Dengue is an acute febrile infectious disease occurring in epide in tropical and subtropical countries. It is characterized by a marl febrile movement with periods of remission, a polymorphic exanth with severe pains in the joints and muscles. The affection is also known break-bone fever, dandy fever, broken-wing fever, etc.

Ætiology.—The exciting cause is still unknown. It attacks all ra both sexes, and occurs at all ages. One attack does not confer immuni The period of incubation is from two to five days.

	Dengue.	Influenza.	Yellow Fever.
Where occurring...	In tropic and subtropic countries.	In all countries.	In tropic and subtropic countries.
Numbers affected...	Large majority of population.	Large majority of population.	Cases limited.
Nature	Contagious.	Contagious.	Contagious.
Duration of epidemic	Two to five months.	Six to eight weeks.	Indefinite.
Affects whom	All races.	All races.	Foreigners particularly.
Period of incubation	Two to five days.	From a few hours to a few days.	One to seven days.
Characteristics	Severe pains in joints and muscles.	General muscular pains.	General muscular pains.
Ambulatory cases...	Frequent.	Frequent.	Rare.
Catarrhal symptoms.	Extremely rare.	Characteristic.	No catarrhal symptoms.
Pneumonia or pleurisy.	Rare.	Frequent.	Rare.
Gastric symptoms ..	Prominent.	Not so prominent.	Nausea and vomiting characteristic.
Diarrhœa	Rare.	Frequent.	Constipation.
Spleen.....	Not enlarged.	Enlarged.	Slightly enlarged.
Pulse and temperature.	Rapid and high fever.	Rapid and high fever.	Pulse slow and temperature not so high.
Eruptions.....	Frequent.	Rare.	Jaundice.
Hæmorrhage.....	Rare.	Rare.	Common.
Albuminuria.....	None.	None.	Constant.
Mortality	Low.	Somewhat higher.	Very high.
Sequelæ.....	Rare.	Common, frequent.	None.

(From Salinger and Kalteyer.)

Symptoms.—The disease begins suddenly with a severe chill, rapidly rising fever, headache, rapid pulse, and a polymorphic eruption which fades very rapidly. Severe pains in the joints occur, particularly in the knee joint. There is marked tenderness of the muscles, especially of the muscles of the back. The tenderness in the joints and in the muscles gives rise to a stiffness and alteration in the gait, this giving the disease its synonyms, break-bone fever and dandy fever. Gastro-intestinal symptoms are prominent. The tongue is thickly coated, there is anorexia and marked thirst. The face is flushed, the conjunctivæ are injected, the eyelids swollen. The prostration is extreme. After a duration of about three days ("three-day fever") the temperature falls; now another eruption (erythema, urticaria, herpes, etc.) becomes prominent, which disappears by desquamation. Convalescence is protracted. There may now be another rise in temperature which, however, terminates rapidly, lasting but a few days. The mortality is less than 1 per cent.

Diagnosis.—The diagnosis depends upon the presence of an epidemic, the abruptness of the onset, the appearance of an eruption at the beginning and the end of the affection, the severe pain in the joints, without redness and with very little swelling, the remissions during the febrile period and the pronounced prostration.

Differential Diagnosis.—*Acute rheumatic fever* is readily separated from dengue by the acid sweats, the cardiac affection, the less acute set, the symmetrical involvement of the joints, with redness and

Influenza and *yellow fever* must also be differentiated from dengue (see table under Yellow Fever).]

MALTA FEVER, MEDITERRANEAN FEVER, ROCK FEVER, NEAPOLITAN FEVER, UNDULANT FEVER

[Malta fever is endemic upon the coast of the Mediterranean, being especially frequent in Malta and Gibraltar. The exciting cause of the affection is a well-characterized micro-organism, the micrococcus melitensis, inoculation experiments having been successful. The affection occurs particularly in men and especially in youthful individuals. The *period of incubation* is from a few days to three or four weeks.

Pathology.—The most constant pathological findings are an enlarged spleen and the hyperæmia of the intestinal mucous membrane. In some cases typical lesions of enteric fever have been noted (Perry).

Symptoms.—After marked prodromes, such as chilliness, lassitude, general malaise, there is a rise in temperature to 104° F. or over, this rise being gradual. Simultaneously there is enlargement of the spleen and marked sweating, accompanied with rheumatic and neuralgic pains. The fever is frequently of a remittent type. This stage may last from one to three weeks, a period of apyrexia occurring, lasting a few days, being followed by a relapse. The duration of the relapse may be several weeks, and after another remission, which lasts a little longer than the first, a second relapse occurs with all the symptoms of the initial attack. The mortality is extremely low, about 2 per cent.

Diagnosis.—The only difficulty arises in cases occurring outside of the area of geographical distribution of Malta fever, but here the serum diagnosis is an important aid. The blood serum of patients affected by Malta fever shows strong agglutinating properties with pure cultures of the micrococcus melitensis, even upon marked dilution.]

PLAGUE—BUBONIC PLAGUE—PEST

[Plague is an acute infectious, contagious disease, characterized by an inflammation of the lymphatics, with a marked tendency to suppuration, with severe constitutional symptoms, and is due to the bacillus pestis.

Ætiology.—The exciting cause of pest is a specific micro-organism, the bacillus pestis, discovered by Yersin and Kitasato. Regarding the method of infection there is still some doubt. It is certain that rats are affected by pest and even frequently before the disease appears among human beings, so that it is quite probable that these animals may be regarded as carriers of the virus. It is undoubted that the contagious principle of pest may be carried by fomites; however, the disease is not contagious in the sense of the acute exanthemata. The prevalence of the affection is favoured by unsanitary conditions, poor food, improper housing, close crowding, such as occurs among the Chinese population in different parts of the world. The pest infection probably is brought about by means of the skin, the mucous membranes of the mouth, nose, pharynx, etc.

Pathology.—The morbid anatomy of plague shows a severe hæmor-

rhagic septicæmia, especially a pyæmia, with a multiple formation of buboes. The toxines eliminated by the bacilli give rise to severe hæmorrhagic, necrotic inflammatory processes, and as the toxines circulate in the blood the severe general symptoms are produced which characterize the affection.

Clinical Picture.—The clinical picture must be looked upon either as a local or as a general infection, according to the method of entrance of the active principle. We must differentiate between bubonic plague and pulmonary plague. The clinical picture of *bubonic plague* is especially well characterized. After a brief period of incubation, which varies from two to five days, the affection begins suddenly, without distinct prodromal phenomena, with a chill, high fever following immediately. The constitutional disturbances are great from the onset. The prostration is especially marked. There is headache, marked lassitude, repeated severe attacks of vomiting, stupor and delirium being by no means rare. After the existence of this affection for several days (usually up to five days), the external lymphatics are noted to be enlarged, especially in the region of the groins, more rarely in the axilla or in the neck; this marked enlargement of the lymphatics in the further course of the affection shows signs of pus formation and ulceration. The bacilli enter the blood from the buboes; severe necrotic inflammatory processes may also appear in the skin in the form of bubonic carbuncles. Secondary buboes may also be noted in the tonsils and in the lymph follicles of the gastro-intestinal mucous membrane. The hæmorrhagic diathesis occurs in the affection, which has given to the disease its ancient name "The Black Death." Hæmorrhages may occur from any part of the body. In some cases the bubonic infection presents a typical pyæmic character, metastatic foci being noted in the lungs, liver, kidneys, spleen and muscles. Enlargement of the spleen is almost constant in well-developed cases.

The *pulmonary form* may appear as a primary bubonic pneumonia or as a metastatic pneumonia, clinically resembling infectious pneumonia. The bacillus pestis is found in the sputum, particularly in cases in which the lung is especially affected.

The *course* of the affection varies from mild cases to fulminant ones. The mortality of this affection is greater than that of any other acute infectious disease.

Diagnosis.—The diagnosis is not difficult in the face of an epidemic. The bacteriological examination of the blood, of the pus from the buboes, of the sputum and the urine, however, is decisive. If the microscopic examination does not give reliable results, culture experiments should be made; guinea-pigs are the most suitable animals for this purpose.]

LEPROSY

[This is a chronic infectious disease in which pigmentary changes arise in the skin, and neoplasms form in the cutaneous and mucous surfaces, also in the nerves, giving rise to ulceration and deformity with a
of sensation.

There can be no doubt of the antiquity of the disease, apart even fr the undoubted mention and description from biblical records.

Ætiology.—The disease shows a wide distribution, from the arctic cle to the tropics. There is still doubt regarding the hereditary transı sion of the affection. Unsanitary surroundings, improper food, filth, no doubt play important predisposing parts. The male sex appears tc especially susceptible. No race is exempt.

The exciting cause of leprosy is the bacillus discovered by Hansen 1871. It is found in the tissues, and in many respects resembles the tul cle bacillus. However, it may readily be differentiated.

The *period of incubation* has not been definitely determined; it is tain that it may extend over many years.

Pathology.—Characteristic granulations, forming neoplasms wh consist chiefly of round cells in which the bacilli are found, occur. Th changes take place in the skin, mucous membranes, nerves, lymph glands and in the various organs of the body. Clinically, three forms the disease are recognised: (1) The *nodular* or *tubercular* form in wh the skin is chiefly implicated; (2) the *smooth,* in which the nerves b the brunt of the affection; (3) the *mixed form,* in which lesions occur both the nerves and the skin.

Clinical Picture—The Nodular or Tubercular Variety.—The prodron may be slight, consisting in irregular, afebrile temperatures, with lassitu drowsiness, gastro-intestinal symptoms, headache, epistaxis and often p fuse perspiration, alternating with rigors. These symptoms may even entirely absent.

The first signs of the disease consist in the eruption, which appears an irregular, shiny, reddish or copper-tinted erythematous patch, rai above the surrounding skin, accompanied with more or less hyperæsthe with infiltrations of the derma. The parts affected first are usually lobes of the ear, the alæ of the nose, the forehead, the eyebrows and lips; briefly, the face is *always* primarily implicated, later the erupt spreads to the extremities and to the trunk. This primary eruption of disappears, but reappears in a nodular form, which gradually enlarges, taining the size of a pigeon's egg. Small blood-vessels are sometimes s over the nodules. The appearance of the eruption is often characteri by a rising temperature, up to 103° F. The lymph glands are large painful, and, when the hands and feet are affected by the new grow desquamation of the nails occurs; after a varying duration of this proc ulceration sets in. Bleeding may become manifest in the ulcerated nodu especially in those located upon the hands and feet. This condition last variable period, death usually occurring from asthenia or some interc rent complication, such as tuberculosis or Bright's disease.

The Smooth Variety (Asthenic or Atrophic Form).—The prodror consist in trophic changes and sensory alterations. At first these are defined, coming on but slowly. Pain along the nerve trunks is comm but, if absent, numbness and tingling occur in the extremities. The sphi ters are often affected. Occasionally the eruption is the first symptom may be erythematous or pigmented; it first appears upon the shoulde

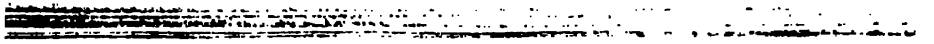
back, loins, knees, elbows, and only exceptionally upon the face. Sometimes the eruption follows special nerve tracts. Arthropathies occur; the bones ulcerate, due to an absence or deficiency of nerve supply. The condition resembles a severe neuritis.

Mixed Form.—The symptoms in this form represent a combination of the first two described.

Diagnosis.—The fully developed disease presents no difficulty in diagnosis. The finding of the bacillus is positive evidence. The prognosis is always grave, the disease being incurable.]

Following the infectious diseases, it would remain for us to discuss the diagnosis of *contagious sexual diseases* and that of *affections of the skin.*

For various reasons, however, I must forego this intention. As necessary as it is not to omit the discussion of these affections which form an integral part of internal medicine, in a text-book of the special pathology and therapy of internal diseases, an enlargement upon the details of the diagnosis of these maladies would not be in keeping with the plan adhered to in this book. The diagnostic deliberation, the condensation of symptom-complexes, the exact consideration which of the prevailing phenomena should be excluded from the diagnostic consummation of the individual morbid picture, etc.—to teach the solution of these problems in the given case is the object of this book—are of a subordinate importance in these affections. Bedside experience, the knowledge of the external form in which an individual disease manifests itself, but, above all, accurate observation and a clinically well-trained eye, are decisive. And this particularly involves the charm of the special study of these diseases in the clinic; a simple description of the manifest symptoms is certainly not sufficient to teach their diagnosis. As far as they concern the diagnosis of diseases of the various internal organs, they have been duly considered in every instance, and regarding the manifold manifestations of visceral syphilis, of the symptomatic exanthems, etc., I must refer to what has been exhaustively discussed in the various chapters.



INDEX

ABDOMEN, scaphoid retraction of, in acute spinal meningitis, 530.
sensitiveness to pressure, in dysentery, 929; in hysteria, 728.
Abdominal muscles, paralysis of, diagnosis of bilateral, 492; of unilateral, 492.
reflex of, in apoplectic attacks, 665.
spastic contractions of, in meningitis, 704.
Abdominal organs, diseases of, 169; diagnostic preliminary remarks on, 169; displacement by mediastinal tumours of, 150; method of investigation, vii; miliary tuberculosis of, 957.
Abdominal tumour, in intestinal stenosis, 326; differentiation from intestinal cancer, 314, 327; from splenic tumour, 228.
Abdominal walls, abscess of, and abscess of the liver, differentiation of the same, 191; carcinoma of the stomach, 286; tumours of, and carcinoma of the liver, differentiation, 204.
Abducens paralysis, affections of the eye in, 466; in affections of the pons, 607, 608, 616; in syringomyelia, 572; in tabes dorsalis, 537, 538.
seat of the cause of, 465.
Abductor paralysis in the larynx, 83.
Abortive forms of pneumonia, 125.
of typhoid symptoms and course, 919.
of variola, diagnosis of, 885, 888.
Abscess formations in mumps, 938.
at the laryngeal cartilages, 72.
hypophrenic, differentiation from abscess of the liver, 191; from pleuritic exudate, 160.
in pylephlebitis, 220.
in typhoid fever, 916.
metastatic, in the lungs, 141.
paranephritic, 367, 370.
peripleuritic and pleurisy, differentiation of the same, 160.
perityphlitic, 313.
Abstinence, sexual, favouring obesity, 843.
Abulia in higher grades of hysteria, 730.
Abuse of alcohol in cirrhosis of the liver, 183.
Accessorius, paralysis of, diagnosis, 479; of the external branch, 480; of the internal branch, 481.
spasm of the, 497.
spasm of the, clonic, 498.
spasm of the, tonic, 498.
Accommodation, disturbances of, in diabetes mellitus, 832; in diphtheria, 944; in hysteria, 729; in mumps, 940; in paralysis of the oculomotor nerve, 465.
Achillo tendon reflex, utilization of, in the diagnosis of myelitis, 575.
Achylia gastrica, 271.
nervous, 272, 304.
Acid formation, excessive, in nervous dyspepsia, 295.
in gastric ulcer, 276.
intoxication in cholera typhoid, 932.
of diabetics, 832.
Acoria, anæsthesia of the sensory nerves of the stomach in, 299.
Acusticus nerve, central termination of, 651.
degenerative changes of, in tabes, 545.
destruction of, by abscess formation in the temporal lobe, 693.
relation of the, to the cerebellum, 621.
Acromegaly, 757.
differential diagnosis from arthritis deformans, elephantiasis, myxœdema, syringomyelia, 758.
neurasthenico-hysteric disturbances in, 757.
pathogenesis of, 757.
pathological picture, 757.
variety of, 757.
Acroparæsthesia, diagnostic signs of, 756.
Actinomycosis of the lungs, 146.
Addison's disease, 405.
diagnosis of, 407.
pathogenesis of, 406.
symptoms of, 405.
Adductor paralysis in the larynx, 85.
Adductor paralysis in the thigh, 493.
Adenoma of the kidneys, 385.
of the liver, symptoms, 200.
Adhesions causing changes of shape and position of stomach, 291.
Adiæmorrhysis cerebri, 699, 700.
cause of apoplectic insult, 665.
Adiposis dolorosa, 844.
Adiposity, general, 839.
Adrenal bodies, disease of, 405.
Ægophony in mediastinal tumours, 148.
pleuritis exudativa, 159.
pneumonia, 123.
Affective movements of the face after an apoplectic insult, 667.
in paralysis of the central facial nerve, 474.
Ageusia, 438.
diagnosis of, in glossopharyngeal affection, 438; in paralysis of the chorda, 438; in paralysis of the facial nerve, 438.
trigeminus anæsthesia in, 438.
Agglutinines, bactericidal properties of, 861.
Akinesia algera upon hysterical basis, 728.
Albumin in the urine, in diseases of the urinary organs, 346.
in amyloid kidney, 365.
in contracted kidney, 362.
in engorged kidney, 348, 349.
in nephritis, acute, 352.
in nephritis, chronic, 357.
in nephritis, suppurative, 367.
in nephrophthisis, 374.
in peritonitis, 410.
in renal infarct, 374.
Albuminuria, 346.
cyclic, 346.
febrile, 347.
in amyloid liver, 195.
in amyloid spleen, 232.
in aortic insufficiency, 25.
in engorged kidney, 348, 349.
in fatty heart, 51.
in jaundice, 208.
in liver atrophy, 183.
in relation to acute nephritis, 352.
in relation to chronic nephritis, 357.
physiological, 346.
Albuminuria as a secondary finding, 347; after epileptic attacks, 740; in anæmia, 786; in cholera asiatica, 934; in diabetes mellitus, 828; in diphtheria, 944; in erysipelas, 893; in influenza, 986; in leucæmia, 795; in meningitis, 704; in meningitis, epidemic cerebrospinal, 951; in parotitis, epidemic, 938; in rabies, 994; in relapsing fever, 904; in scarlatina, 874; in tetanus, 991; in

typhoid fever, 914, 918; in typhus fever, 897; in variola, 886; transitory in trichinosis, 1,000.
Albumosuria, febrile, origin and occurrence of, 347.
in severe anæmia, 786.
Alcohol, abuse of, in cirrhosis of the liver, 183.
Alcohol neuritis, differentiation from tabes dorsalis, 548, 549.
symptoms of, 507, 549.
Alcoholism cause of obesity, 840.
in hæmatoma of the dura mater, 718.
in multiple neuritis, 505, 549.
in ophthalmoplegia, 616.
Alexia, relations between aphasia and, 656.
Alexines of the body, bactericidal property of, 860, 964.
Alveolar infiltration, inflammatory, of the lungs, 121.
Ammonia in the urine of hepatic diseases, 172.
Ammoniæmia in suppurative nephritis, 371.
Amnesia of hysterics, 731.
Amœbæ, ætiological significance of, in dysentery, 928.
Amyloid affection of the gastric mucous membrane; differentiation from atrophy of the mucous membrane and gastric carcinoma, 273.
affection of the organs of the abdomen, in bronchiectasis, 103; in pulmonary tuberculosis, 138; in urogenital tuberculosis, 370.
Amyloid kidney, diagnosis of, 364.
kidney, differential, 367.
Amyloid liver, 195.
carcinoma and, 199.
combined with amyloid kidney, 195.
combined with amyloid spleen, 195, 196.
deviations from the usual conditions in, 195; differentiation of fatty liver and, 194.
hypertrophic cirrhosis and, 186.
principal symptoms of, 195.
the result of syphilis in, 188.
with fatty kidney, 366.
Amyloid spleen, diagnostic signs of, 231.
in fatty kidney, 366.
Anæmia, 776.
ætiological factors in, 789.
aplastic form of, 780.
blood condition in, 777.
blood formation after blood losses in, 778.
chronic, 787.
coagulability of the blood in the various forms of, 781.
condition of the blood plates in, 781.
condition of the erythrocytes in, 778, 779, 780.
condition of the leucocytes in, 780, 781.
condition of the specific gravity of the blood in, 781.
diagnosis of, 787.
diagnosis of the various forms of, 788.
dyspnœa in, 781.
functional disturbances in, 781.
functional disturbances of the circulatory apparatus in, 783.
functional disturbances of the digestive apparatus in, 785.
functional disturbances of the musculature in, 782.
functional disturbances of the nervous system in, 782.
functional disturbances of the respiratory apparatus, 781.
functional disturbances of the stomach in, 272.
functional disturbances of the urinary apparatus in, 786.
general appearance in, 776.
hæmorrhages in, 786.
in relation to gastric ulcer, 276.
metabolism in, 777.
mild form in, 778.
occurrence of, 789.
of the brain, 699.
Anæmia of the spinal cord, 594.
pernicious, 776, 778, 779, 780, 786, 787, 788, 789.
pernicious, nature of, 780.
primary, 787.
pseudoleucæmia infantum, 807.
secondary, 778, 781, 787, 788.
splenica, symptoms of, 802.
stomach contents in, 272.
subsequent symptoms of, 786, 809.
Anæsthesia, 436.
cerebral (central), 433, 442.
contralateral, in lesion of the optic thalami, 644.
cutaneous, 442.
cutaneous, in conus affections, 585.
diagnosis of the various grades of, 436.
differentiation between central and peripheral, 433, 442.
dolorosa, 445.
in affections of the posterior horns and posterior nerve roots of the spinal marrow, 526.
in hysteria, 728.
in myelitis, 574.
in neuralgia, 446.
in neuritis, 503, 504, 506.
in pachymeningitis (cervicalis) hypertrophica, 534.
in spinal meningitis, 530, 533.
in traumatic neuroses, 734.
in trichinosis, 999.
in the trigeminus region, unilateral lesion of the spinal cord, 441, 443.
in tumours of the spinal cord, 584.
Anæsthesia, muscular, 445.
of the facial skin in neuralgia of the trigeminus, 471; in paralysis of the facial nerve, 472.
of the forearm below the elbow, in Klumpke's paralysis, 492.
of the hands, in paralysis of the median nerve, 490; in paralysis of the radial nerve, 498; in paralysis of the ulnar nerve, 489.
of the laryngeal mucous membrane, 81.
of the legs, in cauda equina tumours, 584; in paralysis of the crural nerve, 493; in paralysis of the sciatic nerve, 494, 495.
of the sensory nerves of smell, 437; nerves of taste, 438; nerves of the urinary bladder, 403.
of the sensory nerves of the stomach, 290; of the soles of the foot, 442.
origin of, 433.
sensory, in hysteria, 729.
spinal, peripheral, 433, 434, 442, 446.
symptoms according to localization, 442.
Anal crises in tabes dorsalis, 538.
neuralgias, diagnosis of, 462.
region in, disturbances of innervation of the rectal, 341.
Analgesia, 436.
in posterior poliomyelitis, 571.
in syringomyelia, 571.
in tabes dorsalis, 545.
in traumatic neurosis, 734.
of hysterics, 728.
Anamnesis, vi.
Anarthria, 652.
in affection of the pons, 482.
in paralysis of the hypoglossus nerve due to lesion of the nuclei of the hypoglossus nerve, 483.
in progressive bulbar paralysis, 612.
in progressive muscular atrophy, 566.
Anasarca of the lower extremities in cirrhosis of the liver, 182.
in fatty kidney, 366.
Aneurysm, aortic, 64; of the abdominal aorta, 67; of the ascending aorta, 67; of the descending aorta, 67; of the aortic arch, 64; and gastric carcinoma, differentiation, 285; and pleurisy, differentiation, 155, 160.
auscultation in, 64.

Aneurysm, cardiac murmurs in, 65.
causing œsophageal stenosis, 67, 249; paralysis of the recurrent laryngeal nerve, 89; tracheal stenosis, 67.
icterus in, 212.
of the hepatic artery, 220.
origin of, 64.
palpation of, 65.
perforation into the superior vena cava, 66.
pulsation of an, 64.
pulse changes in, 66.
venous compression of, 66.
Angina, 239.
acute phlegmonous, 240; superficial, 239.
chronic phlegmonous, 241; superficial, 240.
differentiation from neurasthenia, 734.
diphtheritic, simple, without membrane, 242.
glandular, 239.
in acute articular rheumatism, 978; in influenza, 986; in mumps, 938; in purpura variolosa, 886; in scarlatina, 873, 875.
infectious, 242.
lacunar, 239, 240.
Ludovici, in diphtheria of the larynx and pharynx, 943; in scarlatina, 873, 875.
Angina pectoris, 37; differentiation from carcinoma of the palate and, 245; in atheroma of the coronary artery, 58, 62, 63, 64; in fatty heart, 52; in general adiposity, 840; upon a syphilitic basis, 244.
Angioneuroses, pathological pictures of, and their diagnosis, 754.
Ankylostomices in the duodenum and small intestine, causing anæmia, 345.
Anorexia, nervous, diagnosis of, 299.
Anosmia, 437.
central, 437.
examination for, 437.
in facialis paralysis, 472.
nervous, 437.
Anterior columns of the spinal cord, affection of, and symptoms, 525, 555, 572; fibre systems in, 515, 516.
Anterior horn ganglion cells, anatomical location in the cord, 515; connection of, with the motor root fibres, 519; lesion of, and symptoms, 519, 554, 555, 558, 563.
Anterolateral column ground bundles, affection of, and symptoms, 526; course of, in the medulla, 596.
Anterolateral column tracts, affection of, and symptoms, 525; secondary degeneration of, 519, 551; structural conditions of, in the cord, 523, 525.
Anteversion of the liver, 206.
Anthrax, ætiological factors in, 995.
bacilli, action of, in the body, 995; invasion of, 995; morphology, biology, of, 995.
diagnosis of, 995.
differentiation of from enteric fever, 925; erysipelas, of the various forms of, 995, 996.
infection with the virus of, 995, 996.
internal, 996.
of the intestine, 323; of the lungs, 996.
pneumonia, 925.
symptoms, traumatic, diagnosis of, 996; origin of, 996.
virus of, 995.
wounds of the skin in, 996.
Antitoxines, origin and action of, in the specifically infected organism, 858.
Anuria due to hydronephrosis, 381; to uræmic intoxication, 359.
in hysteria, 730.
Aortic aneurysm, 64.
diagnostic symptoms of, 64, 65, 66.
mediastinal tumours, and differentiation of, 152.
Aortic arch, hypertrophy of, in persistence of aortic isthmus, 38.
Aortic insufficiency, 19.
albuminuria in, 25.
angina pectoris in, 58.
auscultation of the arteries in, 23.
Aortic insufficiency, back-stroke elevation in, 22.
combined with endocarditis of the mitral valve, 21, 24.
crural double sound and murmur in, 23.
differential diagnosis in, 24.
dilatation and hypertrophy of the left ventricle in, 20.
inspection and palpation of the cardiac region in, 19.
percussion of the heart of, 20.
peripheral arteries in, 23.
pulse conditions in, 21.
relative mitral insufficiency, 23, 24.
tricuspid insufficiency, 31.
Aortic sounds in aortic stenosis, 26; in mitral stenosis, 18.
Aortic stenosis, 26.
apex beat in, 26.
congenital, 28.
cyanosis in, 28.
diagnosis in, 28.
heart murmurs in, 26.
hypertrophy of the left ventricle in, 26.
in myocarditis, 51.
pulse conditions in, 27.
pure, 26.
Apex beat in aortic insufficiency, 19; cause of normal, 45; in aortic stenosis, 16; in fatty heart, 51; in mitral insufficiency, 12; in mitral stenosis, 16; in myocarditis, 49; in pericardiaca, 45; in pericarditis, 40; in pleurisy, 155; in pulmonary shrinkage, 128; in pulmonary insufficiency, 29.
Aphasia, 651.
ability to read in, 655, 656, 658, 659.
ability to write in, 656, 657, 658.
ætiological factors, 661.
after apoplectic attacks, 665, 667.
anamnetic, 657, 658.
character of, 654, 655.
characterization of, 651; the various forms, 654.
conduction, 655; centre of, 661.
diagnosis, 652, 654.
gustatory, 657.
in cerebral abscess, 693.
in cerebral embolism, 678.
in cerebral tumours, 684.
in diphtheria, 945.
in encephalitis infantum, 696.
in enteric fever, 918.
in focal affections of the central convolutions, 651; in centrum ovale, 661, 662; in frontal convolution, 651.
in meningeal hæmorrhages, 718.
in meningitis, 705.
in paralysis of the hypoglossus, 482.
mixed forms of, 657.
motor, 654, 655, 658, 659; cortical, 654, 658, 659; subcortical, 654, 658, 662; transcortical, 655, 658.
optic, 657, 693.
partial and total, 657, 659.
sensory, 654, 658, 659, 693; cortical, 652, 654, 659, 661; subcortical, 654, 658; transcortical, 654, 657, 658.
tactile, 657.
transitory, 663.
Aphonia, in affections of the medulla oblongata, 608.
in paralysis of the hypoglossus, 483.
Aphthæ of the mouth, 236.
Apoplectiform attacks, 673.
differentiation from cerebral hæmorrhages, 672; differentiation from paralytic attacks, 698.
in brain tumours, 682.
in meningeal hæmorrhages, 719.
in multiple sclerosis of the spinal cord, 591, 592.
in tabes dorsalis, 539, 546.
Apoplexia sanguinea, 664.
absence of apoplectic insult in, 675.
consequences of, direct, 668; indirect, 666; posthemiplegic, 672.

Apoplexia sanguinea, differentiation from symptomatic apoplexy and, 673, 674, 832.
genesis of the insult of, 665.
in septic affections of the brain, 970.
localization of, in the brain, 668, 669, 670, 671.
prodromes of, 665, 676.
symptoms of the apoplectic insult of, 664.
Apoplexy, pulmonary, 139.
Appendicitis, 311, 313.
perforative, 313.
Aqueduct of Sylvius, anatomical location of, and borders of, 528.
dropsy of, 625.
Arm, fore, atrophic paralysis of, 491; muscle atrophy of, 564.
Arm, muscles of the, convulsions of, 499, 500, 501.
paralyses of, upper arm, 484, 485, 486.
combined, 491; in cortical lesion of the central convolution, 649, 650; forearm, 486, 488, 489, 490.
Arnold's ganglion, 438.
Arsenic poisoning, fatty heart in, 52.
Arteries, atheroma of the, 62.
blood-pressure in the, in disturbances of the heart, 1.
changes of the, in aortic insufficiency, 21, 23.
compression of the, by mediastinal tumours, 149.
volume of the, in persistence of the isthmus aortæ, 38.
Arteriosclerosis, 62.
cardiac murmurs in, 62.
hypertrophy of the heart in, 62.
in diabetes mellitus, 830.
in paralysis agitans, 751.
of the arteries of the brain, and symptoms, 676.
pulse condition in, 62.
subsequent phenomena in, 64.
Arthritis, 844.
chronic, 849.
deformans, 849.
diagnosis of, 849; differential diagnosis from acromegaly, 849; differential diagnosis from articular gout, 848; localization of, 854.
Arthrogryphosis infantum, diagnosis of, 750.
Arthropathies, hypertrophying, 757.
differentiation of, from affections of the vertebral articulations, 547.
in syringomyelia, 572.
of tabetics, 545.
Articular gout, diagnosis of, 846, 848.
pains in, differentiations from neuralgias, 447; in anthrax infection, 995; hæmorrhagic diathesis, 807; influenza and, 985.
in polyarthritis acuta, 971.
multiple, in epidemic cerebro-spinal meningitis, 950, 951, 953.
Articulation centres, location of, 651.
Arytænoid cartilage, tubercular ulcers of the, 73.
transverse, paralysis of, 85, 87.
Ascites, 421.
abdominal walls in, 421.
ætiological importance of, in atelectasis of the lungs, 107.
chyliformis and chylosus, 424.
diagnosis of, with slight transudate, 422.
differential diagnosis from gastrectasis, 424; hydronephrosis, 383; ovarian cyst, 423; serous peritonitis, 416.
diseases causing, 2, 422, 423.
Ascites in amyloid liver, 195; in cancer of the kidney, 386; in cancer of the liver, 197; in cirrhosis of the liver, 182; in hyperæmia of the liver, 193; in pancreatic affections, 223; in syphilis of the liver, 188; physical examination of the abdomen in, 421; puncture fluid, examination of, 423; the result of stasis in the portal vein circulation, 219.
Aspiration icterus, origin of, 175.
Association centres of the cerebral cortex, function and number of, 639.
irritable weakness of, in hysterics, 723.
localization of, for the different qualities of sensation, 643, 651.
Association fibres of the cerebral cortex, 634, 635, 638.
Astasia abasia of hysterics, 724.
Asthma, bronchial, 103.
cardiac, differentiation of, 58, 105.
causes of, 105.
differentiation of attacks of, from tonic spasm of the diaphragm, 501.
Charcot crystals and Curschmann's spirals in, 105.
differential diagnosis, 104; between spasm of the diaphragm, 105; pulmonary emphysema, 104; spasm of the glottis, 105.
symptoms of, 103.
Asthma, cardiac, 58.
auscultatory signs in, 61; differential diagnosis, 61; differential diagnosis between paretic and spasmodic, 61; dyspnœa in, 60.
Asthma, cardiac, dyspeptic conditions in, 61.
in adipositas universalis, 840.
in aortic aneurysm, 65.
in atheroma of the coronary arteries, 63.
in gout, 847.
paretic, 59.
pathogenesis of, 58.
percussory symptoms of heart and lung in, 60.
predisposition to the different kinds of, 61.
pulse conditions in, 60.
uræmic conditions in, 61, 359.
Ataxia, acute, after diphtheria, 945.
in Brown-Séquard paralysis, 583.
cerebellar, 550.
conduction, peripheral motor and sensory, due to multiple neuritis, 507, 543.
hereditary (family, spinal), 549, 550; in multiple myelitis, 588, 589; in multiple sclerosis, 589, 590; in multiple neuritis, 507, 508, 510.
peripheral motor, 507; peripheral sensory, 507; in pontine hæmorrhages, 609.
sensory, in affections of the posterior horns and posterior roots of the spinal cord, 526; in sensory lesion of the optic thalami, 644; in posterior poliomyelitis, 571.
static, in lesion of the corpora quadrigemina, 633; in optic thalamus, 630; in tabes dorsalis, 537, 539, 543, 548; nature of, 542, 543.
Atheroma of the arteries, 62; of the cerebral arteries, 63; of the coronary arteries, 63; the cause of rupture of the heart in, 53.
Atheromatosis of the vessels in gout, 845; ætiological significance in cerebral hæmorrhage, 665.
in meningeal hæmorrhage, 718.
Athetosis, 746.
bilateral, 747.
characteristics of, 746.
differentiation of, from chorea, 746, 747; from tremor, 747.
idiopathic, 747.
in capsule affections of the brain, 646.
in lesion of the optic thalamus, 644.
posthemiplegic, 672, 747.
symptomatic, 747.
transition of, into chorea, 747.
Atony of the gut, 341.
of the stomach, 271, 282.
Atrophy of the liver, 176.
of the mucous membrane of the large intestine, 318; of the larynx, 70; of the pancreas, 226; of the small intestine, 319; of the stomach, 271.
Aura epileptica, duration and phenomena, 738.
Auriculo temporalis, neuralgia of, 452.
Auto-suggestion of neurasthenic conditions, 733.
Axillaris, paralysis of, 486.

B

Bacilli, ætiological significance of, in Weil's disease, 214.
in cholera nostras, 309.
in cystitis, 394.
in diphtheria, 242.
in peritonitis, 409.
in pneumonia, 121.
in tuberculosis of the lungs, 128, 130, 131.
in tuberculosis of the kidneys, 375.
Bacilli, pathogenic, as cause of anthrax, 995.
of cholera, 931, 935.
of diphtheria, 940.
of glanders, 997.
of influenza, 985.
of miliary tuberculosis, acute, 955.
of tetanus, 989.
of typhoid fever, 906, 907.
Bacillus coli communis, ætiological relation to peritonitis, 409, 411.
Bacteria, diagnostic significance of the demonstration of pathogenic, 857, 858.
general local effects of, in the organism, 856, 858; in cystitis, 394, 395.
in contents of the intestine, 308; of the stomach, 267.
in diphtheria, 242.
in endocarditis, 7.
in the urine in nephritis, 355.
metastasis forming, 858.
pathogenic, in the oral cavity, 238; in pleuritic exudate, 162; in pneumonic areas, 121.
protective measures of the organism against, 859, 860.
Bacterial immunity of the organism, 860.
Bacteriolysines, occurrence and action of, in immunization, 861.
Bandbox note of lung in bronchial asthma, 103; in pulmonary emphysema, 112.
Barlow's disease, character of, and relation to the hæmorrhagic diathesis, 809.
Basedow's disease (see Graves's Disease).
Basilar meningitis, diagnostically valuable symptoms of, 706.
Bedsores after apoplectic insult, 688; by compression, 580; in capsular affections, 648.
in myelitis, 575.
in syringomyelia, 572.
in tabes dorsalis, 539, 545.
in typhoid fever, 912.
in typhus fever, 899.
Bile capillaries, anatomical arrangement in the liver of, 170.
Bile-duct (excretory), stenosis, respectively occlusion of, 207.
location of, 209.
Bile-ducts, obstruction of, 210.
causes of, 210, 211, 212.
diagnosis of the seat of, 209.
symptoms of, 207.
Bile-pigment reaction, diagnostic significance of, in acute intestinal catarrh, 311.
Bile secretion and disturbances, 173.
Biliary fever, intermittent, 217.
Blackwater fever, 984.
Bladder, function of the, disturbances of, in compression myelitis, 580; in conus affections, 585; in disease of the nerve roots in the spinal cord, 526; in hysteria, 725; in lesion of the cerebral peduncles, 219; in meningitis, 703; in myelitis, 576; in neuritis, 508; in spinal meningitis, 533; in syringomyelia, 572; in tabes dorsalis, 538, 539.
Bladder, paralysis of, 401.
after diphtheria, 944.
discharge of urine in, 401, 402.
in pachymeningitis cervicalis hypertrophica, 534.
in tabes dorsalis, 538, 539.
spasm of the, 402, 403; due to combined spasm of detrusor and sphincter muscles, 403; due to hyperæsthesia of the mucous membrane, 403; due to dysuria and spastic ischuria in, 402; due to enuresis spastica, 403; greatest intensity of, 403.
Bladder stones, causing cystitis, 397; and renal stones, differentiation, 396, 397, 398; sounding of the same, 397.
Bladder, urinary, affections of the, 393.
anæsthesia of the, 404.
carcinoma of the, 399.
carcinomatous particles in the urine of the, 399.
catarrh of, the ætiology of the, 396; diagnosis of the, 393; the urine in, 393.
endoscopic examination of the, 399.
function of the, nervous disturbances of the, 400.
hæmaturia in, 399.
hæmorrhoids of the, diagnosis of the, 400.
hyperæsthesia of the, 403.
inflammation of the (see Cystitis).
neoplasms of the, 399.
Blepharospasm, 497.
relaxation of the, 497.
Blindness, psychical, in cortical lesion of the brain, 642, 649.
nature of, 649.
Blindness, total, in bilateral lesion of the occipital brain cortex, 649.
Blood-casts in the urine in acute nephritis, 352.
Blood-corpuscles, changes of, in anæmia, 778; in hæmoglobinæmia, 811, 812, 813; in leucæmia, 790, 791, 792.
formation and disintegration of the red, 773.
formation and disintegration of the white, 774, 776.
forms of the white, 774.
functions of the white, 775.
Blood crises (von Noorden), 779.
Blood, diseases of the, diagnosis of, 772.
impoverishment of the (see Anæmia and Chlorosis).
Blood in the sputum in bronchiectasis with cerebral abscess, 102.
in connective-tissue hyperplasia of the liver, 186.
in hæmorrhagic infarct, 140.
in hepatic atrophy, acute yellow, 178.
in pneumonia, 121, 125.
in pulmonary alveoli, 118.
in the stools in intestinal cancer, 326.
in ulcer, 320.
in intussusception, 336.
in the urine in chronic nephritis, 357; in the urine in contracted kidney, 362.
Blood, morphological constituents of, and their changes, 772, 776.
Blood plasma, 772.
Blood plates, morphological properties of, 776.
Blood pressure, in heart disease, 1, 41, 54, 55, 59.
in jaundice, 208.
in renal disease, 54, 55, 348.
the result of reflex irritation of the vasomotor centre, 55.
Blood reaction of typhoid patients, 861, 920, 921.
Body, attitude of, in paralysis agitans, 751.
equilibrium of, deficiency of, in conditions of ataxia, 543.
Bone inflammation, diphtheritic, 944; due to septic infection, 966; in enteric fever, 918.
Bone marrow, changes of, in leucæmia, 793, 797, 799; in leucocytosis, 801; in septicopyæmia, 966.
tumours, myelogenous, differentiation from osteomalacia, 851.
Borborygmi in acute intestinal catarrh, 308.
Brachium conjunctivum, origin and course in the cerebellum, 619.
Brach-Romberg's symptom, 539.
Bradycardia in coronary sclerosis, 63.
Brain, abscess of, 691.
acute, 694.
ætiological factors for the diagnosis of, 694.
apoplexy due to, 674.

Brain abscess, diagnosis of acute, 694; subacute and chronic, 694.
differentiation of, from brain tumours, 688; from meningeal hæmorrhage, 694; from purulent meningitis, 694.
encapsulation of, 691.
focal symptoms of, 692.
general symptoms of, 692.
idiopathic, 695.
in central lobe, 693; in diphtheria, 943; in typhoid fever, 916.
latency of, 692.
meningitis in progressing, 691, 710.
of the cerebellum, 693.
of the frontal lobes, 693.
of the occipital lobes, 693.
of the temporal lobes, 693.
Brain, anæmia of, diagnosis of, 699: ætiological, 700.
differentiation from hyperæmia of the brain, 700, 701.
symptoms of, 700.
Brain, anterior, development and structure of, 634, 635.
functions of the, 640.
surface of cortical areas, 637; fissures and convolutions of the, 637, 638.
topico-diagnostic view-points in affections of, 643, 662, 663.
Brain, arteries of the, aneurysms of, causing meningeal hæmorrhages by rupture, 718; tumour phenomena in, 691, 692.
ætiological diagnosis of, 679.
apoplectic stroke in, 677.
differentiation of, from cerebral hæmorrhage, 679.
embolism and thrombosis of, 676.
epileptic convulsions in embolic insult, 677.
stages of, 677, 678.
symptoms of, 678.
Brain, cortex of, association tracts of (fibræ propriæ), 634, 638.
fossæ and convolutions of, 634, 637.
histological structure, 637.
lesions of the, 648; diagnosis of, 648, 649; due to hæmorrhages, 670, 719; paralysis of the facialis due to, 475, 650; paralysis of the muscles of mastication, due to, 467.
motor sphere (centres) of, 640, 641.
points serving for speech, 659.
sensory sphere (centres) of, 641, 642.
symptoms of cortical foci in the central convolutions and paracentral lobes, 649; frontal convolutions, 651; occipital convolutions, 649; parietal convolutions, 649; temporal convolutions, 651.
Brain, diseases of, descending degeneration in the pyramidal lateral-column tracts, 552, 553.
diffuse, 699.
focal, remaining latent, 663.
functional, 722.
of the anterior brain, 634, 665.
of the cerebellum, 619.
of the middle brain, 626.
of the posterior brain, 597.
speech disturbances in, 651–663.
Brain, hyperæmia of, 699.
active and passive, due to stasis, 701, 702.
ætiological factors of, 700.
diagnosis and symptoms of, 699, 700.
Brain, nerves of the, anatomical changes of, in amyotrophic lateral sclerosis, 554, 555; in tabes dorsalis, 538, 539.
central tracts of the motor, 636.
compression of, 617.
neuritic affection of, 506.
paralysis of the motor, 465; in brain tumours, 684, 685; in cerebellar affections, 624; in meningitis, 705; in pons hæmorrhages (alternating), 610, 670.
sclerotic changes, 589.
spasm of the motor, 495.
Brain, nuclei of the nerves of the, anatomical location of, 597, 598, 599, 601, 629.
Brain, degeneration of, in progressive muscular atrophy, 563, 564, 566.
ophthalmoplegia of the, 616.
paralysis of, in progressive bulbar paralysis, 611, 613.
Brain, œdema of, ætiological factors and symptoms, 702.
Brain, softening of the, 676.
apoplectic insult in, 677.
chronic progressive, 680.
differentiation of, from cerebral hæmorrhages, 679; from cerebral tumour, 689.
focal symptoms of, 678.
origin of, 677.
seat and extension of the area of, 679.
syphilitic, 680.
Brain, syphilis of, differential diagnosis of multiple syphiloma from meningitis, 710.
Brain, tubercles of the, in childhood, and their diagnostic signs, 690.
Brain, tumours of the, basal, 685, 686, 690, 691.
carcinomatous, 691.
causing apoplexy, 674.
diagnosis of, 680; of the course of the, 000; of the seat of the, 689; of the variety of the, 689.
differentiation from chronic cerebral abscess, 688; from chronic hydrocephalus, 689; from chronic softening of the brain, 689; from epilepsy, 688; from meningitis, 688; from migraine, 687; from progressive paralysis, 698; from multiple sclerosis, 689; from pons-medulla foci, 609.
due to aneurysms, 691; to parasites, 690.
during childhood, 690.
gliomatous, 690.
gummatous, 690.
in the lobes of the brain, 686.
of the central cranial fossa, 685.
of the posterior cranial fossa, 685.
sarcomatous, 690, 691.
seat of, 684.
symptoms, general, 682, 683, 684; local (focal), 683, 684, 685.
Breathing, diminished, in atrophy of the lungs, 129; in bronchial asthma, 103; in bronchial stenosis, 96; in fibrinous bronchitis, 94; in pleurisy, 154; amphoric, in aortic aneurysm, 65; in cavities of the lungs, 136; bronchial, in mediastinal tumours, 146; in pleurisy, 157; in pneumonia, 123, 127; in pulmonary atelectasis, 106, 108; in pulmonary cavities, 108, 136; in pulmonary consolidation, 109; in pulmonary œdema, 117; metamorphosis of, 137, 138; stertorous, in uræmia, 359; vesicular, in bronchial catarrh, 90; in insufficiency of the pulmonary valves, 29.
Bright's disease (see Nephritis).
Bronchi, compression of, by mediastinal tumours, 97.
diseases of the, 90.
inflammatory thickening of the bronchial wall, as caused by bronchial stenosis, 98.
perforation of, 98.
Bronchial asthma (see Asthma).
Bronchial catarrh, 90.
acute articular rheumatism in, 974.
ætiological factors, 91, 95.
anthrax in, 996.
chronic, 91; differentiation from bronchiectasis, 102.
engorgement symptoms in, 91; hypertrophy of the heart in, 56; impaired expiration in, 90; influenza in, 985, 987; leucæmia in, 794; measles in, 863, 866; miliary tuberculosis in, 956; mumps in, 940; relapsing fever in, 904; rhachitis in, 853; scarlatina in, 875, 876; trichinosis in, 1000; typhoid fever in, 911, 912, 914; typhus in, 899.
infectious, causing broncho-pneumonia, 126.
râles and, 90.
respiratory murmurs in, 90.

Bronchial coagula in the sputum in croupous pneumonia, 121; in fibrinous bronchitis, 94.
Bronchial fremitus, 90.
Bronchiectasis, 99.
combined with pulmonary hæmorrhage, 103.
complicated by amyloid affection of the abdominal organs, 103; complicated by cerebral abscess, 102.
demonstration of cavities in the lungs in, 100.
differential diagnosis between bronchiectatic and phthisical cavities, 100; from bronchitis chronica, 102; from bronchitis putrida, 102; from pulmonary abscess, 101; from pulmonary gangrene, 102; sacculated pyopneumothorax, 101.
due to interstitial pneumonia, 129.
seat of, 100.
sputum ("mouthful" expectoration) in, 99; origin of, 100.
Bronchiostenosis, 96.
auscultation in, 96.
cause of, 97, 98.
cicatricial callous, due to syphilis, 98.
differential diagnosis between laryngeal stenosis, 96; tracheostenosis, 97.
due to fibrinous coagulation, 98; due to foreign bodies, 97.
in hysterics, 98.
location of, 96.
percussion in, 96.
Bronchitis, acute, 90.
bronchiectasis in, 102; perforated empyema, 91; pulmonary gangrene, 91.
capillary (suffocative), 92.
chronic, 91.
differential diagnosis between putrid and fibrinous, 94.
pseudofibrinous, 94.
putrid, 91.
uncomplicated, 90.
Bronchitis, capillary and acute, in miliary tuberculosis, 962.
Bronchophony in bronchiectasis, 100.
in broncho-pneumonia, 127, 129.
in congestion of the lungs, 109.
in exudative pleurisy, 157.
in pneumonia, 122, 123.
in pulmonary atelectasis, 106, 108.
Broncho-pneumonia, 126.
acute, 133.
caseous, 130, 134.
in whooping-cough, 948.
Bronchorrhœa puriformis serosa (pituitosa), 91.
Bronze colour of diabetics, 829.
Brown-Séquard spinal paralysis, 582.
disturbances of discharge of urine and fæces in, 583.
hemiplegia in, 582.
muscular sensation of the paralyzed side in, 582.
sensibility of the skin of the affected side in, 533.
unilateral suppression of tactile sensation on the contralateral side in, 582.
Buccinator paralysis in lesion of the facial nerve, 472.
Bulbar myelitis, acute, and bulbar hæmorrhage, 611, 615.
Bulbar paralysis, acute apoplectiform, 611; due to compression of the pons medulla, 617; functional asthenic (without anatomical findings), 616, 617.
anatomical findings in, 612.
anterior, 616.
chronic progressive, 612.
differential diagnosis, 614; from multiple sclerosis, 615.
disturbances of speech in, 612; secondary, 613.
dysphagia in, 613.
electric irritability of the muscles in, 614.
muscular atrophy in, 612.
of supranuclear origin, 615.
Bulbar paralysis, ophthalmoscopical phenomena in, 612.
paralytic phenomena of the cerebral nerves in, 613, 614; bulbar paralysis, progressive, 615.
reflex irritability in, 614.
respiration and pulse frequency in, 614.
salivation in, 614.
Bulbar phenomena in amyotrophic lateral sclerosis, 556, 614.
in Graves's disease, 762, 763.
in Landry's paralysis, 586.
in polyneuritis, 508.
in spinal progressive muscular atrophy, 566, 570, 614.
in syringomyelia, 572.
Bulimia, nervous phenomena, diagnosis of, 299.

C

Cachexia strumipriva, 759.
Cæcum, inflammation of, 312.
occlusion, 335.
ulceration, 322.
Caisson paralysis, diagnosis criteria, 5 9.
Calves of the leg, spasm of, occurrence of, in Asiatic cholera, 933; in dysentery, 930; in neuritis, 506, 508.
origin of, 503.
Cancer cachexia in gastric cancer, 281.
in hepatic cancer, 198.
in intestinal cancer, 325.
in renal cancer, 386.
Capillaries, blood-current in, in diseases of the heart, 1.
Capillary bronchitis, 92.
acute inflammation of the lungs in, 93.
auscultatory symptoms, 93.
complicated by atelectasis, 93; complicated by acute miliary tuberculosis, 93; complicated by catarrhal pneumonia, 93.
cough in, 93.
Curschmann's spirals in, 94.
inspiratory retraction of the epigastrium in, 92.
pectoral fremitus in, 93.
sputum in, 93.
Capillary pulse in anæmics, 784.
aortic insufficiency, 23.
pulmonary insufficiency, 29.
Capsule, internal, anatomical location of, in the brain, 634.
hæmorrhages into the, 669.
morbid foci of, and symptoms, 645; motor, 645; sensory, 646; vaso-motor, 647.
position of the various fibre tracts in, 648.
Caput medusæ of the umbilical region in cirrhosis of the liver, 182.
Caput obstipum spasticum, attitude of the head in, 498.
Carbohydrates, transformation of, in the liver, 171.
Carbonic-acid gas intoxication in capillary bronchitis, 92; in laryngeal stenosis, 71; in pulmonary atelectasis, 106; in pulmonary congestion, 109; in pulmonary œdema, 118.
Carcinoma of the bladder, 399.
of the gall-bladder, 202; of the intestines, 325; of the kidneys, 385; of the larynx, 79; of the liver, 196; of the lungs, 145.
of the œsophagus, 246; causing œsophageal stenosis, 251.
of the palate and pharynx, differentiation from gummatous nodes, 245.
of the pancreas, 223; of the peritonæum, 419; of the pleura, 168; of the rectum, 392; of the spleen, 232; of the stomach, 280.
Carcinoma recti, differentiation from dysentery, 931.
Carcinoma villosum of the urinary bladder, 399.
Carcinomata, metastatic, in the brain, diagnosis of, 690.

Cardia, occlusion of the, insufficient, of the stomach, 307; spasmodic, 305.
Cardiac dulness in bronchial asthma, 103; in cardiac asthma, 60; in mediastinal tumours, 147, 151.
diffusion of, in aortic insufficiency, 20; in acute endocarditis, 7; in fatty heart, 51; in hypertrophy of the heart, 56; in mitral insufficiency, 12; in mitral stenosis, 16; in pericarditis, 39; in tricuspid insufficiency, 30.
Cardiac murmurs, anæmic, 783, 784.
in acute articular rheumatism, 974; in erysipelas, 893; in Graves's disease, 759; in leucæmia, 794; in malaria, 979.
Cardialgia in duodenal ulcer, 321.
of gouty patients, 846.
of neurasthenics, 733.
Caries of the bones of the skull the cause of cerebral abscess, 695
of petrous bone the cause of abscess of the brain, 695; paralysis of the facial nerve, 479; purulent meningitis, 710.
Caries of vertebral bodies the cause of acute spinal meningitis, 531; myelitis, 581.
Carotid artery, murmur in the, in aortic insufficiency, 23.
Casein digestion, 257.
Catalepsy, 496.
of hysterics, 726.
Cataract in diabetics, 831.
in gout, 846.
Catarrh, 90, 91.
Cauda equina, tumours of, symptoms, 584.
Cavities, bronchiectatic and phthisical, differentiation, 100.
in pulmonary abscess, 142, 143.
in tuberculosis, 136, 137.
large, and pneumothorax, differentiation of, 164.
Cell activity faulty in obesity, 843.
Central convolutions of the cortex of the brain, focal symptoms of, 649; in abscess, 693.
Central nervous system, congenital defective "anlage," 549; rheumatic intoxication of, 974.
Centre ciliospinal, anatomical position in the spinal cord, 524.
ovale, focal affections of, 661; diagnosis of, 662.
vaso-motor, irritation phenomena of, in cerebral anæmia, 700.
Centres of the corpora quadrigemina (for contraction of the cardia and stomach), 630.
of the cortex of the brain, motor, 640, 641, 650, 651; sensory, 639, 640, 642.
of the medulla oblongata for reflex movements, 604 (disturbances of), 617.
of the optic thalami (for reflex movements of expression), 643, 644.
spinal, of the cervical cord, 524; of the lumbar cord, 524; of the vaso-motors, 525.
Cephalæa (see Cephalalgia).
Cephalalgia, ætiological factors for the diagnosis of, 463, 464.
differentiation from headache due to cerebral tumours, 687; due to trigeminus neuralgia, 453, 463.
ophthalmological examination in, 463.
Cerebellar lateral-column tracts, affections of, and symptoms, 527.
communication of, with Clarke's column, 521, 621.
functional implication of, for co-ordination, 521, 542, 543, 621.
their course in the cerebellum, 621, 622; in the spinal cord, 514, 521.
Cerebellum, affections of, anatomico-physiological conditions, 619, 620, 621, 622; diagnosis of, 622.
functions of, 621.
in cerebellar abscess, 693.
in cerebellar tumours, 622, 623, 681.
in disease of the brachium pontis, 623.
in lesion of the vestibular nerve-fibres, 622.
Cerebellum, origin and distribution of cerebellar peduncle fibres in the, 543, 619, 620.
relations of the acoustic nerve to the, 621.
Cerebral abscess in bronchiectasis, 102.
Cerebral arteries, atheroma of, 64.
Cerebral hæmorrhage, 664.
apoplectic insult in, 664; absence of, in cases of, 675; genesis and consequences of, 665.
diagnosis of, 668; differentiation from epilepsy, 674; from meningitis, 672; from sepsis, 675; from softening of the brain, 675; from symptomatic apoplexies, 673; from uræmia, 675.
electrical conditions of the paralyzed nerves and muscles in, 671.
exempted areas in the paralyzed regions in, 668.
focal symptoms of, 666, 668, 669, 671; post-hemiplegic, 672; with absence of insult, 675.
in contracted kidney, 363; in hypertrophy of the heart, 56; pathogenesis of, 665; prodromes of, 665; seat of, 667, 668; topical, 667, 668, 670.
Cerebral peduncles, affections of, clinical observation of, 631, 670; clinical diagnosis of, 630.
paralysis of the extremities (contralateral), 631; of the facialis, 475, 476; of the hypoglossus, 482; of the oculomotorius (alternating, partial and total), 631; pyramidal lateral-column degeneration, 552.
vaso-motor disturbances due to, 631, 632.
Cerebral peduncles, anatomical structure of, 626.
of the lemniscus, 627, 628, 637.
of the pes, 626.
of the tegmentum, 626, 627, 637.
function of, 630.
Cerebral phenomena in atheroma and sclerosis of the coronary arteries, 64; fatty heart, 52; pneumonia, 124.
uræmic, 363, 364.
Cerebral rheumatisms, 972.
case of, 972.
Cerebral sclerosis, diagnosis of diffuse, 696, 697.
of disseminated, 696.
Cerebral sinuses, inflammation of the sinus wall, 714.
thrombosis and phlebitis of, 714; origin of, 714; symptoms of, 715, 716; thrombosis of the sinus cavernosus, 717; of the sinus longitudinalis, 717; of the sinus transversus, 716.
Cerebral symptoms in acromegaly, 757; in acute articular rheumatism, 971, 972; in Asiatic cholera, 933; in diphtheria, 944; in durhæmatoma, 719; in erysipelas, 893; in measles, 867; in multiple spinal-cord sclerosis, 591; in scarlatina, 874, 876; in small-pox, 886; in tabes dorsalis, 539; in typhoid fever, 911, 914, 916, 918.
Cerebral typhus, 919.
diagnosis of, 926, 954.
Cerebro-spinal meningitis, epidemic, 708, 949.
apoplectiform, 953.
causes for the origin of, 953.
complications, 951.
diagnosis, 708, 949; differentiation from cryptogenetic septicopyæmia, 954; from enteric fever, 954; from purulent, non-epidemic meningitis, 954; from tubercular meningitis, 953.
fever (see Epidemic Cerebro-Spinal Meningitis).
infection in, 951, 952, 953.
microbes of, 709, 952, 953.
occurrence in epidemics, 952.
primary cryptogenetic, 954.
prodromal symptoms, 950.
scaphoid retraction of the abdomen in, 950.
sequelæ of, 951.
sporadic occurrence of, 953.

Cerebro-spinal meningitis, symptoms of, 951.
typhoid, 954.
Cerebrum, basal ganglia of the, function of, 642.
Cervical marrow, inflammation of, diagnosis and symptoms of, 577.
Cervical nerves, neuralgias of, 453; paralyses in the four upper, 483; in the four lower, 484; spasms in the region of, 499.
Cervico-brachial neuralgia, diagnosis, 454.
Cervico-occipital neuralgia, diagnosis of, 453, 463.
Changes of position of the kidneys, 391; of the liver, 206; of the spleen, 233; of the stomach, 291, 292.
Charcot's crystals, and bronchial asthma, 104, 105.
fibrinous capillary bronchitis, 94.
in the blood, in leucæmics, 792.
Cheek, flapping of the, 472.
gangrene of the, 237.
Chest, examination of the, upon determination of the diagnosis of an affection, vii.
retraction of the region of the apex-beat in pericardial synechiæ, 46.
Cheyne-Stokes phenomena in apoplectic insult, 664; in fatty heart, 52; in meningitis, 704; in epidemic cerebro-spinal, 950; spinal, 530; pons-medulla affections, 608; uræmic intoxication, 359.
Chicken breast due to rhachitic change of the thorax, 853.
Chicken-pox, 889.
eruption in, 889.
Chills in embolism of pulmonary artery, 140.
in croupous pneumonia, 123.
in embolism of spleen, 230.
in renal abscess, 370.
Chiragra, 844.
Chlorosis, 778.
ætiology, 789; blood formation in, 780; coagulability of the blood in, 781; functional disturbances in, 781; hæmoglobin contents of the blood in, 778, 779, 789; hæmorrhages due to, 786; occurrence of, 789; sinus thrombosis due to, 714.
Choked disk (engorgement papilla), 617.
in brain tumours, 681, 685, 686, 687, 688.
in cerebellar, 624.
in hydrocephalus, 720.
in meningeal hæmorrhages, 719.
Cholangeitis purulent, diagnosis, 217.
differential diagnosis from Weil's disease, 214.
Cholelithiasis, 214.
differential diagnosis of the attacks of fever of, from intermittent fever, 983.
differential diagnosis between abscess of the liver, 190; catarrhal jaundice, 212; gastralgia, 215; gastric ulcer, 216; malarial fever, 216; renal-stone colic, 216.
jaundice in, 215.
Cholera asiatica, cholera infectiosa, 931.
abortive forms of, 932.
ætiological factors of, 935.
algida, 932, 933.
asphytica, 932.
blood inspissation in, 931.
diagnosis of, 932, 933, 935; differential, from cholera nostras, 937; from intoxications, 937.
incubation period, 931.
intestine, conduct of the, in, 934.
local, etc., predisposition to, 936.
origin of, due to bacilli (vibriones), 935.
phenomena of reaction in, 932.
sicca, 934.
significance of the water for the distribution of, 936.
stages of the course of, 932.
symptoms, general, on the part of the circulatory organs, 933; of the muscles, 933; of the respiratory organs, 933.
toxine effects in, 932, 933, 935.
urine excretion in, 933.
Cholera kidney, 350.
Cholera morbus, 933.
differentiation from Asiatic cholera, 937.
Cholera nostras, 309.
bacilli in, 309.
symptoms of, 309.
Cholera *roth* reaction, 935.
Cholera typhoid, 932.
Cholera vibriones, 935.
Cholesterin plates in the sputum, in pulmonary abscess, 142.
Chorda tympani, gustatory nerve-fibres of, 438, 441, 470.
paralysis of, 469, 473.
salivary secretion fibres of, 471.
Chorditis vocalis inferior, 69; superior, 68; tuberosa, 70.
Chorea, 744.
complication with articular rheumatism, 746, 975; with endocarditis, 745.
diagnosis, 746; differential, from athetosis, 746; from myoclonia, 748.
disturbances of co-ordination, 744, 746.
electrica, 745, 746.
Huntington's, 745.
major, of hysterics, minor, 744.
nature of, 745.
pathogenesis of, 745, 746.
predisposing factors of, 745.
prehemiplegic and posthemiplegic, 672, 745.
Sydenham's, 744.
symptoms of, 744.
time of occurrence of, 746.
Choreic movements, 744, 745.
after apoplexies, 672.
in lesion of the optic thalami, 643.
Choreoid tubercles in miliary tuberculosis, 923.
Chorio-retinitis of diabetics, 831.
Cicatrices in the larynx, 77.
in the œsophagus, 250.
in the stomach, 275.
Circulatory disturbances in affections of the heart, 1.
in cardiac asthma, 59; in hypertrophy, 53, 54; in cholera asiatica, 931, 933; in enteric fever, 917; in interstitial pneumonia, 129; in mediastinal tumours, 149; in mitral stenosis, 16; in pericarditis, 41; in pulmonary atelectasis, 107; in pulmonary congestion, 109; in pulmonary emphysema, 112.
of the brain, 664; of the spinal cord, 594.
Circulatory organs, affections of, in anæmia, 783.
in diabetes mellitus, 829, 830.
in gout, 846, 847.
in typhoid fever, 917.
Cirrhosis of the liver, 180.
ætiological factors, 183.
ascites due to, 182; and ascites due to chronic peritonitis, 185; due to pylephlebitis and pylethrombosis, 184.
Caput Medusæ of the umbilical region, 182.
collaterals between portal vein and cava circulation 4n, 182.
differential diagnosis of, 183; from atrophic nutmeg liver, 184; from fatty liver, 195; from simple marantic atrophy of the liver, 184; from syphilis of the liver, 184.
gastric and intestinal catarrh in, 181.
granulation of the liver in, 181.
hæmorrhoids in, 182.
jaundice in, 183.
portal-vein stasis in, 182.
the urine in, 183.
Cirrhosis, hypertrophic, 185.
blood in, 186.
differential diagnosis, 186.
engorgement of the portal vein, 186.
jaundice in, 186.
symptoms of, 186.
urine in, 186.
Clarke's columns in the spinal cord, degenerative—atrophic changes of, in hereditary ataxia, 550.
position and nerve elements of, 515, 517, 521, 522, 524.
Clavus hystericus, 728.
Claw position of the hand in progressive

muscular atrophy, 564; in syringomyelia, 572; in ulnaris paralysis, 489.
Cleidagra, symptoms of, 844.
Club-foot position in acute anterior poliomyelitis, 560.
Cocci, capsular, as generators of pneumonia, 121.
Coccygodynia, origin and symptoms, 462.
Cold, points of, 433.
Colic, 342.
causes of, 343.
diagnosis of the various forms of, 343.
differentiation of, due to intestinal affections from gall-stone and renal colic, 343; from peritonitis, 343.
due to pancreatic calculi, 225.
in intestinal catarrh, acute, 308; chronic, 317.
Colic, nervous, 342.
hysterical, of abdominal wall, 344.
Colitis, acute, diagnosis, symptoms, 315.
Collapse in atheroma of the coronary arteries, 64.
in peritonitis, 408.
in perityphlitis, 313.
symptoms of, in anthrax, 996; in Asiatic cholera, 931; in diabetic coma, 833.
Collateral circulation between the portal vein and cava in hepatic cirrhosis, 182.
the upper and lower halves of the body in persistence of the aortic isthmus, 38.
Collaterals of the nerve processes, course of, in the spinal cord, 516, 517.
functions of, 426.
termination of, in the spinal cord, 517, 522, 524, 543.
Colon, carcinoma, 325.
differential diagnosis between carcinoma of the, and gastric cancer, 327; pancreatic cancer, 224.
occlusion of the, 325.
position of the, in relation to renal tumours, 327, 328.
transverse, inflation of, and atrophy of the liver, differentiation of, 179.
Colour index, 788.
Colour sense, disturbances in hysteria of, 729; in tabes dorsalis, 538.
Columns, lateral, of the spinal cord, diseases of, and symptoms, 527; degenerative, 552, 553.
fibre systems of, 514, 515.
structure of, in the medulla, 596, 597.
Coma in atrophy of the liver, 176.
in cholæmic intoxication, 209.
in uræmic intoxication, 359.
Coma, diabetic, 832.
in cerebral rheumatism, 972.
in cholera, 932.
in meningeal hæmorrhages, 719.
meningitis, 703; in epidemic cerebro-spinal, 950.
varieties, 833.
Comma bacilli (cholera vibriona), 931, 935.
action of the toxines of, 932.
cultures of, 935.
diagnostic importance of, 935.
differentiation from similar spirilli, 937.
occurrence of, 935.
resistibility of, 936.
Commissures of the brain, anatomical conditions of, 635.
nerve-fibres of, 515, 517.
of the spinal cord, 513.
Compensation of cardiac dyspnœa, 39.
circulatory disturbances, of the heart, 3.
resorbability of the stomach in dilatation, 290.
Compression myelitis, 579.
initial irritative symptoms of, 580.
interruption of conduction in the spinal cord, 579.
seat of the compression in, 581.
secondary degeneration due to, 552, 581.
unilateral, 582.
Compression of veins (see Venous Compression).
Compression phenomena of mediastinal tumours, 148, 149, 150.
Compression stenosis of the biliary passages, 211.
of the trachea, 97, 148.
Concretions, examination of the, 389.
formation of, in the kidneys and ureters, 389, 390; in the uric acid, 389.
Condylomata of the palate and pharynx, 244.
of the larynx, broad, 75.
Conjunctivitis, diphtheritic, 943.
in facialis nerve paralysis, 472.
in whooping-cough, 946.
Connective-tissue hyperplasia of the liver (see Hypertrophic Cirrhosis).
Consciousness, disturbances of, in acute miliary tuberculosis, 956.
in apoplectic insult, 665; in cholera, 932; in cryptogenetic septicopyæmia, 967; in diabetic coma, 832; in epileptic attacks, 736, 738.
Constipation in chronic intestinal catarrh, 316.
in intestinal stenosis due to cancer of the bowel, 325.
in peritonitis, 410.
in perityphlitis, 312.
in stasis conditions, 341.
nervous, 341.
Constitutional diseases, diagnosis of, 772.
multiple neuritis in, 505.
Constrained conceptions of neurasthenics, 732.
Consumption, galloping, 134.
Contractures in acute infantile encephalitis, 666.
in brain tumours, 683.
in capsular affections, 646.
in hysteria, 725.
in pachymeningitis cervicalis hypertrophica, 534.
secondary, in anterior poliomyelitis, 560.
spastic, in affection of the pyramidal antero-lateral column tracts of the spinal cord, 525.
subsequent to cerebral hæmorrhages, 671.
Conus affections of the spinal cord, diagnosis of, 585.
nature and symptoms of, 586.
Convexity meningitis, general phenomena and focal symptoms, 707.
Convulsions, 496.
epileptic, 736; in brain abscess, 695; in brain tumour, 681; in cortical affections of the brain, 662; in meningitis, 703, 704, 705; in Asiatic cholera, 932; in cryptogenetic septicopyæmia, 967; in influenza, 986; in poliomyelitis, anterior, acute, 559; in rhachitis, 853.
Co-ordination, centre of, 521, 622.
innervation tracts of, 542, 619, 620, 621, 622.
mechanism of, 542, 622.
Co-ordination, disturbances of, in athetosis, 747.
in cerebellar affections, 623.
in chorea, 744, 745.
in disease of the cerebellar lateral-column tracts, 527, 543.
in multiple sclerosis of the spinal cord, 590.
in neuritis, 507.
in syringomyelia, 572.
in tabes dorsalis, 539; causation of, 542, 543.
subsequent to diphtheria, 945.
Co-ordination spasms, varieties of, 502.
Corona radiata fibres in the brain, 634, 635.
interruption of, and symptoms, 643.
Corona radiata, optic, cerebral (Gratiolet's), 628.
cortical, 635, 642.
Coronary arteries, sclerosis of, 63.
spontaneous rupture of the heart in, 53.
stenocardiac attacks in, 58, 63.
Corpora quadrigemina, anatomical position and structure of, 628, 643.
function of, 630.

Corpora quadrigemina restiformia, anatomical position and structure of, 602, 621, 622.
symptoms and diagnosis of disease of, 630, 633.
Corpus callosum, fibres of, 635.
ciliare (dentatum), position of, in the cerebellum, 619.
striatum, 634; foci of, and symptoms, 645, 661; functions of, 643.
subthalamicum (Luys's body), anatomical position, 626.
Corrugator supercilii, paralysis of, 472.
spasm of, 497.
Corset liver, 206.
and intestinal cancer, 327.
and wandering kidney, 206.
Cortical areas, 637, 638.
psycho-motor, 640; region of, 641, 651.
psycho-sensory, 638, 642.
Cortical hæmorrhages, 670.
Coryza in influenza, 986, 988.
in pertussis, 946.
in typhoid fever, 917.
Cough in capillary bronchitis, 93.
in laryngeal catarrh, 68; laryngitis, 68; diphtheritic, 71.
in pneumonia, 124, 127.
Cough, spasm of, origin of, 501.
Coxalgia, nervous, diagnosis of, 462.
differentiation from sciatica, 459, 460.
Coxitis, differential diagnosis from sciatica, 459.
Cramps, character of, 496.
in the calves of the legs, 503.
Craniotabes, consequences of, 852.
formation of the skull in, 852.
Cremaster reflex in apoplectic attacks, 665.
Crepitatio indux and redux of the lung in pneumonia, 123.
Crepitation and friction sounds, pleuritic, differentiation, 154.
in atelectasis of the lungs, 107, 108.
in pneumonia, 123, 127.
Cricoarytænoid muscles, paralysis of, 87.
Cricothyreoid muscle, paralytic symptoms of, 83.
Cri hydrencéphalique in meningitis, 703.
in epidemic cerebro-spinal, 950.
Crises nephrétiques, differentiation from renal colic, 389.
Croup, characterization of, 941.
Crural double sound in anæmics, 784; in aortic insufficiency, 23.
Crural nerve, neuralgia of, 456.
paralysis of, symptoms of, 493.
Crural vein sounds in tricuspid insufficiency, 31.
mixed double, sound, 31.
Cutaneous emphysema due to œsophageal rupture, 253.
Cyanosis in bronchial catarrh, 91; in congenital aortic stenosis, 28; in diseases of the heart, 1; in mediastinal tumours, 148; in myocarditis, 49; in pericarditis, 410; in pleurisy, 157; in pneumonia, 127, 129; in pulmonary atelectasis, 106; pulmonary stenosis, 29; in tricuspid insufficiency, 30; in tricuspid stenosis, 33.
Cysticerci in the brain substance, diagnosis of, 690.
Cystitis, 393.
acute, 393, 395.
and pyelitis, differentiation, 396.
calculous, 397.
contracted kidney, 395.
croupous diphtheritic, 397.
diagnostic symptoms of, 393; origin of, due to imported bacteria, 394.
gonorrhœic, 397; condition of the urine in, 397; mild and severe forms, 395.
submucous, parenchymatous, 398.
tuberculous, 398; causes of, 395; thickening and expansion of the bladder, 395.
Cystitis in acute articular rheumatism, 974.
in mumps, 940.
in myelitis, 576.
Cystitis in tabes dorsalis, 539.
Cystoplegia, 401.
Cystoscopy in carcinoma of the bladder, 399.
in urinary concrements in the bladder, 397; in the kidneys and ureters, 390.
Cystospasm, 402.
Cysts of the kidneys, 381; of the larynx, 79; of the mesentery, 421.
Cytoryctes variolæ in the blood of smallpox patients, 381.

D

Deafness in focal affections of the temporal cortex, 651.
Decubital ulcer of the œsophagus, 246.
Defluvium capillitii in diabetes, 829.
in erysipelas, 895.
Degeneration, fatty, of heart, 51.
gray, of the cerebral nerve nuclei in progressive bulbar paralysis, 612, 613.
of the motor conduction tract, 552.
of the posterior columns of the spinal cord in tabes dorsalis, 537, 538, 540.
of the pyramidal tracts in progressive bulbar paralysis, 612.
origin of, 519, 520, 521.
parenchymatous, of the muscles in typhoid fever, 918; of the peripheral nerves, 558.
Deglutition, activity of, centre, 604.
disturbances of, in accessorius paralysis, 481; in meningitis, 704; in pons-oblongata affections, 608, 609.
spasms of, hysteria, 726; in hydrophobia, 993; in tetanus, 991.
Deglutition, difficulty in cancer of œsophagus, 246.
perichondritis, 73.
spasm of œsophagus, 249.
stenosis of œsophagus, 247.
Delirium due to cholæmic intoxication, 209.
due to uræmic intoxication, 359.
in acute yellow atrophy of the liver, 176.
Delirium in acute miliary tuberculosis, 955; in cholera, 933; in influenza, 987; in meningitis, 703; in rabies, 993; in rheumatic intoxication of the nervous system, 971; in trichinosis, 999; in typhoid fever, 911, 913; in typhus fever, 896.
Delirium tremens, differentiation from meningitis, 702.
Deltoid muscle, atrophy of, in progressive muscular atrophy, 564.
paralysis of the, 486.
Dementia paralytica, 697.
as ætiological factor of durhæmatoma, 718.
differentiation from brain tumours, 698; from paralysis agitans, 753.
disturbances of the body in, 697.
paralytic attacks in, and their differentiation from apoplectic and epileptic attacks, 698.
pathological findings in, 697.
psychical disturbances due to, 698.
tabetic symptoms of, 697.
Dendrites of the nerve cells, function of, 425.
their anatomical condition in the spinal cord, 518.
Dengue, ætiology, 1002; diagnosis, 1003; differential, 1003.
Depression of the function of the gastric glands, 303, 307.
Depressions, psychical, in chorea minor, 744.
of hysterics, 730.
subsequent to epileptic attacks, 740.
Dercum's disease, 844.
Descusus ventriculi (see Gastroptosis).
Desquamatia furfuracea in measles, 866.
Detrusor spasm of the bladder, 399.
combined with spasm of the sphincter, 403.
Diabetes decipiens, 829.
Diabetes insipidus, 837.
differential diagnosis from diabetes mellitus, 838; from nephritis, chronic, 837; from symptomatic polyuria, 838.
diuresis in, 837.
symptoms of, 838, 839.

Diabetes mellitus, 823.
acid intoxication of the body of diabetics in, 832.
and diseases of the pancreas, 222, 226.
and pulmonary gangrene, 145.
blood condition in, 830.
carbohydrate metabolism in, 834.
coma after, 832; varieties of, 833.
combination of, with acromegaly, 757; with adiposity, 843; with albuminuria, 828; with bronzing, 829; with contracted kidney, 828; with polyuria, 828.
diagnosis of, 823, 833; differentiation from glycosuria, 834, 835, 837; from lactosuria, 837.
epilepsy following, 742.
influence of the pancreas upon occurrence of, 836.
mild and severe forms of, 833, 834.
modification of sugar excretion, 826.
nature of, 834, 835, 836.
predisposition of diabetics to apoplexy, 833; to phthisis and pulmonary gangrene, 829; to sciatica, 458.
reaction of the urine in, to acetone and oxybenzoic acid, 827; to sugars, 824, 825.
sensation of thirst, 832.
symptoms on the part of the circulatory apparatus in, 829; of the digestive organs, 830; of the respiratory apparatus, 829; of the nerves, 830; of the sexual organs, 830.
Diagnosis, anatomical, viii.
changing of, x.
characterization and origin of, i.
clinical, xi.
deductive method of, vi.
determination of, vii.
differential, ix.
epicritic, xi.
examination of the patient before making a special, vi, vii.
inductive method of, vi.
mental digest of symptoms to establish the special, ix.
Diagnosis of diffuse affections of the brain, 699; functional (neuroses), 723.
diseases of the blood and metabolism (constitutional diseases), 772; cerebellum, 619.
focal affections of the anterior brain, 664.
infectious diseases, 856; medulla oblongata and pons, 596; middle brain, 626; muscles, 765.
nervous diseases, 425; peripheral nerves, 426; motor, 465, 495; sensory, 435.
spinal-cord diseases, 512; meninges of, 529; spinal-cord substance, 536.
Diaphragm, high position of, in acute peritonitis, 409.
action of mediastinal tumours on, 149.
low position of, in exudative pleuritis, 155.
Diaphragm, paralysis of the, ætiology, 484; diagnosis, 483.
spasm of, clonic, 500; of hysteric, 726; of tonic, 500; and bronchial asthma, 105.
Diaphragmatic hernia and intestinal stenosis, 337.
Diarrhœa in acute colitis, 315; in Asiatic cholera, 931, 935; in cæcal intussusception, 336; in contracted kidney, 363; in dysentery, 928; in fatty kidney, 366; in Graves's disease, 760; in influenza, 986; in intestinal catarrh, 311; in intestinal ulcer, 319; in invagination, 336; in nephritis, 359; in peritonitis, 410; in septic infection, 967; in tabes, 538; in trichinosis, 999; in typhoid, 908, 911, 912.
nervous, 339, 345; in children, 339; in hysteria, 339; in tabes, 339.
profuse uncontrollable, 345.
Diastolic murmur, in aortic insufficiency, 20, 24, 63.
in combined valvular defects, diagnostic utilization of, 35.
in mitral stenosis, 16.
in pulmonary valve insufficiency, 29.
utilization of, 35.
Diastolic valve closure at the pulmonary orifice in mitral insufficiency, symptoms, 14.
Diazo-reaction of the urine in malaria, 982.
in pulmonary tuberculosis, 139.
in typhoid fever, 914, 919, 923.
Differential diagnosis, methods, ix.
Digestion of albumin products, 257; of fats, 258; of milk, 257; of cane sugar, 257.
action of the nervous system upon, 258.
function of the stomach in, 257, 258.
investigation of, 261, 262.
slowed and its causes, 263.
Digestion, metabolism in, 820.
Digestive disturbances in acute intestinal catarrh, 311.
diagnosis of, 235, 259.
in contracted kidney, 363.
in gastrectasis, 287.
in myocarditis, 49.
in neuroses of the stomach, 294.
Digestive organs, disease of, diphtheritic, 942.
in Asiatic cholera, 934.
in diabetes, 830.
in influenza, 985, 986.
in septicopyæmia, 967.
in typhoid fever, 916.
Digestive tract, diseases of, 237.
Digitalis for diagnostico-therapeutic purposes in pericardial affection, 42.
Dimpling, systolic, in pericarditis, 46.
Diphtheria, 940.
complications of, 710, 866, 875.
diagnosis of, 940; differential, 949.
general infection in, 941.
incubation period in, 942.
mixed infection by streptococci and staphylococci, 941.
mode of infection in, 941.
of the nasal mucous membrane, 941, 943.
of the œsophagus and stomach, 942.
of the pharyngeal mucous membrane, 942.
origin of, by specific bacilli, 940, 945.
pseudomembranes, 941.
symptoms of, local, 941; motor, and sensory, 944, 945.
traumatic, following pharyngeal, 943.
Diphtheria bacilli, morphology, culture and transmission of, 940.
Diphtheria of the larynx, 70, 941, 943; due to scarlatina, 244; of the palate and pharynx, 242.
and angina lacunaris, differentiation, 243.
bacilli as generators of, 242.
character of the membranes, 243.
corroded crusts, differentiation, 242.
gangrenous, 243.
Diplegia facialis in progressive bulbar paralysis, 613.
Diplococci, ætiological relation of, to acute articular rheumatism, 970.
Diplococcus intracellularis meningitides, the cause of epidemic cerebro-spinal meningitis, 708, 952.
Diplococcus pneumonia, 121, 127.
Displacement signs in mediastinal tumours, 146, 147, 148, 149.
in pleurisy with effusion, 155.
in pneumothorax, 166.
Distoma hepaticum in the bile-ducts causing jaundice, 211; obstructing the bile ducts, 211.
Diverticular sound to diagnosticate "pulsions divertikel," 253.
homonymous, 466.
in paralysis of the trochlearis, 466.
subjacent, 466.
Dorsal nerves, paralyses in the course of the, 492.
Double images of the field of vision, synonymous adjacent in abducens paralysis, 465, 466.
Double sensations of tabetics, 545.
Dropsy in contracted kidney, 363.
in engorged kidney, 349.
in fatty (amyloid) kidney, 366.
in heart disease, 1.

Dropsy nephritis, acute, 352; chronic, 357.
vesical filiæ, 217; differential diagnosis, 218.
Dropsy, intermittent articular, 756.
due to acute rheumatism, 975.
of the cerebral ventricles, 719.
Drug exanthems, differentiation from measles, 867; from varicella, 891.
Drum, cavity of the, diphtheria of the, 943.
Duchenne's disease, 612.
glossopharyngeal-laryngolabial paralysis in, 612.
Duchenne-Erb paralysis, diagnosis, 491.
Ductus Botalli, persistence of the, 37.
cysticus, obturation of, 207; gall-bladder ectasis due to, 207.
differential diagnosis between stenosis of the orifices, 37.
thoracicus deficient deflux of chyle from the, in pulmonary emphysema, 114.
Duodenal carcinoma, 326; causing jaundice, 221.
differential diagnosis between, and gastric carcinoma, 285, 327; pancreatic carcinoma, 224.
Duodenal catarrh, acute, djagnosis, 309.
Duodenal occlusion, due to cancer of the pancreas, 221.
Duodenal ulcer, 321, 322; differential diagnosis between gastric and duodenal ulcer, 277, 321.
Dura mater, inflammation of (see Pachymeningitis), tumours of, symptoms of, 584.
Dysarthria in hereditary ataxia, 550.
in pons affection, 608, 616.
in progressive paralysis, 697.
in pseudobulbar paralysis, 615.
Dysentery, 928.
ætiological factors of, 928.
chronic, 930, 931.
complications of, 930.
condition of the stools in, 929.
contagiousness of the dejecta, 928.
differential diagnosis of, 930; from trichinosis, 1000.
infectious substance of, 928.
intestinal stenosis in the train of, 930; ulcerations in, 324.
nostras epidemica, 928.
occurrence of, 928.
relation of, to pharyngeal diphtheria, 928.
symptoms of, 928, 929.
tenesmus in, 929.
tropic, 928.
typhoid, 930.
Dyspepsia, 264.
causes of, 296.
chemical examination of the stomach contents in, 295.
determination of, from other gastric neuroses, 297.
examination of the stomach with the sound, 294; time of digestion, 294.
in cancer of the liver, 198.
nervous, 294.
symptoms of, 294.
time of digestion in normal, increased and decreased acid production, 295.
with excessive acid production, 295.
with normal acid production, 295.
with subacidity or inacidity, 295.
Dyspeptic phenomena in acute articular rheumatism, 974.
in erysipelas, 894; in gout, 846, 847; in influenza, 986; in leucæmia, 794; in nervous, 733, 782; in septicopyæmia, 967; in trichinosis, 999; in typhoid fever, 916; in typhus fever, 899.
Dysphagia in hypoglossus paralysis, 481, 482.
in meningitis, 704; in œsophageal stenosis, 247; in pericarditis, 41; in pons-oblongata paralysis, 607, 613, 617; in vago-accessorius paralysis, 479; in vagus neuritis, 506.
paralytic, 255.
Dyspnœa, bronchiostenosis in, 97; in hæmorrhagic infarct, 140; in hypertrophy of the heart, 56.
Dyspnœa, cardiac, 52, 59; character of, and differentiation from laryngeal dyspnœa, 60.
expiratory, in bronchial asthma, 104; in pulmonary emphysema, 110.
inspiratory, in laryngeal stenosis, 77; in bronchiostenosis, 96.
mediastinal tumours in, 146; paralysis of the posticus in the larynx, 88.
mixed, 60.
pulmonary œdema in, 118; with stridor in spasmus glottidis, 82.
Dyspnœa, diabetic, 830, 832.
in acute miliary tuberculosis, 956; in anæmia, 781; in anthrax, 996; in diaphragm paralysis, 483; in spasm, 501; in diphtheria of the larynx, 943; in hypoglossus paralysis, 483; in leucæmia, 794; in neuritic affection of the vagus, 508; in obesity and corpulence, 840; in trichinosis, 999.
spastic, 255.
Dystrophia muscularis progressiva (Erb), 568.
anatomical basis of, 568.
condition of the nervous system of, 557, 558.
differential diagnosis of, from spinal muscle atrophy, 570.
facial, muscle atrophy in the infantile atrophic form of, 569.
pseudohypertrophy of the muscles of, 569, 570.
Dysuria spastica, 402.

E

Ear affections, diphtheritic, 943.
in influenza, 987.
in mumps, 939, 940.
in scarlatina, 876.
in sinus thrombosis, 714.
Ear, auricle of the, deposit of gout tophi in the subcutaneous cellular tissue of, 844.
paralysis of the muscles of, in lesion of the facialis external to the Fallopian canal, 478.
Echinococci in the brain substance and ventricles, 690.
Echinococcus in the bile-ducts, 210.
in the liver, 204.
in the pleural cavity, 168.
multilocularis and cancer of the liver, differentiation, 199.
of the kidney, 384.
of the lungs, 145.
of the spleen, 232.
Echinococcus of the liver, 204; pleural exudate, 168.
differential diagnosis, 205.
enlargement of the liver in, 204.
exploratory puncture in, 205.
forms of, 204.
hydatid thrill in, 204.
multilocular and hypertrophic cirrhosis, 186.
purulent sac of, and abscess of the liver, 192, 205.
results of physical examination, especially of palpation, 204.
Echinococcus, renal, 384.
consequences of spontaneous evacuation of, 384.
differentiation from hydronephrosis, 383; from renal abscess, 371.
immovability of the tumours of, 384.
perforation of, 384.
puncture fluid of, 384.
Eclampsia of anæmic women and children, 782.
differentiation from epilepsy, 743.
Ectropium paralyticum in facialis paralysis, 472.
Elastic fibres in the sputum in pulmonary abscess, 142; in pulmonary tuberculosis,

135; in the urine in tuberculosis of the kidney, 374.
Electric irritability in amyotrophic lateral sclerosis, 556.
in chorea, 745.
in facial paralysis, central, 475; peripheral, 477.
in Graves's disease, 760.
in hysteria, 725.
in myelitis, 57, 575.
in myotonia congenita, 753.
in neuritis, 506.
in poliomyelitis anterior, acute, 558; chronic progressive, 565.
in syringomyelia, 572.
in tetany, 749.
in traumatic neurosis, 736.
in trichinosis, 999.
of the cerebral cortex, causing epileptic attacks, 737.
subsequent to apoplectic insult, 671.
Elephantiasis, differentiation from acromegaly, 757.
due to erysipelas, 895.
Elephantiasis of the liver, 185, 186, 199 (see also Hypertrophic Cirrhosis).
Embolism of the pulmonary artery, 139.
diagnosis of, in occlusion of the trunk and main branches, 140; of the smaller branches, 140; objective symptoms, 140, 141; origin of, 139.
of the renal arteries, 373.
of the spleen, 230.
Emphysema, interlobular, 116.
mediastinal, 117.
senile, 116.
subpleural, 117.
vicarious, 116.
Empyema, bacteriological examination of the exudate, 162.
differentiation from putrid bronchitis, 92; from pulmonary abscess, 142.
sacculated, 101.
vesicæ fellis, 207.
Encephalitis, acuta infantum, 696.
after infectious diseases, 696.
sclerotic, 696.
suppurative (see Cerebral Abscess).
Encephalomalacia, 676.
ætiological diagnosis of, 679.
apoplectic attack in, 677.
chronic progressive, 680.
differential diagnosis, 679.
focal symptoms of, permanent (direct), 678.
transitory, 678.
Enchondroma of the larynx, 78.
Endarteritis, neuritic affections in, 505.
syphilitic, of the cerebral arteries and its consequences, 675.
Endocarditis, acute, diagnostic characteristics, 6.
ætiological factors, 7.
deviation from the usual clinical picture, 7.
differentiation from accidental heart murmurs, 8.
differentiation of acute from chronic and recurrent, 9.
murmurs caused by myocarditis; by pericarditis, 9.
objective findings in, 6.
of the aortic valves, 7; in infections, 8; in gonorrhœa, 8; septic, 8.
of the mild form, 10; mitral valve, 6.
recurrent, 8; severe form of the, 10.
secondary (metastatic) phenomena, 10.
Endocarditis, acute malignant, 11.
varieties of, 11.
symptoms of, 11.
Endocarditis, chronic (valvular defects), diagnosis of, 11.
of the mitral valves, combined with aortic insufficiency, 22.
Endocarditis in acute articular rheumatism, 974; genesis of, 974.
in diphtheria, 944.
in influenza, 986.
in mumps, 940.
in scarlatina, 875.
Endocarditis, septic (malignant), 965, 966.
subsequent to pertussis, 949.
Endocardium, disease of the, 6.
Enteralgia, 342.
diagnosis of, 343.
varieties of, 343, 344.
Enteric fever, eruption upon the back in, 912; temperature during convalescence in, 910.
Enteritis, acute diffuse (simple), 308.
chronic, 316.
febrile, 309.
membranacea, 317.
phlegmonous and diphtheritic, 315.
special forms of, 309, 310.
Enteritis, complication of acute articular rheumatism and, 975; of scarlatina, 876.
differentiation from typhoid fever, 927.
Enteropathy, nervous, with membrane formation, 317.
Enteroptosis, origin, 292.
enterospasms, 340.
Enterostenosis, consequences, 331.
diagnosis, 330.
location and nature, 333.
peritonitis in, 332.
Enuresis, spastic, 402.
Epigastrium, inspiratory retraction of the, in capillary bronchitis, 92.
pulsation of, in mitral insufficiency, 12; in mitral stenosis, 16.
Epiglottis detractors, paralysis of, and symptoms, 83.
Epiglottis, diagnostic characteristics, 68.
of syphilitic, 75.
Epilepsy, 736.
beginning of, in form of the aura epileptica, 738.
characteristics of, 736.
consequences of epileptic attack, 739.
diabetic, 742.
diagnosis of idiopathic, 737, 739; differential, 741; from apoplexy, 674; from eclampsia, 743; from epileptiform attack in brain tumours, 688; from hysteria, 727, 742; from reflex epilepsy, 742; from symptomatic, 736, 737, 741; from uræmic attacks, 743.
due to functional disturbances of the nervous system, 737, 738.
equivalents of, psychical, 739; somatic, 740.
état de mal due to, 740.
idiopathic (primary), 741, 742.
intervals in, 737, 740.
irregular forms of, 740, 742.
Jacksonian (symptomatic or secondary), 741; in meningeal hæmorrhages, 719.
nature of, 737.
partial, 737.
senile, 742.
simulated, 743.
spasms in, 739; clonic, 739; tonic, 739.
symptoms of, 738, 739, 740.
unconsciousness in the epileptic attack, 738.
upon the basis of an hereditary epileptic or neuropathic taint, 742.
varieties of, 740.
Epileptiform attacks, 736, 737.
beginning of, 739.
characterization of, 737.
differentiation from paralytic attacks, 674.
in brain tumours, 688.
in diphtheria, 945.
in embolism, 677.
in encephalitis, 697.
in Graves's disease, 760.
in multiple sclerosis of the spinal cord, 591.
in pons hæmorrhages, 610.
in tabes dorsalis, 539, 547.
phenomena and consequences of, 739, 740, 741.
prodromes, 739.
rudimentary, 738, 739.
Epithelioma of the larynx, 78.

Erection centre, position of, in the spinal cord, 513.
Erosion ulcer of the larynx, 73.
Eructation, nervous, 306.
Eruption, roseolar in, 867.
differentiation of, from measles, 867.
due to septic infection, 966.
in acute miliary tuberculosis, 957.
in enteric fever, 911, 912, 920, 925.
in typhus fever, 897.
in varicella, 889.
prodromal in small-pox, 881.
Erysipelas, 891.
bullosum, 892.
complications of, 893; with meningitis suppurativa, 710.
cryptogenetic, 891.
diagnosis of, 892; differential from anthrax, 896; from erythema, 896; from lymphangeitis, 895; from phlegmon, 895; from scarlatina, 878.
dissemination of, 892.
exanthems of, 892.
faciei, 892.
incubation period of, 892.
infection, mode of, 891, 892.
migrans, 892.
miliary, 892.
of the mucous membranes, 894.
pemphigoides, 892.
phlegmonosum, 893.
prodromal stage of, 892.
sequelæ of, 895.
symptoms of, 892, 893.
transmission of erysipelas cocci, 895.
Erysipelas, surgical, due to invasion of streptococci, 891.
Erythemata, differentiation of, from erysipelas, 896; from scarlatinal exanthem, 878.
in acute miliary tuberculosis, 957.
in cryptogenetic septicopyæmia, 966.
in influenza, 986.
in measles, 865.
prodromal in small-pox, 881, 887.
Erythromelalgia, symptoms and diagnosis of, 755.
Eudiæmorrhysis of the brain, 699.
Eustachian tubes, diphtheria of, 943.
Examination of the body, vi.
Examination routine, of the patient, to determine the diagnosis, vi.
Exanthem, acute, giving rise to spinal meningitis, 531.
of measles, 865.
of rötheln, 879.
of scarlatina, 874.
of small-pox, 882.
of varicella, 889; differentiation from typhoid, 925; differentiation from varicella, 890.
syphilitic, differentiation from varicella, 890.
Exanthems in acute articular rheumatism, 974.
in Asiatic cholera, 932; in cryptogenetic septicopyæmia, 966; in epidemic cerebrospinal meningitis, 950; in influenza, 986; in myelitis, 575; in neuralgias, 447; in neuritis, 504; in pachymeningitis hypertrophica, 534; in relapsing fever, 901; in tabes dorsalis, 999; in typhoid fever, 912, 925; in typhus fever, 897.
Exophthalmic goitre (see Graves's Disease).
Exophthalmos in Graves's disease, 759, 769.
in myxœdema, 759.
in sinus thrombosis, 717.
Expansile pulsation in aneurysm, 285.
Expiration centre, location of, 605.
Expiration, impaired, in bronchial asthma, 103.
in bronchial catarrh, 90.
in bronchial pulmonary emphysema, 110, 111.
Exploratory puncture in hydronephrosis, 382.
Exploratory puncture in hepatic echinococcus, 205.
in mediastinal tumours, 151, 153.
in pancreatic cysts, 225.
in pericarditis, 45.
in peritonitis, 408.
in pleural exudate, 161.
in pleural tumours, 168.
Expression of the stomach contents after Ewald's method for diagnostic purposes, 259.
Extensor digitorum communis of the arm, paralysis of, 488.
of lower leg, paralysis of, 494.
Extensors of the lumbar region, paralysis of, 492.
Extremities, curvatures of, due to rhachitic processes, 853.
Extremities, paralysis of, after apoplectic stroke, 666, 667, 670; after diphtheria, 944.
in affections of the pyramidal antero-lateral column tracts of the spinal cord, 525.
in focal affections of the central convolutions of the paracentral lobule, 650.
in focal affections of the cerebral peduncles, 631; of the internal capsule, 646; of the pons oblongata, 606, 607, 608.
in myelitis, 574, 575; by pressure, 580.
in neuritis, 506, 510.
in paralysis of the facial nerve of the same side, 475, 476; of the opposite side, 475, 476; of the cervical nerves, 482; of the lumbar and sacral nerves, 492; of the nerves of the muscles of the eyes (alternating and of the same side), 465.
in poliencephalitis, acuta infantum, 696.
in poliomyelitis, anterior, 559, 562.
in relaxed, in affections of the anterior horns and of the anterior roots of the spinal cord, 525; in tabes dorsalis, 540.
in spinal-cord hæmorrhages, 594.
Exudate, peritonitic, sacculated, and hydronephrosis, differentiation, 383.
masking of, by adhesion of anterior borders of the lungs and emphysema of the lungs, pressure symptoms, 42.
pericardial condition of the, 45; differentiation from mediastinal tumours, 151.
pleuritic, condition of the, 161, 162; differentiation from pulmonary infiltration, 158.
Eye muscles, paralysis of, 465.
after influenza, 987.
central, 466.
diphtheritic, 944.
in abducens lesions, 466.
in cerebral tumours, 681, 682.
in lesion of the middle brain, 629, 633; of the oculomotorius, 465.
in multiple sclerosis of the spinal cord, 590.
in poliencephalitis superior, 696.
in progressive bulbar paralysis, 612; in paralysis of the insane, 697; in tabes dorsalis, 537; in trochlearis lesion, 466.
localization of the cause of, 466.
peripheral, 466.
progressive nuclear, 616.
total and unilateral, 466.
with hemiplegia, 466.
Eyes, conjugate deviation of the, in apoplectic insult, 664, 665, 669.
in lesions of the parietal cortex, 649.
in meningeal hæmorrhages, 719.
in pontine affections, 609.
Eyes, cryptogenetic septicopyæmia, 967.
findings of the, in anæmia, 786.
in diabetes mellitus, 831; in gout, 846; in Graves's disease, 759, 760, 764; in hysteria, 729; in influenza, 986; in Klumpke's paralysis, 492; in leucæmia, 794; in measles, 863; in meningitis, epidemic cerebrospinal, 950; tuberculous, 954; in mumps, 940; in scarlatina, 876; in tabes dorsalis, 537; in thrombosis of the cavernous sinus, 717; in trichinosis, 999.

F

Face, asymmetry of, congenital, differentiation from unilateral atrophy of the face, 755.
Face, atrophy, progressive unilateral, 754.
affection of the tongue and palate in, 754.
symptoms of, 754, 755.
Face, expression of, in myxœdema, 758.
in paralysis agitans, 751.
Face, hypertrophy of, unilateral, symptoms of, 754.
Face, paralysis of the, after diphtheria, 944.
lesions of the cortex of the central convolutions, 650.
mimic, phenomena of, 468, 471, 472.
unilateral, in diseases of the pons oblongata, 606.
with central facialis paralysis, 474; peripheral facialis paralysis, 477.
Face, spasms of the, diffuse, 496.
masticatory, 496; in tetanus, 990, 993.
Facial nerve, spasm of, 497.
cause of, 498.
clonic, 498.
diffuse, 498.
partial, 497.
tonic, 498.
Facial nerve, paralysis of, 468.
ætiological factors, 478.
after apoplectic insult, 666, 667, 670, 671.
bilateral, 472, 476.
central, 474, 475, 479.
diagnosis of, 440, 468, 474; of the seat of, 469, 474, 477.
disturbances of taste in, 438, 440, 477.
electric irritability in, 474, 476.
in brain tumours, 685.
in focal affections of the central convolution of the cortex, 650; of the internal capsule, 476, 646.
in lesion of the cortex, 475, 476, 651.
in mumps, 940.
in syringomyelia, 572.
of the central and lower branches, 474, 477, 478.
of the newborn, 479.
partial, of the trunk at the base of the brain, 477, 478.
symptoms of, 468, 471; central, 474, 475; in lesion of the interior branches, 473; of the peripheral, 477; of the ramus frontalis, 472.
unilateral, 472; in pons oblongata affections, 606, 607.
Facial nerves, anatomical distribution of, central, 469, 603, 648.
Chvostek's phenomena in disease of, 749.
communication of, with the trigeminus glossopharyngeal nerves, 438, 470.
functions of the, 471.
Facies tetanica, characteristics of, 990.
Fæcal tumours, differentiation from intestinal cancer, 327; from movable spleen, 233; from paranephritic abscess, 373.
Fæcal vomiting in perityphlitis, 312, 313.
Fæces, discharge of, centre of, in the spinal cord, in spinal meningitis, 530.
Fæces in aneurysm of the hepatic artery, 220.
in cholera nostras, 309.
in discoloration of the, in jaundice, 208.
in gastric carcinoma, 280; catarrh, 265; ulcer, 275.
in hepatic acute yellow atrophy, 177.
in intestinal carcinoma, 325; catarrh, acute, 308, 310; chronic, 316, 317; stenosis, 331, 336; ulcers, 319, 320.
in pancreatic affections, 222, 225, 226.
Fæces, retention of, in enterostenosis, 331, 332.
jaundice due to, 212.
Falling sickness (see also Epilepsy), 736.
False vocal cords of larynx, tubercular ulcers of, 87.
Fat, formation of, in the body, 841.
Fat, resorption in the intestine, importance of bile for, 173.
Fatty degeneration of the heart (see Fatty Heart).
Fatty heart, ætiological factors, 51.
diagnostic signs, 51, 52.
differential diagnosis, 52; from myocarditis, 52.
insufficiency of the heart due to, 53.
Fatty liver, 194.
and amyloid liver, differentiation, 194.
causes of, 194.
cloudy swelling of the parenchyma of the liver in, 195.
physical examination in, 194.
Fatty stools, diarrhœa, in pancreatic stone, 226.
Febris biliosa-hæmoglobinurica, 984.
comitata in malaria, 979.
continua in malaria, 979; in typhoid, 920.
intermittens, 976, 978; anteponens, 979; duplicata, 979; erratica, 981; larvata, 983; perniciosa, 983, 984; postponens, 979; quartana, 978; quotidiana, 978; tertiana, 978; triplicata, 979.
in typhoid fever, 909.
recurrens, 901; fever attacks in, 903, 904.
remittens in malaria, 984; in typhoid fever, 909.
variolosa, 885.
Fermentations, abnormal, in the stomach, in gastrectasis, 287.
in action of HCl on, 256.
Fever, anæmic, 788; in leucæmia, 794.
hectic and intermittent, 983.
in abscess of the brain, 692.
in acute articular rheumatism, 971, 972; in acute infantile encephalitis, 696; in acute miliary tuberculosis, 956; in acute myelitis, 577; in anthrax, 995; in diphtheria, 942; in dysentery, 928; in erysipelas, 893; in gouty attacks, 844; in influenza, 985; in malaria, 979; in measles, 863, 864, 865.
meningitis, 703; in acute spinal, 530; in epidemic cerebro-spinal, 949, 951; in mumps, 938; in neuritis, circumscribed, 504; in multiple, 508; in paroxysmal, hæmoglobinuria, 814; in poliomyelitis anterior, 559; in polymyositis, 766; in pseudoleucæmia, 802; in relapsing fever, 903, 904; in rhachitis, 854; in rötheln, 880; in scarlatina, 872; in septicopyæmia, 965; in small-pox, 881, 884, 885; in tetanus, 990, 991; in trichinosis, 999; in typhoid fever, 909, 910, 911, 912, 918, 919, 920; in typhus fever, 897, 898, 899; in varicella, 890.
Fever, enteric, in pulmonary atelectasis, 107.
and uræmia, differentiation from, 360.
and Weil's disease, 213.
in splenic tumour, 229.
with pneumonic infiltrations, 123.
Fever in acute intestinal catarrh, 309.
in acute yellow atrophy, 177; in hæmorrhagic infarct, 140; in infectious jaundice, 213; in laryngeal croup, 71; in liver abscess, 190; in myocarditis, 49.
in nephritis, acute, 353; symptomatic, 370; tuberculous, 375.
in pericarditis, 41.
in peritonitis, 408, 410.
in perityphlitis, 312.
in pleurisy, 157.
in pneumonia, 123, 127.
in pulmonary abscess, 142; œdema, 119; tuberculosis, 133, 134, 135.
in splenic abscess and infarct, 230.
in uræmia, 361.
Fibre-column systems of the spinal cord, 514, 515.
affections of the, 536.
course of the longitudinal and their collaterals, 515, 516; and their motor, 518; and their sensory, 515, 520.
Fibrin coagula in the sputum of asthmatics, 105.

Fibromata of the larynx, 79.
papillary, of the stomach, 286; of the urinary bladder, 399.
Finger, hypertrophy of the, in syringomyelia, 572.
Fistula formation in the rectum due to disintegration of tuberculous ulcers, 139.
throat in laryngeal perichondritis, 73.
Fistular murmur, inspiratory metallic, in valvular pneumothorax, 167.
Flat-foot position due to acute anterior poliomyelitis, 560.
Fleece in the cerebellum, 619.
Flint's murmur in aortic insufficiency, 21.
Flukes, in liver (see Distoma Hepaticum).
Focal affections of the brain, 644.
diagnosis of, 664; typical of foci of the central convolution of the paracentral lobe, 649; of the centrum ovale, 661; of the frontal cortex, 651; of the internal capsule, 645, 646; of the nucleus lentiformis and nucleus caudates, 645; of the occipital cortex, 649; of the optic thalamus, 644; of the temporal cortex, 651.
latent, 663.
of the medulla oblongata and pons, 606, 608, 609; of the spinal cord, multiple (brain), 588.
Focal phenomena of the brain in uræmia, 359.
Follicular ulcers of the intestine, diagnosis of, 325.
Food stuffs, transformation in the body, 817, 818.
Foot, positions of, anomalous in hereditary ataxia, 550.
in peroneus and tibialis paralysis, 494, 495.
in poliomyelitis anterior, 560.
in tetany, 749.
Foramen Magendii, anatomical position and function of, 597.
Foramen Monroi, anatomical position and function of, 634.
Foramen ovale, patulous, 36.
Foreign bodies in the larynx, 80; in the œsophagus, 250; in the stomach, 265; in the trachea and bronchi, 98.
Fornix, position and distribution, 635.
Fossa Sylvii, anatomical position in the cerebrum, 634, 637.
Fractures, spontaneous, in tabes dorsalis, 546.
Frenum, ulcers of, in pertussis, 948.
Frémissement cataire, palpable, at the apex of the heart in mitral insufficiency, 12; stenosis, 16.
Friction sounds, differential diagnosis of, 44, 154.
in pericarditis, 40.
in pleurisy, 154, 157.
perihepatitic, 205.
peritoneal, in cholelithiasis, 215.
Friedreich's change of note over pulmonary cavities, 136.
Friedreich's disease, 549.
cerebellar form of, 550; differentiation from other cerebellar affections, 551.
character of, 550.
differential diagnosis, 551.
hereditary character of, 549.
relation to tabes dorsalis, 549.
spinal-cord degeneration in, 550; form of, 550.
symptoms of, 549, 550.
Frontal cortex, foci of the, disturbances of speech in, 651, 659, 660, 693.
thinking capacity in, 651.
Frontal temporal pontine tract fibres, anatomical course of, 602, 636.
Functional paralyses, 724.
Fungi in the mouth, 235, 238.
Furuncles, formation of, in diabetes mellitus, 829.
in typhus, 898.

G

Gait, ataxic, 549, 550.
heel, of tabetics, 539.
spastic-paretics, in amyotrophic lateral sclerosis, 555; in spastic spinal paralysis, 553.
wobbling, in adipositas universalis, 839.
Gall-bladder carcinoma, 218.
conduct of the, in cholelithiasis, 217, 218; in jaundice, 209.
differentiation from carcinoma of the liver, 202; of the stomach, 284; tumour and floating kidney, differentiation between, 392.
differentiation from hepatic tumours, 218.
dropsy of, 217.
Gall-stone colic, 214.
and gastralgia, differentiation, 215, 266, 298.
and gastric ulcer, differentiation, 279.
differential diagnosis from intercostal neuralgias, 455.
Gall-stones, jaundice caused by, 210, 214, 215.
pylephlebitis suppurativa, caused by, 220.
Ganglion cells of the brain of the cortex, 637, 638.
spinal cord, 515; atrophy of, in amyotrophic lateral sclerosis, 555.
tegmentum of the cerebral peduncles, 636.
Gangrene of the cheeks, 237.
symmetric, of nervous character, 756.
spontaneous, of the extremities in arteriosclerosis, 64.
Gangrene of the skin in diabetes mellitus, 829.
in erysipelas, 892.
in hysteria, 730.
in neuritis, 504; in multiplex, 508.
in typhoid fever, 918.
Gas pressure in the pleural cavity in pneumothorax, 166.
Gastralgia, 297.
differential diagnosis from cholelithiasis, 215, 298; causes of, 298; from gastric ulcer, 278, 298; from intercostal neuralgia, 278, 298; from intestinal colic, 298; from pleuritic pains, 158.
pathological picture, 297; due to cicatrization in the stomach, 275; in peritonitis, 410.
Gastralgia and intestinal neuralgia, 455.
Gastrectasis, 287.
determination of the capacity of the, 289.
due to continuous juice secretion, 302.
Gastric crises of tabetics, 538, 546.
Gastric fever, differential diagnosis from typhoid, 928.
Gastric glands, function of the depression of the, 303.
Gastric juice, condition of, in gastric catarrh, 264, 265.
constituents of normal, 256.
deficient or reduced, 303.
excessive, 302, 303.
formation of, in gastric ulcer, 276.
in acid gastric catarrh, 269.
secretion of,
Gastritis, 264.
acute, 264.
chronic, 268.
mycotica, 267.
phlegmonous, 266.
purulent, 266.
toxic, 267.
Gastritis in erysipelas, 894; in scarlatina, 876.
febrile and typhoid fever, 928.
Gastrodynia, 297.
Gastro-enteritis, febrile (infectious), diagnosis of, 928, 929.
Gastro-enteritis infantum, acute, 309.
Gastroptosis, diagnosis, 292.
differentiation from megalogastria, 291.
Gastrosuccorrhœa, continuous, 302, 303.
periodic, 301.
Gastrosuccorrhœa, differentiation from continuous, 302.

Gastrosuccorrhœa, differentiation from intermittent, 301.
from nervous, 301.
from hyperchlorhydria, 300.
Gastroxynsis, 302.
Gemeingefühle (subjective sensory anæsthesia), abnormal irritability in the region of the same in anæmic conditions, 782.
anæsthesia in, 437, 445.
Genital neuralgias, diagnosis of, 462.
Genitalia, diphtheria of the, 943.
erysipelas of the female, 894.
Genu recurvatum, due to poliomyelitis anterior acuta, 560.
Giant growth (see Acromegaly).
Girdle sensation in compression of the spinal marrow, 580.
in tabes dorsalis, 538.
in unilateral lesion of the spinal marrow, 583.
Glanders, bacilli of, 997.
invasion of the human body by, 998.
Glanders in man, 997; diagnosis of, 998.
by bacteriological examination, 998.
differentiation, from tuberculous and syphilitic ulcers, 998; from variola, 888.
symptoms, 998.
transmission of, from animal to man, 998.
Glanders, nodes and ulcers of, 998.
general infection due to, 998.
Glands, hypertrophy of the, in chronic laryngitis, 70.
Glands, swelling of the metastatic, in mediastinal tumours, 150.
Glaucoma due to circulatory disturbances in gout, 846.
Glia cells in the neuroglia of the spinal marrow, 515.
Glia proliferation in paralysis agitans, 751.
in spinal-cord disease, 536, 550, 589.
Gliomata of the brain substance, 691.
Gliosis spinalis, 571 (see also Syringomyelia).
Globus hystericus, 726.
Glomerulo-nephritis, 356.
Glossopharyngeal nerve, paralysis of, 479, 607.
taste perception of, 438, 439; in affection of, 440.
Glossopharyngo-laryngolabial paralysis, 612.
Glossoplegia, 481.
Glottis abductors, isolated spasm of the, 82.
paralysis of, 87.
Glottis adductors, paralysis of, 85.
Glottis œdema, 72.
and submucous laryngitis, differentiation, 69.
in angina, 241.
in chronic, 72.
in cryptogenetic, 72.
Glottis, œdema of, in erysipelas of the pharyngeal mucous membrane, 894.
Glottis, spasm of, 82, 83.
and bronchial asthma, differentiation, 105.
in tetanus, 990.
in whooping-cough, 947.
suffocation symptoms in, 78, 83.
Glutæi nerves, paralysis of, 493; spasm of, 502.
Glycogenesis of the liver in diabetes mellitus, 835.
Glycosuria, alimentary and physiological, 825.
pathological alimentary, 825; in sciatica, 461; in neuroses, 735; (see also Melituria).
Goitre, cause of œsophageal stenosis, 249.
cause of tracheal stenosis, 97.
Goitre, exophthalmique, 759 (see Graves's Disease).
Goitre expiration, causal connection of, with tetany, 750.
Goitre in Graves's disease, 759, 764.
Gonagra, phenomena of, 844.
Gout, 844.
acute, 844.
attacks of, 844; duration of, 844.
Gout, blood condition in, 845.
chronic (atypical), 846.
deposit of uric acid in the tissues and joints in, 845, 846.
diagnosis of, 844; chronic, 846; differential, of articular gout, 848; visceral, 846.
localization of, 844.
nature of, 845.
neuralgias in, 461; regular, 847.
symptoms of, 844; on the part of the brain, 847; of the eyes, 846; of the internal organs, 846; of the vessels, 847.
typical, 844.
visceral, 846.
Gout, articular, diagnosis of, 846, 848.
cerebral manifestation of, 847.
Gout, renal, 847.
diagnosis of, 848.
primary, 848.
symptoms, 847, 848.
Gouty fingers due to Heberden's nodes, 845.
due to uric-acid excretions in such, 845.
Gouty nephritis, diagnostic critera, 847.
Gouty nodes (tophi), growth of, 845.
localization of, 844, 845.
Graefe's symptom, 760.
Granulation tumours, infectious origin of, 858.
Graphospasm, nature of, 502.
Gravel, renal, 387.
Graves's disease, 759.
articular affections due to,
cardinal symptoms, 579.
cause of, 762.
diagnosis, 759; differential, 763.
digestive disturbances in, 761.
diminution of the resistance of the skin to electric current, 760.
goitre, 759.
Graefe's symptom in, 760.
metabolism in, 761.
Mœbius's symptom in, 760.
nervous symptoms, 760.
paralytic symptoms, 762.
pulse in, 759.
relation of, to myxœdema, 759.
sensation of heat in, 761.
Stelwag's symptom, 760.
subsequent symptoms of, 761.
tremor in, 760.
Gravitation abscess of the vertebral column and renal abscess, 370.
cause of bronchiostenosis, 98.
Gray matter of the cavities, central position and structure in the middle brain, 628.
Grippe, 985 (see also Influenza).
Growth, anomalies of, in acromegaly, 757.
in rhachitis, 852.
in spinal infantile paralysis, 559.
Grüber-Widal reaction, diagnostic significance of, in enteric fever, 712, 920, 921.
Gummata of the brain, 690.
Gummata of the palate and pharynx, 244.
differentiation of, from carcinoma of the palate, 245.
in the head, 53.
in the larynx, 76.
in syphilis of the liver, 188.
Gums, hæmorrhages in anthrax, 996.
in hæmorrhagic diathesis, 808.
in scorbutic affection of, 808.
Gutta cadens, 137.
Gymnastics of resistance for diagnostic purposes in cardiac disease, 43.

H

Hæmatemesis in aneurysm of the hepatic artery, 220.
in cirrhosis of the liver, 181.
in gastric cancer, 280.
in gastric ulcer, 274.
upon rupture of œsophageal varices, 182.
Hæmatemesis in purpura variolosa, 886.
in scarlatina, 876.
in typhoid fever, 916.
in typhus, 899.

Hæmatoidin crystals in the sputum in pulmonary abscess, 142.
in pulmonary gangrene, 144.
Hæmatoma of the dura mater, 718.
ætiology, 718.
diagnosis of, 719.
diffusion of, into the subdural space, 718.
gradual formation of, 718.
location of, 718.
Hæmatomyelia, diagnosis of, 595.
localization of, 595.
manifestations of, 594.
Hæmaturia in carcinoma of the bladder, 399.
of the kidney, 386.
of pneumonia, 124.
Hæmaturia, differentiation of, periodically occurring from paroxysmal hæmoglobinuria, 816.
in leucæmia, 795.
Hæmoglobinæmia, 811.
conditions of the blood in, 811.
diagnosis of ætiological, 813; differential, 816.
subsequent conditions of, 814.
urinary changes in, 812.
Hæmoglobinuria, 811.
absence of red blood corpuscles in the urine in, 812.
causes of, 813; occasional, 813, 814.
diagnosis of, 813; differential, from periodically occurring hæmaturia, 816.
paroxysmal, 813.
spectroscopic picture of the urine in, 812.
Hæmoglobinuria, paroxysmal, nephritis in, 355.
Hæmopericardium, diagnostic signs of, 42.
Hæmophilia, 807.
condition of the blood in, 807.
congenital, 807.
origin of, 807.
pathognomonic factors of, 807.
renal, 807.
Hæmoptysis in purpura variolosa, 886.
in pneumonia, 126.
in fibrinous bronchitis, 95.
in pulmonary tuberculosis, 126, 132, 136.
Hæmorrhages, anæmic conditions in, 786.
in anthrax, 996.
in chlorosis, 786.
in Graves's disease, 761.
in hæmorrhagic diathesis, 807 (of the mucous membrane), 808.
in hysterics, 730.
in the brain, 664.
in the medulla oblongata, 610.
in the pons, 610.
in the spinal cord, 594.
in whooping-cough, 948.
into the cerebral ventricles, 663; into the meninges, 718.
Hæmorrhages in acute yellow atrophy of the liver, 177.
in cirrhosis of the liver, 181.
in contracted kidney, 363.
in hypertrophy of the heart, 56.
in Weil's disease, 213.
into the mediastinal space, 153.
Hæmorrhagic diathesis, 807.
ætiological diagnosis of, 809.
condition of the blood in, 807, 808, 810.
in dysentery, 929.
in leucæmia, 795.
in pseudoleucæmia, 802.
joint affections in, and their differentiation from rheumatic polyarthritis, 975.
nature of, 808, 809.
subsequent to scarlatina, 873.
symptoms of, 808, 809.
Hæmorrhagica cerebri, 664.
meningealis, 663.
spinalis, degenerative phenomena in, 594.
Hæmorrhoidal plexus, phenomena upon overfilling of the venous, and differentiation from tabes, 547.
Hæmorrhoids of the bladder, 400.
of the rectum in carcinoma, 329; in cirrhosis of the liver, 182.
Hæmothorax, diagnosis, 168.
differential diagnosis between, and pleurisy, 160.
Hallucinations before and after epileptic attacks, 739.
in rabies, 994.
Hand, muscles of the, paralysis of, in Klumpke's paralysis, 492; in medianus paralysis, 490; in radialis paralysis, 486; in ulnaris paralysis, 489.
Hand, muscles of the, spasms of the, 501.
Hand, position of the, in medianus paralysis, 490.
in pachymeningitis hypertrophica, 534.
in radialis paralysis, 486.
in syringomyelia, 572.
in tetany, 749.
in ulnaris paralysis, 489.
Hands, anæsthesia of the, 443.
hypertrophy and crippling of, in syringomyelia, 572.
Hay asthma, origin and diagnosis of, 106.
Headache, differential diagnosis from occipital neuralgia, 453, 463; from trigeminus neuralgia, 453.
due to infection, 463; due to intoxication, 463.
idiopathic, 463.
in anæmic conditions, 463, 681.
in cerebellar affections (back of the head), 624.
in cerebral abscess, 692; in cerebral tumour, 681.
in circulatory disturbances in the brain, 463.
in disease of the brain and meninges, 463.
in epidemic cerebro-spinal, 949.
in Graves's disease, 760.
in measles, 863.
in meningitis, 703.
in paralysis of the facialis, 475.
in pathological processes of the bones of the skull, 463.
in septicopyæmia, 965.
in small-pox, 381.
neurasthenic, 464.
of hysterics, 464.
reflex, 464.
symptomatic, 464.
Headache, frontal, 464.
Headache in acute nephritis, 354.
in congested kidney, 363.
Hearing, difficulty of, in facialis paralysis, 474.
in tabes dorsalis, 539, 545.
Hearing, disturbances of, in affections of the pons and medulla, 607.
in lesion of the temporal cortex, 651.
in paralysis of the facial nerve, 474.
Hearing, impression of, sense centre for the, 642, 651.
Hearing, sphere of, cortical, of the brain, 651.
Heart, activity of, in acute articular rheumatism, 971, 974.
in anæmia, 783, 784; in cholera, 933; in diabetes, 829; in diabetic coma, 832; in diphtheria, 944; in Graves's disease, 759; in influenza, 986; in meningitis spinalis, 530; in neuritic affection of the vagus, 506; in obesity, 840; in pons-oblongata affection, 608; in septicopyæmia, 965.
Heart and large vessels, defects of, 36.
Heart crises of tabetics, 539.
Heart defect cells in the sputum in mitral stenosis, 19.
Heart, defects of the valves of the, 11; combined, 35.
diagnostic decision in combination of systolic and diastolic murmurs, 25; in purely diastolic murmurs, 34; systolic, 34.
diagnosis of aortic insufficiency, 19; of aortic stenosis, 26; of mitral insufficiency, 12; of mitral stenosis, 15; of pulmonary insufficiency, 29; of pulmonary stenosis, 29; of tricuspid insufficiency, 30; of tricuspid stenosis, 33.

Heart defects, non-compensated and pulmonary emphysema, 115.
Heart, dilatation of the, in aortic insufficiency, 20; in aortic stenosis, 26.
in hypertrophy of the heart, 56.
in mitral stenosis, 16.
in myocarditis, 49.
in pulmonary atelectasis, 107; in pulmonary emphysema, 113.
Heart, disease of, 1.
consideration of compensation, 3; affections of the endocardium, 6; of the heart muscle, 47; of the pericardium, 39.
physical examination in, vii.
preliminary remarks, 1.
Heart, displacement of the, in mediastinal tumours, 149.
examination of the, vii.
malformation of, 36; in engorged kidney, 349.
Heart, energy of the, decrease of, in fatty degeneration of the heart, 51.
in hypostasis of the lungs, 108.
Heart, enlargement of, due to retraction of the lungs, differentiation from pericardial effusion, 43; weakness of the heart, differentiation from pericarditis, 42.
in pulmonary emphysema, 113.
Heart, fatty, from anomalies of metabolism, 52.
Heart, fatty degeneration (see Fatty Heart).
Heart, hypertrophy of the, due to traumatic neurosis, 735.
in acromegaly, 757.
in diabetes mellitus, 830.
Heart, hypertrophy of the, 53.
ætiological factors, 53, 54.
diagnostic signs of, of the left ventricle, 56; of the right ventricle, 56.
due to overexertion, 54.
fatty degeneration of the musculature of the, caused by, 54.
idiopathic, 53.
in aortic insufficiency, 20; in aortic stenosis, 26; in arteriosclerosis, 62; in bronchiectasis, 101; in contracted kidney, 358; in hydronephrosis, 382; in mitral stenosis, 16, 17; in nephritis, 55; acute, 353; chronic, 358; in pulmonary emphysema, 113; origin of, 3.
Heart, insufficiency of, acute, 49.
in atheroma of the aortic system, 63.
chronic, in fatty heart, 51, 52.
origin of, 3.
Heart murmurs, accidental, and their differentiation from endocardial (organic), 4, 8.
diagnostic differentiation in combination of systolic and diastolic, 35.
diastolic, in aortic insufficiency, 20, 21; in mitral stenosis, 17.
in atheroma of the aorta, 62.
in myocarditis, 8, 49, 50.
in pulmonary valve insufficiency, 29.
interrupted modified diastolic, 17.
pericarditic, 40.
presystolic, 17.
systolic (blowing) in mitral stenosis, 17; in aortic stenosis, 26; in mitral insufficiency, 13; in pulmonary stenosis, 29.
Heart muscle, deficient blood supply in stenocardiac attacks, 58; diseases of, 48.
Heart-muscle degeneration in typhoid fever, 917, 918.
Heart, neuroses of, 733, 735.
due to paralysis of vagus fibres, 480.
Heart, neuroses of, diagnostic signs, 57.
Heart, palpitation of the, nervous, 57.
origin of, 57.
Heart, rupture of, spontaneous, 53.
diagnosis, 53.
Heart sounds in aortic insufficiency, 20, 21.
in aortic stenosis, 26; in fatty heart, 51; in hypertrophy of the heart, 56; in mitral insufficiency, 13; in mitral stenosis, 16, 17; in myocarditis, 49; in pneumopericardium, 47; in pulmonary emphysema, 113; muffled and split in diseases of the heart, 5.
Heart sounds, resonance of, in the stomach, 47.
Heart, spasm of the, overfilling of the lungs with blood in, 59, 120.
Heart, stenosis of, due to calcareous deposits, 51.
Heart, vagus centre, anatomical position, 605.
partial paralysis, 479.
Heart, valvular defects of, due to rheumatic endocarditis, 974.
Heat exhaustion, 771.
Heel-foot position in paralysis of muscles supplied by the tibial nerve, 495.
Helminthiasis, diagnosis of, 345.
Hemianæsthesia after apoplexy, 667, 672, 678.
contralateral, in parietal cortical areas, 649.
in brain tumours, 684.
in capsular diseases, 646, 647.
in focal affections of the centrum ovale, 662.
in hysteria, 729.
in pons-oblongata disease, 606, 609.
in traumatic neurosis, 734.
Hemianopsia in cerebral abscess in the occipital lobe, 693.
in lesion of the optic thalamus (contralateral homonymous), 644.
Hemiathetosis in acute infantile encephalitis, 696.
affections of the internal capsule, 646.
symptoms of, 746.
Hemiatrophia progressiva facialis, 754.
differentiation from congenital facial asymmetry, 754.
Hemichorea, 745.
in acute infantile encephalitis, 696.
in capsular affections, 646.
in lesion of the optic thalamus, 644.
Hemicontractures, irritative, due to brain tumours, 683.
Hemihypertrophia progressiva facialis, 754.
Hemiopia, contralateral homonymous, in cerebral hæmorrhages, 668.
in lesions of the occipital cortex, 649.
homonymous in capsular affections, 647.
Hemiplegia, alternating, 519.
following apoplectic insult, 667; contralateral, 667, 668, 670; diphtheria, 944; typhoid fever, 918.
hysteric, 724; differentiation from apoplectic, 675.
in aphasia, 659.
in Brown-Séquard's paralysis, 582.
in cerebral abscess, 693; in cerebral tumour, 684.
in encephalomalacia, 678, 679, 680.
in focal affection of the internal capsule (contralateral), 645, 669; of the pons oblongata, 606, 607, 610.
in Graves's disease, 762.
in meningitis, 705, 950.
spinal, 559; in tabes dorsalis, 545.
topical diagnosis of motor, 519, 670.
with eye-muscle paralysis, 466.
with paralysis of the facialis, localization of the focus, 475; of the radialis (cerebral), 487.
Hemiplegia, crossed, 467.
Hemiplegia cruciata, 608; spastica infantilis, 696.
Hemitetanus, 991.
Hepatic artery, aneurysm of, 211.
Hepatitis, interstitial, atrophic, 180; syphilitic, 184, 188.
suppurative, 189.
the consequence of gall-stones, 192.
Heredo-ataxia, 549; cerebellar, 550.
pathological picture, 549, 550.
Hernia, diaphragmatic, diagnosis of, 164.
Herniæ due to paroxysms of cough in whooping-cough, 948.
Herpes, in compression of the spinal cord, 580; facialis, differentiation from varicella, 890; in acute miliary tuberculosis,

957; in cryptogenetic septicopyæmia, 966; in facialis paralysis, 471; in influenza, 986; in malaria, 980; in meningitis, 704; in cerebro-spinal, epidemic, 950, 953; in myelitis, 575; in neuralgia, 447; cervical, 454; in neuritis, 504; multiple, 508; in pachymeningitis hypertrophica, 534; in tabes dorsalis, 545; in tetany, 749; zoster in intercostal neuralgia, 455.
Herpes labialis, in fibrinous pneumonia, 124.
laryngeal, 73.
Hiccough, clonic spasm of the diaphragm, 500.
History, taking of the, vii.
Hodgkin's disease, relation of, to leucæmia, 802.
Horse-shoe kidney, diagnosis, 391.
Hunger, metabolism during, 821.
Hunger and satiation, disturbances of sensations of, 299.
Hydatid thrill in echinococcus of the liver, 204.
Hydræmia and pulmonary œdema, 120.
Hydrarthrus nervosus intermittens, 756.
supersecretion, 269.
supersecretion, nervous, 294, 295.
supersecretion in ulcer, 276, 277.
Hydrocephaloid and meningitis, 713.
Hydrocephaloid in intestinal catarrh of children, diagnosis, 309.
Hydrocephalus, 719.
angeioneurotic, 721.
congenital, 720.
differential diagnosis of, from rhachitis of the skull bones, 720, 854; chronic, from brain tumour, 721.
following meningitis, 706, 720, 951.
idiopathic, chronic, 720.
internus et externus (intermeningealis), 719.
in tumours of the cerebellum, 625; of the posterior cranial fossa, 681, 721.
symptoms of, 720, 721.
Hydrochloric acid, in gastric juice, action of free, 256.
absence of, in anæmia of the stomach, 264.
in chronic gastric catarrh, 269, 270.
in gastric carcinoma, 281.
qualitative and quantitative determination, 261, 262.
Hydromyelus, nature and clinical symptoms, 571.
Hydronephrosis, 381.
ætiological reasons for the diagnosis of, 384.
contents of the hydronephrotic sac, 382.
differential diagnosis, 382; from ascites, 383, 424; from ovarian cyst, 382; from renal abscess, 383; from renal cysts and echinococcus, 383; from sacculated peritoneal exudate, 383.
in renal tuberculosis, 376.
intermittent, 381.
origin of, 384.
symptoms of, 381.
Hydropericardium and pericarditis, differential diagnosis, 42.
in heart diseases, 1.
in pulmonary emphysema, 115.
Hydrophobia, diagnosis of, 993.
hysterical, 726.
Hydrothorax, 161.
and pleurisy, differentiation of, 161.
in disease of the heart, 1.
in pulmonary emphysema, 115.
right-sided, in cirrhosis of the liver, 182.
Hypæsthesia, 436.
in poliomyelitis posterior, 571.
Hypalbuminosis in anæmics, 777.
Hyperalgesia, 436.
in Brown-Séquard paralysis, 583.
in neuralgias, 446.
Hyperæmia of liver, 192, 193.
of kidney, 348.
of spleen, 228.
Hyperæmia of the brain, 699.
of the spinal cord, 594.
Hyperæsthesia, 436.
diagnosis of, 445.
in compression of the spinal cord, 583.
in conus disease, 586.
in hysteria, 728.
in meningitis, 703, 704; epidemic cerebro-spinal, 949; spinalis, 530, 533.
in neuralgia, 445.
in neuritis, 503; multiplex, 506.
in pachymeningitis hypertrophic, 534.
in trichinosis, 999.
in unilateral lesion of the spinal cord, 583.
of the facial skin in trigeminus neuralgia, 451; of the nerves of taste and smell, 445; of the nerves of the skin, from peripheral causes, 445.
Hyperæsthesia of the abdominal wall, 344.
in peritonitis, 410; mucous membrane of the bladder, 403; of the larynx, 81; of the stomach, 299.
Hyperchlorhydria, nervous symptoms and diagnosis, 300, 301.
Hyperdiæmorrhysis, cerebral, 699, 701, 702.
Hyperemesis hysterica, 726.
Hyperextension of the toes in hereditary ataxia, 550.
Hyperhidrosis in acute articular rheumatism, 974; in acute miliary tuberculosis, 957.
in Graves's disease, 761.
in intermittent fever, 979.
in neuritis, 508.
in polymyositis acuta, 767.
in rhachitis, 853.
in syringomyelia, 583.
in tabes dorsalis, 545.
Hyperkinesis of the detrusor vesicæ, 402; of the stomach, 304; of the sphincter vesicæ, 402.
Hypertrophy of the extremities in acromegaly, 757.
Hyperorexia, diagnosis, 299.
Hyperplasia, connective tissue of the liver, 185; of the spleen, 228; of the tonsils and uvula, 241.
Hypersecretion jaundice, origin of, 176.
Hyperthyreoidism, ætiological significance of, in exophthalmic goitre, 763.
Hypertrophy of the heart in acute nephritis, 353; of the mucous membrane of the larynx in chronic catarrh, 70.
Hypoglossal nerve, paralysis of, 481.
bilateral, 481, 482.
central, 481.
cortical, 482.
due to apoplexy, 667, 668, 669.
in brain tumour, 685.
in lesion of the cortex of the central convolution, 481, 650; of the internal capsule, 482, 645; of the medulla oblongata, 608; of the nucleus of the hypoglossal nerve, 482; of the trunk of the hypoglossal nerve, 483.
peripheral, 481, 483.
symptoms of, 481; in lesion of the nucleus, 482.
unilateral, 481, 482.
spasms of the, 498.
Hypophrenic abscess, differentiation from liver abscess, 191.
Hypophysis cerebri, hyperplasia of the, in acromegaly, 757.
Hypotonia of the muscles of tabetics, 544.
Hysteria, 722.
ætiological factors of, 724.
arc de circle in, 726.
condition of the centres of special senses and association tracts in, 723.
determination of the conception of, 722.
diagnosis of, 722; differential, 731; from epilepsy, 727, 742; from hypochondriacal condition, 731; from meningitis, 532, 711; from multiple sclerosis, 593; from tetany, 750.
flexibilitas cerea in, 726.
in anæmics, 782.
in Graves's disease, 762.
neuralgic pains in, 447, 450, 460.

Hysteria, paralytic symptoms in, 724, 725.
psychical conduct in, 730.
rapid occurrence of, 723.
relation of, to paramyoclonus multiplex, 748; to psychoses, 722.
spasmodic attacks, 726, 727; their cause, 723.
suggestion in, 727, 730.
symptoms of, 724; cataleptic, 726.
transfer in, 729.
varieties of, 731, 734.
vascular innervation in, 729.
Hystero-epilepsy and epilepsy, 727.
Hysterogenous zones, production of spasm by pressure upon, 727.

I

Icterus liver and cancer of the liver, differentiation, 199.
and hypertrophic cirrhosis, differentiation, 199.
Ileo-cæcal murmur in enteric fever, 911.
Ileo-typhoid (see Enteric Fever).
Ileum, acute catarrh of, 310.
occlusion of, 335.
Ileus, causes and manifestations of, 337.
due to enterostenosis, 331.
nervous paralyticus, 337.
spasticus, 337.
Iliac fossa, right, inflammations of, 311, 312, 313, 314.
Immunization and immunity against infectious diseases, factors of, 859, 860.
Impaction symptoms in floating kidney, 393.
Impotence of diabetics, 831.
of neurasthenics, 733.
of tabetics, 538.
Inacidity in nervous dyspepsia, 300.
Inactivity, atrophy of, in myelitis, 575.
Incontinence of the pylorus, 307.
Incontinentia alvi in myelitis, 576.
urinæ in meningitis, epidemic cerebro-spinal, 950; in neuritis multiplex, 508; in spinal, 530.
vesicæ et alvi in lesion of the cerebral peduncle, 630.
Indican secretion, diminished in pancreas affections, 222, 224.
increased in enterostenosis, 331; in peritonitis, 410.
Infarcts, hæmorrhagic, of the heart, due to endocarditis, 10.
multiple, 10; of the lungs (see Pulmonary Infarct); kidneys (see Renal Infarct).
Infection, causing anterior chronic poliomyelitis, 562.
protective measures of the body against, 859.
septic, due to the entrance of bacteria into the circulation, 963.
Infectious diseases, 856.
classification according to localization of the infection, 858.
diagnosis of, 857, 862.
due to invasion of micro-organisms, 856.
ecchymoses of skin in, 810.
effects of the bacteria in the various, 858.
hæmoglobinæmia due to, 813.
immunization against, 858.
incubation period in, 858.
leucocytosis due to, 800.
meningitis due to, 710.
multiple encephalitic foci due to, 696.
muscular affections following, 766, 770.
nature of, 856.
nervous diseases (peripheral), due to, 434; due to neuralgia, 449; due to neuritis multiplex, 505.
occurrence of, in endemics and epidemics, 856.
proof of specific pathogenic bacteria in the various forms of, 857, 858.
relation of, to ascending paralysis, 588.
tetany due to, 750.
Infiltration, chronic, of the intestinal wall, differentiation from intestinal cancer, 329.
hæmorrhagic, of the pancreas, 223.
of the lungs in catarrhal pneumonia, 127; in fibrinous pneumonia, 122, 123.
tuberculous, 135; differentiation from croupous, 126.
Inflammation symptoms, differentiation from neuralgias, 447.
Influenza, 985.
catarrhal, 985.
complications of, 574, 987.
contagiosity of, 985.
diagnosis of, 985, 987; differentiation from bronchitis, 988; from measles, 870, 988; from miliary tuberculosis, 988; from typhoid fever, 988.
gastro-intestinal, 985.
microbes of, production, 985.
nervous, 985.
prodromal phenomena, 985.
sequelæ, 987.
specific generator of, 985.
symptoms of, 985; general, 985; nervous, 987.
Inhalation of dust in bronchitis, 91; in catarrhal pneumonia, 126.
Inhibition nerves of the heart, 605.
centre of,
Insanity, post-epileptic, 739.
Inspection in examination of the patients, vii.
of the abdomen in enterostenosis, 330.
of the chest in pericarditis, 40; in pneumonia, 123; in pulmonary emphysema, 111; in valvular defects of the aorta, 19; of the mitral valve, 12, 16.
of the gastric region in gastrectasis, 288.
Inspiration, forced, in pulmonary emphysema, 110.
Inspiration, centre for, 605.
sighing, in multiple sclerosis of the spinal cord, 590; in progressive bulbar paralysis, 614.
Insufficiency of the stomach, 290; of the cardia, 307; of the pylorus, 307.
of the valves of the heart, 11.
relative, 11; of the aorta, 19; of the mitral valve, 12; of the pulmonary valves, 29; of the tricuspid valve, 30.
Intelligence, disturbances of, in abscess of the brain, 692.
in myxœdema, 758.
Intention tremor in multiple cerebro-spinal sclerosis, 589, 592, 753; in multiple myelitis, 588.
Interarytænoid fold, tuberculous ulcers of, 73.
Interarytænoid, transverse, paralysis of, 87.
Intercostal neuralgia, differential diagnosis from gastralgia, 298; from gastric ulcer, 278; from pleurisy, 158; in renal cancer, 386.
Intercostal neuralgia, 454.
ætiological factors, 455.
diagnosis of, 454; differential, 455, 765.
varieties of, 456.
Intercostal space, retraction of that which is situated at the locality of the apex beat upon systole of the ventricle, 45.
Intermittent fever (see Malaria).
Intermittent fever and gall-stone colic, differentiation, 216; liver abscess, differentiation, 191.
in croupous pneumonia, 124.
Interossei of the hand, atrophy of, in progressive muscular atrophy, 564.
paralysis of, 489.
Intestinal affections, causal connection with tetany, 750.
in Asiatic cholera, 934.
in leucæmia, 794.
in paralysis of the vagus, 480.
in scarlatina, 876.

Intestinal anthrax, 996.
diagnosis of, 996.
secondary, 996.
symptoms of, 996.
Intestinal cancer, 325.
and chronic infiltration of bowel wall, differentiation, 329.
determination of location, 329.
differential diagnosis, 327; between fæcal tumours, 327; between gastric carcinoma, 327; between movable spleen and kidney, 327; between ovarian tumours, 328; between renal tumours, 329; between sacculated peritoneal exudate, 328.
intestinal stenosis in, 325.
location, 329.
perforation of the gut, 326.
ribbon-shaped fæces in, 325.
symptoms, 326.
tumour in, 326.
Intestinal catarrh, acute, 308.
abnormal constituents of stools, 316.
and cholera nostras, 309.
atrophy of the intestinal mucous membrane, 318.
bile-pigment reaction in, 311.
chronic, 316.
constipation in, 316.
deficient digestion in, 311.
dejecta in, 308.
fever in, 309.
in cirrhosis of the liver, 181; in emphysema of the liver, 114.
of children, 308.
of the duodenum, 309; of the jejunum and ileum, 310.
of children, 318.
secondary symptoms of, 318.
small and upper, large bowel, combined, 310.
symptoms of value in diagnosis of, 308.
Intestinal catarrh in erysipelas, 893.
in measles, 867.
in typhoid fever, 913.
in typhus, 898.
of rhachitic children, 853.
Intestinal colic, 342.
and gastralgia, differentiation, 298.
enterospasm, 340.
stenosis, 332, 336.
Intestinal crises of tabetics, 538, 546.
Intestinal diphtheria, 315.
Intestinal diphtheria, dysenteric, 928.
Intestinal discharges (see Fæces).
Intestinal hæmorrhages in hepatic cirrhosis, 182; in ulcers, 320.
Intestinal hæmorrhages in typhoid fever, 915.
Intestinal invagination, 336.
hæmorrhagic-mucous diarrhœa in, 336.
Intestinal invagination, internal, 336.
Intestinal loops, 333.
Intestinal movements, centres for, 644.
Intestinal mucous membrane, atrophy of, in chronic catarrh, 318.
in chronic catarrh, 317.
in cloudy epithelia of the,
Intestinal nerves, disturbances of function of, 338; of motion, 339; of secretion, 342; of sensation, 345.
Intestinal neuroses, 733.
Intestinal occlusion, 330.
in perityphlitis, 332.
Intestinal paresis, 341, 342.
Intestinal perforations due to typhoid ulcers, 912, 915.
Intestinal peristalsis in intestinal catarrh, 310; in intestinal stenosis, 331, 332, 334.
decreased, 341.
increased, 305; upon a nervous basis, 339, 340.
Intestinal stenosis, 330.
by axial torsion, invagination, and loop, 336.
conduct of bowel portion in front of stenosis, 333.
Intestinal stenosis, consequences of, 337.
determination of, 331.
differential diagnosis, 332; from gall-stone, intestinal and renal-stone colic, peritonitis, poisoning, typhlitis, floating kidney, diaphragmatic hernia, 332.
due to enterospasm, 337; due to intussusception, 336.
examination of the abdomen, 333, 335.
fæces in, 331.
hernial rings, rectum and vagina, 335.
location, 333.
nature of obstacles causing, 335.
pseudoligaments, 337.
tumour in, 335.
urinary conditions in, 331.
Intestinal stenosis due to dysentery, 930.
Intestinal trichinæ, development of, in the human intestine, 999.
Intestinal tuberculosis, miliary, 957; in children, 323.
Intestinal tumours, differentiation from cancer of the liver, 203.
Intestinal ulcer, 319.
catarrhal, 325.
causes of, 322.
complicated with peritonitis, 321.
dysenteric, 324.
embolic, 323.
fæces in, 319, 320.
infections, 323.
peptic, 322.
symptoms, 317, 320, 321.
syphilitic, 324.
traumatic, 325.
tuberculous, 323; in pulmonary tuberculosis, 139.
typhoid, 323.
Intestinal worms, symptoms, 345.
Intestine, atony of, 341.
atrophy due to catarrh, 318, 319.
Intestine, axial torsion of the, 336.
Intestine, diagnosis of diseases of, 306.
of nervous (functional), 337.
Intestine, large, catarrh of the, diagnostic points in, 310, 317.
combined with catarrh of the small intestine, 309.
stools in chronic, 310.
varieties of, 316.
Intoxication, acute, nephritis as a result, 355.
and anæmia, 361.
and occlusion of the gut, 332.
Intoxication, cholæmic, in jaundice, 209.
and uræmia, differentiation, 360.
Intoxications, bacterial, of the body, 856, 857.
causing chorea, 746; hæmoglobinuria, 813; leucocytosis, 800; nervous diseases, peripheral, 434; neuralgia, 450; polyneuritis, 505; rheumatic, 972; septic, 964; tetany, 750.
Intussusception, ileocæcal, 336.
cause of enterostenosis, 337.
Inversion of cane-sugar in stomach, 257.
Iritis in diabetes, 831.
in gout, 846.
Irritation symptoms in compression of the medulla oblongata, 617; of the spinal cord, 580; due to tumours, 573, 585.
lesion of the optic thalamus, 644.
neuralgic, central, 448; peripheral, 448.
neuritis, 503, 506, 509.
Irritative conditions of the gastric motor nerves, 304.
Ischuria in hysteria, 730.
Ischuria paradoxa, 401.
spastica, 402.
Island of Reil, anatomical position in the cerebrum, 634.
lesion of, and symptoms, 661.
Isthmus aortæ, persistence of, 38.
collateral circulation in, 38.
systolic murmurs in, 38.

J

Jactitation in meningitis, 703, 704.
Jaundice, 175, 207, 208, 215.
acute febrile infectious, 213.
cause and nature of, 213 (see also Weil's Disease).
causes of occlusion of the bile ducts, 211.
conduct of the gall-bladder in, 209.
due to accumulation of fæces, 212; due to carcinoma of the duodenum or head of the pancreas, 211; due to catarrh of the bile ducts, 215; due to distoma hepaticum, 211; due to duodenal ulcer, syphilis of the liver, perihepatitis, 212; due to external compression of the bile ducts, 211; due to gall-stones, 210; due to parasites of the bile ducts, 211; due to tumours in the porta hepatis, 211; of the abdomen, 212.
effects of, upon the nervous system, 208.
forms of, 175; in acute yellow atrophy, 176; in aneurysm of the aorta, 212; of the hepatic artery, 220; in cancer, 198; in cholelithiasis, 210, 212, 214; in cirrhosis, 183, 184; in gastric catarrh, 265; in hyperæmia, 192; in hysteria, 179; in infectious diseases, 179; in liver abscess, 190; in syphilis, 188.
Jaundice, anomalies of metabolism in, 208.
Jaundice, in pancreas affections, 223; in pneumonia, 124, 190.
location of occlusion of the bile ducts, 209.
swelling of the liver in, 209.
symptoms of, 207.
the urine in, 208.
with urobilinuria, 174, 175.
yellow decoloration of the skin in, 207.
Jaundice in hæmoglobinæmia, 815; in neuritis, 508; in relapsing fever, 905.
septic, 967.
Jaundice, parasecretion, origin of, 176.
Jejunum, acute catarrh of, 310.
occlusion of the, 335.
Joint affections in erysipelas, 893.
in gonorrhœa, differentiation from acute rheumatism, 975.
in gout, 845; diagnosis of, 848.
in Graves's disease, 761.
in hæmophilia, 807.
in measles, 867.
in mumps, 940.
in polymyositis acuta, 767.
in scarlatina, 874.
in syringomyelia, 572.
in tabes dorsalis, 545.
in typhoid fever, 916.
inflammatory, in septicopyæmia, 966.
Joints, swellings of the, in acute articular rheumatism, 971.
in attacks of gout, 845.
in hæmorrhagic diathesis, 807.
in neuritis, 504; in multiple, 508.
Jugular valve sound in tricuspid insufficiency, 36.
Jugular veins, pulsation of, in diseases of the heart, 2.
malformations of the heart, 36.

K

Keratitis, neuroparalytic and parenchymatous, in diabetics, 831.
Kidney, abscess of, 367.
adenoma of, 385; differentiation of, 348; in cholera and pregnancy, 350; hypertrophy of the heart in, 54.
amyloid degeneration of, 364.
anomalies of form and position, 391.
cause of, 371.
congenital absence of one, 391.
diagnostic symptoms, 371.
differentiation from hydronephrosis, 371, 383; from paranephritis, 372; from suppurative pyelitis, 372.
fluctuation of, 370.
formation of concrement, 387.
Kidney, large red, 357; large white, 357.
movable, 391.
perforation into the renal pelvis, 367.
stasis hyperæmia of, 348.
tuberculosis of, 374.
Kidney, contracted, primary, 364.
arteriosclerotic, 364; arteriosclerotic, genuine (primary), 362, 364.
cardiac hypertrophy from, 363.
development of, 364.
diagnosis from cystitis, 395.
differential diagnosis, 368.
digestive disturbances, 363.
dropsy, 363.
hæmorrhages in, 363; hæmorrhages in brain, 363.
ophthalmoscopic findings in, 363.
uræmic symptoms in, 363.
urine in, 362.
Kidney, contracted, secondary, 361, 362.
anasarca in, 363.
differential diagnosis, 368.
symptoms, uræmic in, 361.
urine in, 361.
Kidney, contracted, with chronic uræmia, differential diagnosis from cerebral tumour, 687.
Kidney, fatty, 364.
amyloid degeneration of liver and spleen in, 366.
anæmia and cachexia in, 366.
anasarca and dropsy in, 366.
causes of, 366.
diagnosis of, 366.
diarrhœa in, 366.
urine in, 365.
Kidney, hyperæmia of, 348.
condition of urine in, 348, 349.
cyanosis in, 350.
differential diagnosis, 368.
dropsy in, 349.
from heart disease, 2.
heart and lungs in, 349.
liver in, 350.
physiology of, 348.
with nephritis, 350.
Kidneys, carcinoma of, 385.
consistence of the tumour in, 385.
differential diagnosis in, 386.
hæmaturia in, 386.
in children, 387.
secondary symptoms, 386.
urine in, 386.
Kidneys, functional disturbances of, in anæmia, 786.
toxic irritation of, in hæmoglobinuria, 815; in malaria, 980; in septicopyæmia, 967.
tuberculosis of, in acute miliary tuberculosis, 957.
Kidneys, hypertrophy of, 381; diffuse and partial, 381.
induration of, arteriosclerosis, 364; focal, 362; secondary, 362.
infarct of, hæmorrhagic, 375; in the course of acute endocarditis, 373.
Kidneys in pregnancy, 350.
Kidneys, sarcoma of, 387.
differentiation from renal cancer, 387.
sclerosis of (see Contracted Kidney).
stones of, 387; and stones of the bladder, 390; colic, 388; and cholelithiasis, 216; enteralgia, 343.
Kidneys, tumours of the, 376.
cystic, 380.
differential diagnosis of, from cancer of the gut, 328; from tumours of liver, 202, 203; from tumours of ovary, spleen, and retroperitoneal, 378, 379.
examination by palpation, 376; bimanual, 377.
influence of respiration upon motility of, 377.
jaundice due to, 212.
neuralgic pain of the belly wall in, 377.
position and extent, 376; to the colon, 376; to cancerous, 385.
solid, 385.

Klumpke's paralysis, diagnostic criteria, 492.
differentiation from Duchenne-Erb paralysis, 492.
Kyphosis in hereditary ataxia, infantile spinal paralysis, 560.
osteomalacia, 850.
rhachitis, 853; differentiation from tuberculosis, 853.

L

Labferment of the gastric juice, 256.
effect of, in digestion, 257.
Labyrinth, disease of, in mumps, 940.
Lactic acid in the stomach contents, 262; in the urine in acute yellow atrophy of the liver, 177.
increase of, in gastric cancer, 282.
Lactosuria in pregnancy and lactation, 826.
differentiation from diabetes, 837.
Lagophthalmos in facial paralysis, 468, 472, 477.
Landry's paralysis, 586.
ætiological factors of, 588.
clinical observations of, 587.
diagnostic important symptoms of, 586.
differentiation of, from acute myelitis, 578; from multiple neuritis, 510.
electric irritability of the paralyzed muscles in, 587.
infectious nature of, 588.
prodromes of, 586.
reflexes in, 587.
Laryngeal cancer, 79.
catarrh, acute, 67; chronic, 70; in small children, 69.
condylomata, 75.
croup, 70; and pseudocroup, differentiation, 71.
fever in, 71.
stenosis of the larynx in, 71.
Laryngeal erysipelas, primary, 72.
Laryngeal muscles, paralyses of, 83; ætiological diagnosis, 88; examination upon determining same, 89.
spasm of, 82.
Laryngeal neoplasm, 78.
benign, 79.
differential diagnosis, 80.
malign, 72.
Laryngeal nerve, inferior, paralysis of, 85.
recurrent, paralysis of, 83, 84.
superior, motor paralysis of, 83.
Laryngeal nerves, paralyses, 83.
Laryngeal neuroses, diagnostic phenomena of, 81.
motor, 82.
sensory, 81.
Laryngeal stenosis, 77.
acute suffocation in, 78.
and bronchial stenosis, differentiation of, 96.
consequences of, 78; in laryngeal croup, 71; in perichondritis, 72.
differential diagnosis of, 78.
dyspnœa, expiratory and inspiratory in, 78.
Laryngeal ulcers, 73.
catarrhal inflammatory, 73.
gummatous, 76.
infectious, 74.
pressure, 74.
syphilitic, 75.
tuberculous, diagnosis, 75; origin, appearance, seat, 74.
typhoid, 76.
Laryngismus stridulus, 82.
Laryngitis, acute, 68; "sicca," 69; submucosa, 69.
atrophy and hypertrophy of the mucous membrane in chronic, 70.
chronic, 70; sicca, 70; submucosa, 70, 72.
differential diagnosis of, from glottis œdema and perichondritis, 69; from pseudocroup, 69.
diphtheria and, 70.
forms of, 68.
in enteric fever, 75.
syphilitic, 75.
Laryngitis, croupous, 941, 942.
diphtheritic, 941, 942, 943.
erysipelatous, 894.
in influenza, 986; in typhus fever, 898.
Laryngo crises of tabetics, 538.
Laryngoscopical findings in aortic aneurysm, 65.
laryngeal catarrh, acute, 68, 69; chronic, 70; perichondritis, 72.
laryngeal diphtheria, 71; syphilitica, 75; tuberculosa, 74, 75.
mediastinal tumours, 146.
paralysis of the recurrent laryngeal nerve, 84, 85.
Laryngospasm, 82.
acute suffocation in, 82.
differential diagnosis of, 82.
functional, phonic and respiratory, 83.
Laryngospasm of rhachitic children, 853.
Larynx, diagnosis of cicatrices in, 79; diseases of, 68; foreign bodies in, 78; neoplasms in, 78.
downward movement in aortic aneurysm, 65.
Larynx, croup of the, 941; epidemic, 942.
diphtheria of the, 943; ascending, 943; complication of, 944; conduct of the lungs in, 943; descending, 942; differentiation from non-diphtheritic affections of the larynx, 945, 946; general symptoms, 943; glottis, stenosis in, 943; laryngoscopical picture in, 943; primary, 942; secondary, 942.
Larynx, muscles of the, atrophy and functional weakness in bulbar paralysis, 612.
paralysis of, 479; in diphtheria, 944; in trichinosis, 999.
spasm of, in tetanus, 990.
ulcers of the, 917.
Lateral sclerosis, amyotrophic, 554.
degeneration of both motor neurons in, 555.
diagnostically important symptoms, 555.
differential diagnosis of, 556; from chronic myelitis, 557; from multiple sclerosis, 557, 592; from pachymeningitis hypertrophica cervicalis, 534, 557; from poliomyelitis anterior, acute, 554, 566; chronic, 563, 566; progressive, 557, 566, 570; from spinal-cord tumours, 557; from syringomyelia, 557.
pathological condition in, 555.
propagation of the affection in, 555.
reaction of degeneration in, 556.
symptomatic, 557.
with bulbar symptoms, 556, 614.
Latissimus dorsi, paralysis of, 484.
Laughing, movements of, from facial paralysis, loss of, 473, 645.
Lavage of the stomach, 259.
Lead-poisoning the cause of paralysis of the radial nerve, 487; of poliomyelitis anterior chronica, 562.
Leg, musculature of, neuralgias of the, 456, 457; cortical lesion of the central convolutions, 649, 650; crutalis paralysis, 493; glutæi lesions, 493; obturatorius lesion, 493; paralysis of the peroneus tibialis, 494.
Lemniscus, decussation of, in the medulla oblongata, 520, 597.
in the middle brain, 627, 635.
Leprosy, 1005.
ætiology of, 1006.
clinical picture of, 1006.
diagnosis of, 1007.
pathology of, 1006.
smooth variety of, 1006.
Leptothrix in the sputum in pulmonary gangrene, 144.
buccalis and its symptoms, 238.
Leucæmia, 790.
ætiology of, 798.
anæmia in, 794.
and pseudoleucæmia, 803.
blood condition in, 790; Charcot's crystals in, 792; hæmoglobin in, 790; leucocytes in, 791; red blood corpuscles in, 790.
differential diagnostic character of the different forms, 796.

Leucæmia, genesis of, 796.
hæmorrhagic diathesis of the leucæmias, 795.
leucocyte (myelogenous leucæmia), 797.
lymphocyte (lymphatic), 796; acute and chronic, 797.
mixed forms of, 791.
nature of, 798.
symptoms of, 793, 794, 795.
Leucæmia, intestinal tumours in, 325.
Leucanæmia, 803.
and anæmic pseudoleucæmia infantum, 790, 803, 804, 805.
clinical observation of, 803.
Leucin in the urine in acute yellow atrophy, 177.
Leucocytosis, 800.
active, 800.
changes in the blood in, 800, 801.
eosinophile, 800, 801.
in anæmia, 780.
occurrence of, 800.
passive, 800.
pathological, 800.
physiological, 800.
polymorphonuclear, 800.
relation of, to leucæmia, 801.
symptomatic, occurrence of, 800.
Leucocytosis in connective-tissue hyperplasia of the liver, 186; acute, in fibrinous pneumonia, 125.
Leucomyelitis posterior chronica, 537. (see also Tabes Dorsalis).
Leucopenia, occurrence of, 801.
Levator anguli oris, paralysis of, 472.
Levator anguli scapulæ, spasm of, 500.
Lid muscles, paralysis of, in lesion of the facialis, 472.
Lids, spasm of, 496.
Lien mobilis, 233 (see also Spleen, Floating).
Lientery, diagnostic signs of, 311.
Lingualis, neuralgia of, 452; perception of taste of, 438.
Lips, muscles of, paralysis of the facial, 472; in progressive bulbar paralysis, 612.
Little's disease, 554.
Liver abscess, 189; abdominal abscess, 191; cholelithiasis, 192; hypophrenic abscess, 191; intermittent fever, 191; pleuritic exudate, 191; purulent echinococcus sac, 192, 205; pylephlebitis, suppurative, 220; consistence of the liver in, 189.
fever in, 190.
pain in, 189.
surface of the liver in, 189.
volume of the liver in, 189.
Liver, abscess of, differentiation of fever attacks of, from intermittent fever, 983.
in dysentery, 930.
in septic infection, 967.
in typhoid fever, 917.
Liver, amyloid degeneration of, 195.
anatomical conditions of, 170.
changes of form and position of, 206; in exudative pleurisy, 137.
diagnosis of diseases of, 170.
diagram of diseases of, 221.
examination of,
function of, 171, 172.
surface of, in amyloid degeneration, 195; in syphilis of the liver, 197.
Liver, atrophic, Laennec's, 180; secondary, 187.
Liver atrophy, acute yellow, 176.
and dilatation of the transverse colon, 179.
and jaundice in hysteria, 179; in infectious diseases, 179.
and changes of the blood in, 180; of the urine in, 177.
clinical picture of a case of, 178.
diagnosis of the various stages, 178.
initial stage of, 176.
nervous symptoms in, 176.
phosphorus poisoning in, 176, 177.
reduction in volume of the liver, 177, 178.
simple chronic, 180; marantic, 180.
stage of the full development, 176.
Liver atrophy, termination of, 176.
acute yellow, following enteric fever, 917.
Liver, borders of, percussory in amyloid liver, 186.
atrophy of the liver, 176, 178.
cancer of the liver, 197.
cirrhosis of the liver, 180, 185.
corset-lobe liver, 206.
echinococcus of the liver, 204.
fatty liver, 194.
pulmonary emphysema, 112.
syphilis of the liver, 187.
fatty degeneration of, 194.
Liver, cancer of, 196; and abdominal wall tumours, 204; and echinococcus multilocular, 199; and fatty liver, liver abscess, echinococcus simplex, amyloid liver, 198, 199; and gall-bladder carcinoma, 201; and gastric cancer, 201; and hypertrophic cirrhosis, 186, 199; and icterus liver, 199; and sarcoma of the liver, 200; and syphilis of the liver, 188; and tumours of the kidney, 202.
combination with rectal carcinoma, 200.
consistency and surface of the liver in, 197.
primary and secondary, 200, 220.
result of palpation and percussion, 197.
special forms of, 201.
subsequent symptoms of, 198.
volume of the liver in, 197.
Liver, carcinoma of, diagnostic symptoms, 145.
secondary in renal carcinoma, 386.
Liver cells, anatomical arrangement to the canal in the liver, 170.
Liver, cirrhosis of, of diabetics, 830.
Liver, cirrhosis of, caused by connective-tissue hyperplasia, 185.
enlargement of, in abscess of the liver, 189.
in jaundice, 209.
in phosphorus poisoning, 180.
in the initial stage of acute yellow atrophy, 176.
Liver, enlargement of, due to fat deposits in theobese, 841.
in cryptogenetic septicopyæmia, 967.
in hæmoglobinæmia, 814, 815.
in leucæmia, 794.
in malaria, 980.
in relapsing fever, 902.
in typhoid fever, 914.
Liver, hyperæmia of, 192.
causes of, 193.
changes in the size of the liver in, 193.
differential diagnosis from fatty liver, 194.
fluxionary, 193.
physical examination in, 192.
vicarious, 193.
Liver, connective-tissue hyperplasia of, 185.
condition of blood and urine in, 186.
differential diagnosis of, from amyloid liver, cancer of the liver, icterus liver, multilocular echinococcus of the liver, 186, 187.
enlarged liver in, 187.
mixed form with atrophic cirrhosis, 187.
sclerotic enlargement of the spleen, 187.
Liver, neuralgia of, differentiation from gall-stone colic, 216.
Liver, swelling of, cloudy, of the parenchyma, 195.
in gall-stone colic, 215.
in heart diseases, 1; in fatty heart, 51; in pericarditis, 41.
syphilis of, 187; amyloid degeneration due to, 195; and cancer of the liver, 188; and cirrhosis of the liver, 183; bile-duct occlusion due to cicatricial shrinking, 212; gummata in, 188; pains in the hepatic region, 188; perihepatitis in, 188; special changes of the liver in, 187.
tuberculosis of, miliary, 957.
tumours and pleuritic exudate, 160; and renal tumours, 377.

Liver, veins, pulse of, in tricuspid insufficiency, 31.
vessels, disease of, 207.
Localization, diagnosis of, of spinal-cord disease, 525.
according to the height of the segments, 528.
in lesions of the horns, 525, 526; and antero-lateral ground bundles, 526; cerebello-lateral column tracts, 527; nerve roots, 526; posterior columns, 526; posterior horns, 526; pyramidal tracts, 525.
sense, disturbance of, in tabes dorsalis, 545.
Locomotor ataxia, 537.
Longitudinal bundle, posterior, in the medulla oblongata, anatomical position and structure of, 603, 628.
Lumbago, symptoms of, 765.
Lumbar cord, affections and tumours of, as cause of sciatica, 461.
anæmia of, 594; myelitis of, 576; due to pressure, 579.
muscles, paralysis of, 492; spasms of, 502.
nerves, neuralgias in the region of, 456; paralysis in the region of, 492; spasms in the region of, 502.
puncture, diagnostical significance in meningitis, 532, 707, 710, 957.
Lumbo-abdominal neuralgia, diagnosis of, 456.
Lumbricales of the hand, atrophy of, 564; paralysis of, 490.
Lung, abnormally large, differentiation from pulmonary emphysema, 115.
abscess of, 141; and bronchiectasis, 101; and gangrene of the lung, 145; and phthisis with cavity formation, 142; ætiological factors, 141; cavities in the lung due to, 142; metastatic, due to embolism of the pulmonary artery, 141; sputum in, 142.
actinomycosis of, 146.
apices of, tuberculous catarrh, diagnosis of, 131, 132.
atelectasis of, 106; and capillary bronchitis, 93; and catarrhal pneumonia, 128; causes of, 107; circumscribed, 108; congenital, 107; differential diagnosis of, 107; stasis symptoms of, 107.
Lungs, atrophy of, in bronchiectasis, 101; in interstitial pneumonia, 128; in syphilis of the lungs, 145; in tuberculosis of the lungs, 134.
borders of the, percussory in, asthma, 60, 103; atrophy of the lungs, 129; emphysema of the lungs, 110, 112.
cavities of the, differential diagnosis of, in the neighbourhood of the heart from pneumopericardium, 48; differentiation of, from cavities of the lungs in abscess formation, 142; in pneumothorax, 164; percussory and auscultatory phenomena, 136, 137, 138; sputum in, 135.
Lungs, chronic tuberculosis of the, 130; predisposition, 133; hereditary, 133; in pulmonal stenosis, 29; sputum in, 135; tubercle bacilli in the sputum, 130; tuberculin reaction in, 131; first stage, 131; changes in the apices, 131; combined with pleurisy, 133; hæmorrhages in, 132; results of percussion and auscultation of the apices, 132; second stage, 134; fever in, 135; percussory conditions in, 134; phthisical form of thorax, 134; sputum in, 135; stasis phenomena, 135; third stage, 135; auscultatory phenomena, 137, 138; cavity symptoms, 136; changes of note, 136; differential diagnosis between cavities and pneumothorax, 138; results of percussion, 136; secondary symptoms, 138.
Lungs, collapse of the, 106.
Lungs, congestion of the, 108; and hæmorrhagic infarct, 109; combined with pulmonary œdema, 109; hypostatic pneumonia after, 109; origin of, 108; symptoms of, 109.
Lungs, echinococcus of the, diagnosis, 145.
Lungs, emphysema of the, 110; and bronchial asthma, 104; and inflation of the lungs, 114; and non-compensated heart defects, 115; and pneumothorax, 115; and pulsus excessions, 115; circulatory disturbances, 112; differential diagnosis, 114; dilatation and hypertrophy of the heart in, 113; interlobular and subpleural, 117; mediastinal, 117; pathological findings of the lung, 110; physical examination results, 111; respiratory changes in, 110; senile, 116; stasis symptoms in the greater circulation, 112; vicarious, 116, 134.
Lungs, gangrene of, 143; and bronchiectasis, 102, 144; and putrid bronchitis, 91, 144; diabetes mellitus in reference to, 145; differential diagnosis, 144; from pulmonary abscess, 143; sputum in, 143; symptoms of gangrene, obliteration of the pulmonary tissue, 144.
Lungs, hæmorrhage of the, in bronchiectasis, 103; in hypertrophy of the left ventricle, 56; in pulmonary tuberculosis, 132.
Lungs, hæmorrhagic infarct of the, 139; ætiological diagnosis, 139; differential diagnosis from congestion of the lungs, 109; from croupous pneumonia, 126; due to, embolism of the trunk and large branches of the pulmonary artery, 140; due to obstruction of smaller pulmonary artery branches, 140; in mitral insufficiency, 14.
Lungs, inflation of the, acute and pulmonary emphysema, 114; in bronchial asthma, 103; in capillary bronchitis, 93.
neoplasms of, 145.
Lungs, œdema of, 117; and pneumonia, 126; ætiological factors, 118; complicated by congestion of the lungs, 109; differential diagnosis of stasis œdema, 119; hydræmia, 120; in cardiac asthma, 60; inflammatory, 118; pathogenesis of, 118; stasis œdema, 119; symptoms of, 117.
retraction of, cardiac dulness in, 43.
syphilis of, clinical diagnosis, 145.
Lymphadenia, symptoms of, 803.
Lymphadenoma of the wall of the stomach, 286.
Lymphangeitis, differentiation from phlegmonous, 998.
in glanders infection, 998.
purulent in enteric fever, 916.
Lymphæmia, 791, 792.
acute, 797.
Lymph glands, enlargement of the bronchial as cause of bronchiostenosis, 97.
metastatic enlargement of, in diphtheria, 242; in mediastinal tumours, 153.
swelling of, in erysipelas, 893; in leucæmia, 793, 797; multiple, in pseudoleucæmia, 802, 803; in pharyngeal diphtheria (of the neck), 942.
tumours of, and gastric cancer, 285.
Lymphocytosis, character and occurrence of, 800, 801, 802.
Lymphoma, formation of, in the skin of leucæmics, 794.
Lyssophobia, origin of, 994.

M

Maladies des tics convulsifs, differentiation from paramyoclonus, 748.
Malaria and cholelithiasis, 216; splenic tumour in, 229.
cachexia, symptoms of, 984; differentiation from other forms of cachexia, 984.
cause and transmission, 978.
diagnosis of, 978; differentiation, 981; from fever attacks in cholelithiasis and liver abscess, 983; from septicopyæmia, 968, 981; from tuberculosis, 982.
fever forms, 978; stages, 979; chill, 979; heat, 979; sweat, 979.
incubation period, 978.

Malaria and cholelithiasis, infection with malarial parasites, 976, 977, 978.
irregular course of, 983.
masked, 983.
neuralgias in, supraorbital, 449, 983.
parasites, course of development of, 976; groups of, 977; transmission of, upon man, 978.
pernicious, 984.
prodromes of, 978.
remittent, 984.
symptoms of, 979, 980; nervous, 984.
time of the occurrence of the fever, 979.
varieties of, 978.
Malleus humidus, symptoms and diagnosis, 997.
Malta fever, 1004.
Malum perforans pedis in tabetics, 546.
Maniacal attacks of diphtheritics, 945.
of influenza pat., 986.
of typhoid fever, 911.
Marasmus causing atelectasis of the lungs, 107.
of the tuberculous, 138.
Mastication, movements of, centres for, 604.
muscles of, paralysis of the, 467; bilateral, 468; in bulbar paralysis, 468, 613; in cortical lesions, 468; in paralysis facialis, 472; in pontine disease, 608; in tabes dorsalis, 545; in trigeminus lesion, 443, 444.
spasm of, clonic and tonic, 496; differentiation from tetanus, 992; hysteric, 726; reflex nature of, 496.
Mastodynia, diagnosis of, 456.
Measles, ætiology of, 863.
anomalies of the course of, 866, 867.
complications of, 866; affections of the mucous membrane, 866; of the skin, 866.
contagiousness of the toxine of, 863.
defervescence in, 865.
desquamation stage in, 865.
diagnosis of, 866; differentiation from drug eruption, 867; from influenza, 870, 988; from roseola, 867; from röthein, 870; from scarlatina, 869, 878; from small-pox, 869, 887; from typhoid fever, 868; from typhus fever, 868.
eruptive stage of, 864.
exanthem of, 865.
hæmorrhagic, 865.
incubation period of, 863.
papulous, 865.
pathognostic symptoms of, 863, 864, 865.
prodromal stage, 863.
relapses of the eruption of, 866.
sequelæ, 867.
vesiculous, 865.
Meckel's ganglion, 438.
Medianus, paralysis of, diagnosis and symptoms, 490.
Mediastinal abscess, 153.
due to perforation of the œsophagus, 254.
emphysema, 117.
hæmorrhages, 153.
tumours, 146; auscultatory changes in, 148; causing tracheal and bronchial stenosis, 78, 149; combined with phlebitis, 147; differential diagnosis from aortic aneurysm, 152; from mediastinal abscess hæmorrhages, 153; from neoplasms of the pleura, 151; from pericardial exudate, 151; from pleuritic exudate, 107.
dyspnœa in, 146.
exploratory puncture for diagnostic purposes, 153; metastatic lymph-gland swelling in the neck, 150.
nature of, 153.
pectoral fremitus in, 147, 148.
pressure upon the œsophagus and the nerves of the thoracic viscera, 149; symptoms on the part of the circulatory system in, 148.
pulsation of, 149.
relation to œsophageal stenosis, 240.
symptoms on the part of the respiratory organs in, 146.
vascular murmurs in, 149.
Mediastinitis, purulent, in enteric fever, 916.
Mediastino pericarditis, calcareous, 45.
inspiratory swelling of the veins of the neck in, 47.
pulsus paradoxus in, 46.
Mediastinum, affections of, 146.
Mediterranean fever, 1004.
Medulla oblongata, anatomical conditions, 596, 602; blood-vessel distribution centres of the, for reflex movements, 605.
centres of the, for reflex movements, 604.
compression of, 617.
differentiation of, from basal tumours of the brain, 609; from pontine affections, 608, 609, 611; hypoglossus paralysis due to, 482, 608.
disease of, 596, 606; chronic, 612, 615; diagnosis of, 596, 606.
hæmorrhages in, 610, 615, 670.
processes of softening in, due to embolism and thrombosis, 610, 611, 615.
size of, in hereditary ataxia, 550.
Megalogastria, differentiation from gastrectasis, 291.
Melituria after epileptic attacks, 740.
in acute yellow atrophy of the liver, 178.
in apoplectic insult, 664.
in Graves's disease, 761.
in meningitis, 706; in epidemic cerebro-spinal, 950.
in pancreas affections, 222.
in pregnancy and lactation, differentiation from diabetes mellitus, 826.
in rabies, 994.
in syringomyelia, 572.
Memory, weakness of, of diabetics, 831; paralysis, 697.
Meningeal hæmorrhage, 718; diagnostic points of support, 719; differentiation from brain abscess, 694; genesis of, 718; in the new-born, 718.
tumours, spinal, 534, 582, 583; anæsthesia dolorosa due to, 584; compression symptoms of, 582, 584; of the cervical cord, differentiation from pachymeningitis cervicalis hypertrophica, 534; seat of, 584; unilateral lesion due to, 584.
Meningitis, cerebral, ætiological factors of, 691, 707.
acute spinal, 529; ætiology, 531; ascending, 531; descending, 532; diagnosis of, 530; differential, 532; from acute articular rheumatism of the vertebræ, 532, 975; from myelitis, 533, 578; from tetanus, 532, 991; with the aid of lumbar puncture, 532; infectious, 531; neuralgias in the sciatic nerve in, 461; pathological conditions in, 529; seat and extension of the inflammation, 531; symptoms, 530; rarer, 530; tuberculous, 532, 709.
Meningitis, anatomical findings in, 705.
and uræmia, 360.
basilar, 706.
cerebral, subsequent to spinal meningitis, 532, 707.
cerebro-siderans, 953.
cervicalis interna, 535.
chronic, 533 (see also Spinal); differential diagnosis, 534; in chronic alcoholism, 533; of the dura mater, 534; symptoms of, 533; upon syphilitic basis, 533.
combination of cerebral with spinal, 531; 707.
convexity, 707.
descending, 532.
diagnosis of, 702; the nature of, 707; differential of, 710; from acute miliary tuberculosis, 959; from cerebral abscess, 710; from cerebral hæmorrhage, 672; from cerebral syphiloma, 710; from cerebral tumours, 688; from delirium tremens, 711; from hydrocephaloid, 713; from hysteria, 711; from septicopyæmia, 712; from tetanus, 991; from typhoid fever, 712, 926; from uræmia, 711.
Meningitis, diffuse, 703.
epidemic, 708, 954 (see Cerebro-spinal Meningitis, Epidemic).
focal symptoms of, 705, 711.

Meningitis, general symptoms of, 703.
in childhood, 713; in diphtheria, 710; in mumps, 940; in pneumonia, 710; in scarlatina, 876; in typhoid fever, 710, 916, 918, 921; in typhus, 899; metastatic, 710; modifications of the course, 706; primary cryptogenetic, 954.
purulent, 702, 710, 953; differentiation from cerebral abscess, 694.
rheumatic, 710, 975.
seat and extent of, 706.
serous, 707.
tuberculous, 709, 953, 954, 957; course of, 706.
Meningococci, ætiological significance in epidemic cerebro-spinal meningitis, 952.
Meningo typhoid, diagnosis, 926; differential from epidemic cerebro-spinal meningitis, 954.
Mental disturbance, condition of, in epileptics, 739; in progressive paralysis, 697.
Mercurial tremor and tremor of multiple sclerosis, 593.
Mercyism, diagnosis and origin of, 307.
Mesenterial cysts, clinico-diagnostic significance of, 421.
differentiation from hydronephrosis, 383, 421; from retroperitoneal and pancreatic cysts, 421.
Metabolism, 817, 823.
anomalies of, diagnosis of, 772, 817; in anæmia, 776, 777; in diabetes, 823, 830; in gout, 845; in Graves's disease, 761; in obesity, 841; in rhachitis, 852.
Metallic note of the percussion sound in cavities of the lung, 137.
in pneumothorax, 163.
Metencephalon, 634.
Meteorism due to disturbances of innervation, 341.
in dysentery, 930; in hysteria, 726; in intestinal catarrh, 308; stenosis, 331, 332, 333, 334.
in typhoid fever, differentiation from perforation of the intestine, 915.
Metrorrhagia following enteric fever, 918.
Micrococci, ætiological significance, in acute articular rheumatism, 970.
in diphtheria, 940, 941; in epidemic cerebro-spinal meningitis, 952; in erysipelas, 891; in influenza, 985; in pertussis, 947; in septicopyæmia, 963, 964.
Micro-organisms, ætiological significance in chronic articular rheumatism, 850; in infectious diseases, 856, 857; protective measures of the organism against spread of, 859.
causing croupous pneumonia, 121; cystitis, 394; diphtheria, 71; endocarditis, 7; gastritis, 267; nephritis, 361, 367, 368; pericarditis, 409; tuberculosis, 130.
in the fæces in intestinal affections, 309.
in the oral cavity, 238.
in the pleuritic exudate, 162.
inthe sputum in pulmonary gangrene, 162.
Middle brain, anatomical structure and composition of, 628.
connection with the anterior, 634.
functions of the different parts of, 630; of the nerve nuclei, 629.
symptoms of, in disease of the cerebral peduncles, 630; of the corpora quadrigemina, 633.
Migraine, combined with paralytic phenomena, in the region of the oculomotorius, 453.
differentiation from headache due to cerebral tumours, 687; from occipital neuralgia, 448, 453, 463; from trigeminal neuralgia, 452, 463.
of tabetics, 546.
Miliary tuberculosis, acute, 955.
character of, 955.
clinical observation of, 958.
complicated with capillary bronchitis, 93; with pulmonary tuberculosis, 139.
diagnosis of, 955; differentiation from capillary bronchitis, 962; from catarrhal pneumonia, 127; from enteric fever, 922, 960; from influenza, 988; from meningitis, 959; from septic infection, 958, 969; from uræmia, 360, 959.
Miliary tuberculosis, friction sounds, 154.
of the kidney, 957.
of the liver, 957.
of the lungs, 956, 959.
of the meninges, 957.
of the peritonæum, 957.
primary focus of infection, 955.
symptoms of, 955.
tubercle bacilli in the blood and urine, 957.
Milker's cramp, 502.
Miserere due to enterostenosis, symptoms of, 331.
Mitral insufficiency in endocarditis, acute, 6; chronic, 13.
complication of aortic insufficiency, 21, 25, 36.
diagnosis of, by determining changes of pulmonary artery, 13; by means of auscultation, 13; by means of inspection, 12; by means of percussion, 12; by means of sphygmography, 14.
differential diagnosis from accidental systolic heart murmurs, 15.
in myocarditis, 49.
relative, 15.
subsequent symptoms, 14.
uncomplicated, 34.
with tricuspid insufficiency, 32.
stenosis, 15; absence of heart murmur in, 17; compensated, 19; complication with mitral insufficiency, 15, 18; with parenchymatous nephritis and hypertrophia cordis, 17; diagnosis of, with the aid of auscultation of the heart, 16; of inspection of the chest, 16; of palpation of the apex-beat, 16; percussion of the heart, 16; in endocarditis and its symptoms, 6; pulmonary artery in, 18, 19; radial pulse in, 18; sputum of patients with, 19; subsequent symptoms in the circulatory apparatus, 18.
Mixed infection by microbes in diphtheria, 242.
in croupous pneumonia, 121, 122.
in diphtheria, 941.
in enteric fever, 916.
in influenza, 985.
Mœbius's symptom, 760.
Mogigraphia, 502.
paralytic, 502.
Monilia caudita and oidium albicans, 238.
Monocontractures due to brain tumours, 683.
in meningeal hæmorrhages, 719.
Monoplegia after apoplexy, 671.
brachial, 480.
glossofacial, 482.
in brain tumours, 683.
in focal affections of the central convolution, 649, 650, 693; of the centrum ovale, 661, 671.
in meningitis, 705; epidemic cerebro-spinal, 950; spinal, 559.
Monotony of speech in multiple myelitis, 588.
in sclerosis cerebro-spinal multiplex, 590.
Morbilli, 863.
læves, 865.
miliares, 865.
Morbus maculosus Werlhoffi, 807.
chronic course of, 808.
hæmorrhages of the mucous membrane in, 808.
Morvan's disease, 572.
Motor disturbances in amyotrophic lateral sclerosis, 555.
in cortical lesions, 640, 641; of the central convolution and of the paracentral lobe, 649.
in Landry's paralysis, 587.
in myelitis, 573; due to pressure, 579; in neuralgias, 446, 453, 459; in neurasthenia,

732; in neuritis, 504, 506; in optic thalamus disease, 644; in syringomyelia, 572; in tabes dorsalis, 537, 539, 544.
Mouth affections of typhoid patients, 916.
aphthæ of the, 236.
catarrh of the, 236 (see also Stomatitis).
cavity of the, diagnosis of diseases of the, 235; of fungi in, 238.
musculature of the, paralysis of the, in facialis paralysis, 471, 472, 473.
scurvy of the, 237.
ulcers, syphilitic, differentiation from stomacace, 236; from tuberculous, 237.
Movements, associated, in central paralysis, 434; in hemiplegia of apoplectics, 669; in infantile, 696.
constrained, 496; in cerebellar lesions, 622; in pons oblongata disease, 608.
Mucous colic in colitis, 315.
fever and enteric fever, 927.
membrane atrophy in chronic gastric catarrh, 271; in intestinal catarrh, 318.
membrane hæmorrhages in Weil's disease, 213.
membrane, inflammation of, in erysipelas, 894; in influenza, 985; in measles, 863; in scarlatina, 871, 874, 875; in small-pox, 882.
Mumps, diagnosis of, 937.
disturbances of sight after, 940.
epidemics of, 938.
secondary localizations of, 939.
Murmur, systolic, in aortic insufficiency, 21.
in aortic stenosis, 26; in atheroma of the aorta, 62; in combined valvular disease, 35; in endocarditis, acute, 6; in mitral insufficiency, 13; in persistence of aortic isthmus, 38; in pulmonary tuberculosis, 135; in tricuspid insufficiency, 30; in water-wheel, 47.
Muscle abscesses in enteric fever, 916.
atrophy, neural, 567; beginning, localization, and symptoms, 567, 568; degeneration of peripheral nerves in, 554, 558, 559; differential diagnosis from spinal, 568; progressive spinal, 558, 563; anatomical condition in, 563; cause of, 565; degeneration of the anterior horn cells in, 555; differential diagnosis of, 566; from amyotrophic lateral sclerosis, 557, 566; from myopathic progressive muscle dystrophy, 570; from pachymeningitis cervicalis hypertrophica, 534; from poliomyelitis anterior chronica, 563, 566; from syringomyelia, 567; family character of, 567; peroneal type of, 567; symptoms of, 565, 566; bulbar, 566, 570, 614.
dystrophy, myopathic progressive, 568, 765; atrophy of the muscles of the face in, 569; beginning of, 568; conduct of the muscles in, 568; of the electric, 569; of the nervous system, 569; course of the atrophy of the muscles in, 568; differential diagnosis of, from amyotrophic lateral sclerosis, 570; from spinal progressive muscular atrophy, 570; family occurrence of, 569; infantile atrophic form of, 569; preponderance of pseudo-hypertrophy of some muscles over atrophy in, 569.
Muscles, sense sphere, centres of special senses of, 640, 642; pains of the, differentiation from neuralgia, 447; in the prodromal stage of relapsing fever, 902; of trichinosis, 999; rigidity of the, 495; in meningitis, 703, 704; in multiple sclerosis, 590; in paralysis agitans, 752; weakness of the, in anæmic conditions, 782; in cerebellar affections, 624; in commencing spinal progressive atrophy, 564; in myasthenia gravis pseudoparalytica, 617.
Muscles, inflammations of, metastatic, in septic infections, 966; of rheumatic, 974.
irritability, electric, in dystrophia muscularis progressiva, 568; in facial paralysis, central, 476; peripheral, 477; in myositis, 766; in myotonia congenita, 753; in neuritis, 503; in progressive muscular atrophy, 559, 565; in radialis paralysis, 487; in reaction of degeneration, 429; in Thomsen's disease, 753; in trichinosis, 999; in writer's cramp, 502.
Muscles, mechanical in myoclonia, 747.
relaxation of, in passive and active movements of tabetics, 544.
twitchings of, 496; after cortical hæmorrhages of the brain, 671; fibrillary in conus affection, 585; in progressive muscular atrophy, 565; in compression of the spinal cord, 580; in myoclonia, 747; in pachymeningitis cervicalis hypertrophica, 534; in spinal meningitis, 590.
Muscular atrophy, course of, in poliomyelitis anterior, chronic progressive, 564.
degenerative, 555, 558; in spinal pressure paralysis, 579.
in cervico-brachial neuralgia, 454; in disease of the anterior horns of the spinal cord, 525; in compression of the spinal cord, 579; in neuritis, 504; multiplex, 506; in pachymeningitis cervicales hypertrophica, 534; in polioencephalitis acuta infantum, 559; of adults, 561; in progressive bulbar paralysis, 612; in syringomyelia, 572; in tabes dorsalis, 548.
juvenile forms of, 568.
of the fingers, in Klumpke's paralysis, 492; in medianus paralysis, 491.
of the lower leg in paralysis of the peroneus, 494; tibialis, 495.
of the upper extremities in amyotrophic lateral sclerosis, 555.
primary myopathic, 568.
rheumatism and pleurisy, 158.
symptomatic, 566.
unilateral, in the face, 754.
Myasthenia gravis pseudoparalytica, 616.
Mycosis intestinalis, origin and nature of, 995, 996.
pathological picture of, 995.
Myelæmia, 792.
Myelitis, 573.
acute, 573, 577.
ætiological points for, 579.
bladder-rectum functions in, 576.
cervical, 577.
chronic, 573, 577.
condition of the reflexes in, 574.
degenerative processes in transverse, 552.
diagnosis of diffuse, 573, 577; differentiation of acute, from Landry's paralysis, 578; from polyneuritis, 578; from spinal meningitis, 533, 578; chronic, from amyotrophic lateral sclerosis, 557; diffuse, from infantile spinal paralysis, 560; paralytic symptoms from hysteric paralysis, 578.
diffuse, 573.
disseminated acute (multiple), 588; chronic, 589.
dorsalis, 577.
due to compression, 579, 580.
lumbalis, 576.
symptoms of, motor, 573; sensory, 573; sexual, 576; trophic, 575; vaso-motor, 575.
transverse, 573, 578.
traumatic, in the lowest portion of the spinal cord, 584.
Myelomalacia, diagnosis of, 595.
embolic and thrombotic, 595.
Myocarditis, 48.
acute, 49.
ætiological factors of, 49.
chronic, 50.
complication of, with endocarditis and pericarditis, 50.
degenerative, in enteric fever, 917.
differential diagnosis, 49; from endocarditis, 8.
heart sounds in acute, 49; in chronic, 50.
in acute articular rheumatism, 974; in scarlatina, 875.
interstitial, in diphtheria, 944.
pulse changes in, 48.
stasis symptoms in, 48.
syphilitica, 50.
uncomplicated, 50.

Myoclonia, 747.
characteristics of, 747, 748.
diagnosis, 747, 748.
occurrence in hysterics, 748.
reflex activity in, 748.
Myomata of the stomach, 286.
Myosis in Klumpke's paralysis, 492.
in tabes dorsalis, 537.
Myositis, acute multiple, 766; clinical observation of, 767; diagnosis of, 766; differentiation from polyneuritis, 770; from trichinosis, 769; electrical condition of muscles in, 767; infectious character, 766, 770; pathological findings in, 768; symptoms, general, 766; local, 769.
ossificans, 770.
purulent, 770.
syphilitic, 770.
Myotonia, electrical and mechanical reaction of nerves and muscles in, 753.
pathological condition of muscles in, 753, 754.
Myxœdema, 758.
and Graves's disease, 759.
and tetany, 759.
and thyreoid gland functions, 758.
cachectic conditions due to, 758.
differentiation from acromegaly, 758.
mucin accumulation in the subcutaneous cellular tissue, 758.
nervous disturbances of, 758.
tremor in, 758.
Myxoma of the meninges of the spinal cord, 584.

N

Neapolitan fever, 1004.
Neck, muscle spasms of the, 499, 500; due to occipital neuralgia, 453; hysteric, 726.
rigidity of the muscles of the, 500; cause of, 949; in meningitis, 703, 704; epidemic cerebro-spinal, 949; spinal, 533, 534; in pachymeningitis hypertrophic, 534.
Neoplasms in the larynx, 78.
of the bladder, 399; of the kidneys, 385; of the liver, 196; of the lung, 145; of the mediastinum, 146; of the œsophagus, 246; of the pancreas, 223; of the peritonæum, 419; of the pleura, 168; of the spleen, 232; of the stomach, 280.
Nephralgia, 389.
Nephritis, 351.
acute, 351; ætiological factors, 355; condition of urine, 351; differential diagnosis, 355; from febrile albuminuria, 356; diffuse, 351; dropsy due to, 353; hæmoglobinuric, 355; hypertrophy of the heart in, 353; in disease of the bile ducts, 208; in pulmonary tuberculosis, 139; in the course of chronic Bright's disease, 355; parenchymatous, 351; in valvular defects, 17; pulse conditions, 354; pyæmic, septic, suppurative, 367; symptoms, 352, 353; uræmic, 354.
chronic, 357; ætiological diagnosis, 361; differential diagnosis, 360; from diabetes insipidus, 837; dropsy due to, 357, 358; following whooping-cough, 948; hæmorrhagic, 363; hypertrophy of the heart in, 363; in acute articular rheumatism, 974; in diphtheria, 944; in enteric fever, 918; in epidemic cerebro-spinal meningitis, 951; in erysipelas, 893; in gout, 847; in influenza, 986; in measles, 866; in mumps, 938; in scarlatina, 874; in septic infection, 968; indurating, 364; inflammation of the serous membranes in, 358; interstitial, 362; parenchymatous, 357; pulse in, 358; subacute and subchronic, 357; uræmic symptoms of, 358; urine in, 357.
Nephrocrises of tabetics, 538.
Nephrolithiasis, 387.
concretions in the urine in, 389.
diagnostic symptoms, 388.
uræmia due to, 388.
urine in, 388.
Nephrophthisis, 374.
complications, 375.
diagnostic symptoms of, 374, 375.
primary, 374.
proof of tubercle bacillus in the urine, 375.
seat of, 376.
secondary miliary metastatic, 374.
Nephroptosis (see also Floating Spleen, 391).
Nerve cells, 426; column cells, 517; glia, 515; motor, 515, 517; of the gray substance, 517; protoplasm processes, 517; spinal cord, 515, 517; interior cells, 517; supporting structures, 515.
conduction of, 426, 517; apparatus of conduction for, 427; cellulifugal, 426; cellulipetal, 426; centrifugal, 427, 516, 543; centripetal, 427, 516, 521, 537, 543, 544; centripetal-centrifugal, 427; interruption of, and consequences, 429, 445, 552; isolated, 427; motor, 427, 517, 518; of reflex processes, 522, 523, 524; of sensory irritations, 427, 435, 515, 520; in tabes, 542, 543, 545.
irritability, electric, 429; decreased, 434; in anæmic conditions, 782; in disturbances of nutrition, 434; in facialis paralysis (central), 476; in facialis paralysis (peripheral), 477; in myotonia, 753; in neuritis, 506; in tetany, 749; increased, 434; mechanical, in tetany, 749; in Thomsen's disease, 753.
processes of the spinal cord, 517.
Nerve roots, anatomical structure and physiological functions of, 516, 517, 518, 519; diseases of the anterior, 525; diseases of the posterior, 526, 537, 540; by pressure, 580; motor innervation of, 529.
Nerves, degeneration of, due to diphtheria, 944; of peripheral nerves in amyotrophic lateral sclerosis, 555; in neuritis, 504, 508; in poliomyelitis anterior, 558; of the spinal cord, 536; in tabes dorsalis, 540.
diseases of, diagnosis of, 425; of the peripheral, 426; ætiological points, 434; of the sensory nerves, 444; due to scarlatina, 876; functional, 722; in cryptogenetic septicopyæmia, 996; in diabetes, 830; in influenza, 985, 987; in relapsing fever, 904; in small-pox, 886; in typhoid fever, 914, 918; in typhus, 899.
Nervousness (see Neurasthenia).
Nervous symptoms in acute yellow atrophy of the liver, 176.
in jaundice, 208.
in pancreas affections, 223.
Nervous system, decrease of energy of, in anæmic conditions, 782.
disease of, 425; of the peripheral, 426.
structure of, 426.
Nervous system, effect of, upon gastric-juice secretion, 258.
examination of, vii.
Neuralgia cervico-brachialis, 454.
cœliaca (in trichinosis), 999.
cruralis, 456.
in affections of the pancreas, 223.
inframaxillaris, 452.
intercostalis, 454.
mesenterica, 342.
obturatoria, 457.
occipitalis, 453.
of the region of the kidneys in renal tumours, 377; of the liver, in aneurysm of the hepatic artery, 220; of the stomach, differentiation from gastric ulcer, 278.
ophthalmica, 451.
painful areas of the nerves in, 446.
peripheral, 448.
phrenica, 454.
predisposition to, 450.
pudendo-coccygea, 462.
reflex, 450.
rheumatic, 450, 974.
seat of, 448.
supramaxillaris, 452.
Neuralgias, 436.
accompanying symptoms, sensory, 446; trophic, 447; vaso-motor, 446.
central, 448.

characteristics of, 446.
diagnosis of, 446; ætiological, 449; differential, from rheumatic pains due to inflammation, 447.
due to chemical noxa, 449; due to infectious substances, 449; due to intoxications, 450; due to refrigerations, 450; due to traumatic and mechanical influences, 449.
Neuralgias in anæmia, 450, 782.
in compression of the spinal cord, 580.
in diabetes mellitus, 830.
in gout, 846, 847.
in malaria, 450, 983.
in trichinosis, 999.
of cervical nerves, 453.
of cruralis, 456.
of cutaneous femur, lat., 456; posterior, 456.
of glans penis, 462.
of hysterics, 728.
of lower extremities and tabes, 546.
of lumbar nerves, 456.
of mixed nerves, 448.
of obturatorius, 457.
of plexus ischiadicus, 458.
of sacral nerves, 458; of scrotal and pubic plexus, 462.
of the joints, diagnostic points of support, 462; examination of the motility of the joints in, 462; in traumatic neuroses, 734; psychical effects in, 462.
trigeminus, 451; in sinus thrombosis, 716.
Neurasthenia (nervousness), 731.
acute, 733.
ætiological factors, 732, 733.
chronic, 733.
circular, 732.
diagnosis of, 733; differential, from angina pectoris, from cerebral affections, 734; from nervous stomach, intestinal affections, 733, 734; and tabes, 733, 734.
dyspeptic, 299.
functional weakness of the nervous system in, 732.
insomnia in, 733.
modified forms of, 733.
psychical conduct of neurasthenics, 732.
sexual, 733.
spinal, differentiation from tabes dorsalis, 547.
symptoms in, of internal organs, 733; of motor, sensory, and vaso-motor spheres, 732; tabetic, 733.
Neuritis, structure and function, 426.
ætiological points of support for the diagnosis of, 505.
after enteric fever, 18.
beginning and course of, 508.
characteristics of, 503.
chronic and progressive, 568.
circumscribed, 503, 509.
differential diagnosis of, 509.
duration of, 509.
in childhood, differentiation from spinal infantile paralysis, 561.
in diabetes, 831.
in tabes dorsalis, 544.
interstitial, 504.
motor disturbances in, 434, 504, 506, 566.
multiple, 503, 504; differentiation from acute myelitis, 578; from spinal infantile paralysis, 561; from tabes dorsalis, 548; intestinal ulcers in the course of, 323; origin of, 505; symptoms, 505; rarer, 508.
nodosa, 504.
optic, in meningitis, 950; in multiple neuritis, 511, 549.
parenchymatous degeneration due to, 504.
potatorum, differentiation from tabes, 511, 549.
spontaneous, 505.
under the picture of acute amyotrophic spinal paralysis, 562; of bulbar paralysis, 508.
utilization of therapeutical success in diagnosis, 511.
Neuroma, motor disturbances due to, 434.
Neurone systems, affections of, 536.
Neurones of the nervous system, anatomical structure of, 426, 517.
central and peripheral, 427.
conduction of, 427, 518.
construction of the nervous system from, 426.
degeneration of (Waller's law), 427; diseases, motor, 554, 558.
disease of motor central, 525, 554; of motor peripheral, 433, 525, 554, 558; combination of central and peripheral, 526, 554; sensory peripheral, 540.
function of, 426, 427, 517.
stimulation of, 427, 428.
Neuropilem, anatomical structure of, 426.
Neuroses, course of, under the picture of multiple sclerosis, 594.
diagnosis of, 722.
of motility of the intestine, 339; depressive, 341; of the larynx, 82; of the œsophagus, 255; of the stomach, 304; of the urinary bladder, 401.
of the heart, 57.
of the larynx, 81.
of the œsophagus, 292.
of the stomach, 292.
of the urinary bladder, 401.
of traumatic, 734.
Neurosis of fright, 748; secretion of intestines, 345; of stomach, 300.
traumatic, 734; ætiology, 734; diagnosis of, 735; differential, 735; hysteric neurasthenic symptoms in, stimulation of, 735.
Nicotine tabes, 747.
Nictitation, 497.
Nitrogen equilibrium of the body, 820.
excretion in anæmics, 777, 786; in diabetics, 827.
Nodding spasms, diagnostic criteria, 497, 498, 500.
Noma, 237.
and anthrax, 237.
Nose, mucous membrane of diphtheria of the, 941, 943; complicated, 943.
glanders of the, 998.
Nucleus caudatus, 634; focal symptoms of, 645; funiculi gracilis and funiculi cuneati, 596.
lentiformis, 634, 637; focal symptoms of, 645.
tegmenti, location and structure, 617, 626.
Nun's murmur in anæmic conditions, 784.
Nutmeg liver, atrophic, 193.
and cirrhosis of the liver, 184.
Nystagmus in cerebellar affections, 623.
in multiple myelitis, 588.
in sclerosis of the spinal cord and brain, 590, 592.

O

Obesity (corpulence), 839.
ætiological factors, 841; favouring, 842.
anomalies of metabolism in, 841.
differential diagnosis, 843; from cutaneous emphysema, œdema, pseudo-hypertrophy of the muscles, 843.
excretion of water in, 841.
form of the body and weight in, 839.
insufficiency of the heart action in, 840.
nervousness of, 841.
power of resistance of the organism in, 843.
respiratory disturbances in, 840.
Obliquus capitis inferior, spasm of, 80.
Obstetric palsy, cause and symptoms, 491.
differentiation from infantile spinal paralysis, 560.
Obturatorius, neuralgia of, 457.
paralysis of, 493.
Occipital cortex, centres of, 642.
diagnostic, valuable, symptoms in affections of, 639, 649.
Occupation neuroses, 501.
diagnosis of, 502.
Oculomotor nerve, paralysis of, 465.

Oculomotor nerve, alternating, 465, 631.
bilateral, 465.
central, 465.
due to disease of the cerebral peduncles, 482, 631, 632; in apoplectic stroke, 670.
in lesion of the corpora quadrigemina, 633.
in tabes dorsalis, 537, 539.
nuclear, 205.
partial, 465, 631.
peripheral, 466.
recurring, 453.
seat of the cause of, 465, 466.
symptoms of, 465.
total, 465, 631.
with hemiplegia, 466.
Oculomotorius, nucleus of, location and structure, 629.
Œdema, acute angioneurotic, 755; in anæmia and chlorosis, 777, 786; of the eyelids in influenza, 986; in sinus thrombosis, 717; in trichinosis, 999.
general, in pulmonary tuberculosis, 138; of the larynx, 72; due to perichondritis, 72; of the lower half of the body in cirrhosis of the liver, 182; in nephritis, 353; in renal cancer, 386; in renal stasis, 349; of the upper half of the body in mediastinal tumours, 149.
of the skin, acute circumscribed, 755; hysteric (" blue "), 729; in anthrax, 996; in erysipelas, 892; in glanders, 998; in Graves's disease, 761; in leucæmia, 794; in myositis, 767; in myxœdema, 758; in neuritis, 504, 508; in scarlatina, 875; in sinus thrombosis, 717; in small-pox, 882; in syringomyelia, 572; in trichinosis, 999.
Œsophageal crises of tabetics, 122.
sound, in œsophageal stenosis, 248.
Œsophagismus, 255.
Œsophagitis, 245.
phlegmonous, 245.
Œsophagus, affections of, 245.
cancer of, 246; difficulty of deglutition, 246; pneumopericardium following, 48; stenotic symptoms, 247.
compression of, by aortic aneurysms, 66; by mediastinal tumours, 149.
dilatation of, 251; partial, 251; total, 251.
diverticulum of, 249.
hæmorrhages of, 254.
loss of continuity, 253.
neoplasms of, 246.
neuroses of, 255.
paralysis of, 255; hysterical, 255, 725.
perforations of, 254.
polypi of, 250.
rupture of, spontaneous, 253.
sounding of, 248.
spasm of, 249.
stenosis due to, 249.
stenosis of, 247; auscultatory symptoms, 247; causes of, 248, 250; difficulties of deglutition, 247; disturbances of nutrition in, 248; due to carcinoma of the œsophagus, 251; nature of, 248; regurgitation of food particles, 247; sounding of the œsophagus in, 248.
stricture of, cicatricial, 250; spastic, 249.
ulcers of, 245; due to pressure, 246; peptic, syphilitic, tuberculous, 245.
Oidium albicans in the œsophagus, 250.
in the oral cavity, 238; in the stomach, 267.
Oligæmia of pneumonia, 125.
Oliguria in neuritis, due to irritation of the sympathetic nerve, 504.
in acute intestinal catarrh, 308.
Olives, anatomical location in the medulla oblongata, 596, 602.
relation to the lemniscus, 627.
Omentum, tumours of, 421.
and gastric cancer, 285.
compression of the bile ducts due to, 212.
tuberculous, 420.
Ophthalmia neuroparalytica in sinus thrombosis, 718.
Ophthalmoplegia, acute alcoholic, 205.
interior, 205.
progressive, 205; nuclear, 205.
Ophthalmoscopical findings in brain tumours, 681, 682, 685, 688.
in cryptogenetic septicopyæmia, 967.
in diabetes mellitus, 831.
in Graves's disease, 760.
in leucæmia, 794.
in meningitis, 706; in epidemic cerebro-spinal, 950; in tuberculosis, 957.
in multiple sclerosis of the spinal cord, 548, 590.
in sinus thrombosis, 718.
in tabes, 537, 548.
Opisthotonus in meningitis, 704; in epidemic cerebro-spinal, 949.
in tetanus, 990.
Oppression, sensation of, in angina pectoris, 57.
Optic nerve, atrophy of, in hydrocephalus, 720; in lesion of the corpora quadrigemina, 630; in multiple sclerosis of the spinal cord and brain, 592, 689; in progressive paralysis, 697; in tabes dorsalis, 630.
centres of the, primary, location of, 635, 643.
thalami, anatomical, location of, 634; foci in, and symptoms of, 644; structure and formation of, 634, 635.
Orbicularis oris, paralysis of, 472.
palpebrarum, paralysis of, 468, 472; spasm of, 496.
Orchitis in mumps, 939.
in small-pox, 886.
in typhoid fever, 918.
Ossification of rhachitic bones, 852.
Osteitis deformans, 854.
clinical picture, 854.
pathology, 855.
Osteoarthropathy, hypertrophic, thickening of the finger phalanges in, 757.
Osteomalacia, 850.
causes of, 851.
changes of form of the skeleton in, 850.
differentiation of, from myelogenous sarcomata and cancerous bone infiltration, 851; from rhachitis, 851.
general symptoms of, 850, 851.
products of excretion of the urine in, 851.
puerperal, 851.
Otitis media in epidemic cerebro-spinal meningitis, 951.
in epidemic parotitis, 939.
in influenza, 987.
in typhus fever, 898.
Ovarialgia of hysterics, 728.
Ovarian cysts and ascites, 328; and hydronephrosis, 382.
tumours and cancer of the intestine, 328; and tumours of the kidneys, 379; causing jaundice, 212.
Oxalate stones in the urine in nephrolithiasis, 389.
Oxyacids, aromatic, in the urine in acute yellow atrophy of the liver, 177.
Oxygen consumption in Graves's disease, 762.

P

Pachyacria, 757.
Pachydermia of the larynx, diffuse, 79; verrucose, 79.
Pachymeningitis spinalis externa, 533.
hæmorrhagica (interna), 534; combined with durhæmatoma of the brain, 534; differentiation of, from apoplexy, 673; origin of, upon the basis of psychoses and the abuse of alcohol, 534.
hypertrophica (cervicalis interna), 534; differential diagnosis, 534; from amyotrophic lateral sclerosis, 534.
stages of, 533.
Pædatrophy, 318.
Paget's disease, 854.
Pain, in Addison's disease, 405.
in amyloid liver, 196.
in angina pectoris, 57, 454.
in colitis, acute, 315 (in the left arm), 57.

Pain, in cystitis, 393.
in gastric ulcer, 274.
in intestinal cancer, 325; in intestinal ulcer, 321.
in irritations, summation of, 435.
in liver abscess, 189; in liver atrophy, 176; in liver cancer, 198; in liver syphilis, 188.
in locomotor ataxia, 537, 538.
in nephritis, acute, 353.
in nephrolithiasis, 388.
in neuralgia of the belly wall in renal tumours, 377; of the trigeminus, 451.
in pericarditis, 41.
in peritonitis, 408.
in perityphlitis, 312.
in pleurisy, 154.
in sciatica, 459.
in Weil's disease, 213.
irradiation of, 435.
points of, of the nerves, diagnostic utilization of, in neuralgia, 446; in crural, 456; in intercostal, 445; in lumbo-abdominal, 456.
sensations, causation of, central, 436; in facialis paralysis, 471; in peripheral, 436; decreased, 436; in affections of the posterior horns of the spinal cord, 526; in syringomyelia, 571; in anæsthetic areas of the skin, 445; increased, 436; in hysteria, 727; in meningeal tumours, 584; in muscular rheumatism, 765; in neurasthenia, 732; in neuritis, 503, 505, 509; in rhachitis, 853; in spinal meningitis, 530, 533, 949; nerve tracts of, 435; in the spinal cord, 520, 571.
Palate, affections of the, diagnostic symptoms of, 239; of diphtheria, 242; of syphilis, 244; of tuberculosis, 244.
paralysis of the, after diphtheria, 944.
spasm of the muscles of the, 497.
velum of the, paresis of the, with oblique position of the uvula in facialis paralysis, 469, 473, 478.
Palpation of the abdominal organs in affections of the latter, 167; walls, in acute peritonitis, 410.
apex beat in aortic stenosis, 26; in mitral insufficiency, 12; in mitral stenosis, 16; in pericarditis, 40.
chest in emphysema of the lungs, 112; in mediastinal tumours, 147; in pleurisy, 156.
liver in amyloid liver, 195; in fatty liver, 194; in liver abscess, 189; in liver atrophy, acute yellow, 177; in liver cancer, 197; in liver cirrhosis, 180; in corset lobe of the, 206; in echinococcus, 204; in mediastinal tumours in the jugular fossa, 150; in pancreatic cancer, 224; in renal tumours, 377.
of spleen, 227.
of stomach in gastrectasis, 288.
of voice in pneumonia, 123.
Palpebral fissure, narrowing of the, in Klumpke's paralysis, 492.
Palsy, shaking (see Paralysis Agitans), 751.
Pancreas, affections of the, 222; and diabetes mellitus, 836; ascites in, 223; effect of, upon the solar plexus, 223; upon the urine, 222; inhibition of cardiac action in, 223; jaundice in, 223; tuberculous, syphilitic, and purulent, 226.
cancer of, 223; differential diagnosis, 224; palpation of, 224, 225; from mesenteric cysts, 421; exploratory puncture in, 225.
hæmorrhage of, acute, 223.
stone of the, colic in, 225; diagnosis of, 225, 226.
Papilloma of the larynx, 79; diffuse, 79.
of the urinary bladder, 399.
Paracentral cortical portions of the cerebrum, focal symptoms of, 649.
Paracystitis, 398.
Paræsthesia, 436.
in hysteria, 728.
in spinal meningitis, 533.
in syringomyelia, 571.
Paræsthesia, in tabes dorsalis, 538, 545.
in traumatic neuroses, 734.
of the arms in pachymeningitis cervicalis hypertrophica, 534.
of the larynx in hysteria and neurasthenia, 82.
of the rectum in tabetics, 538.
Paragraphia and aphasia, 657.
Paralysis, 465; agitans, 751; differential diagnosis of, 752; from multiple sclerosis, 593; muscular rigidity in, 752; pathology of, 751; propulsion and retropulsion in, 752; tremor in, 751.
alternating, 466; in facialis paralysis, 476; in pons-medulla disease, 607.
amyotrophic, in syringomyelia, 572.
atrophic, of the forearms and hands in Klumpke's paralysis, 492; muscles in multiple neuritis, 548.
Paralysis, Brown-Séquard's, 582.
central, 433, 466.
combined paralyses of the nerves of the arm, 491.
crossed and alternating, 466.
differential diagnosis between central and peripheral, 433, 434.
diphtheritic, 944.
Duchenne-Erb, 491.
electric irritability of nerves and muscles in, 431, 432.
glossopharyngolabialis cerebralis et cerebrobulbaris, 204.
hysteric, 724; course of, 725; differentiation from paraplegias due to myelitis, 578, 724.
Paralysis in cervico-brachial neuralgia, 454.
in cryptogenetic septicopyæmia, 967.
in the dorsal nerve region, 492.
in the lumbar and sacral nerve regions, 492.
in the medianus region, 490.
in the radialis region, 486.
in the ulnaris region, 489.
infantile, spinal (essential), 558; beginning of, 558; characteristics of, 559; differentiation of, from peripheral paralyses and polyneuritis, 560; intensity and distribution of, 559.
Klumpke's, 492.
of nerves, etc., of the larynx, 83; of the œsophagus, 255; of the rectum, 341, 342.
of the abducens, 466; of the accessorius, 465, 479; of the arm muscles, 486, 491; of the cerebral nerves (motor), 465; of the cruralis, 493; of the eye muscle nerves, 466; of the facialis, 465, 468; of the glutæi, 493; of the hypoglossus, 465, 481; of the obturatorius, 493; of the oculomotorius, 463; unilateral, in migraine, 453; of the phrenicus, 483; of the sciatic nerve, 494; of the trigeminus (motor), 467; of the trochlearis, 466; of the vago-accessorius, 479.
Paralysis, peripheral, 430, 433, 434, 465, 466; in diphtheria, 944; in neuritis, 503, 506.
progressive, diagnostic, valuable symptoms of, 674, 697, 698.
relaxed, in anterior horn disease of the spinal cord, 519, 525; in Landry's paralysis, 586; in neuritis, 503, 506; in poliomyelitis anterior, 559, 561.
spastic, in myelitis, 575; due to compression, 579.
spinal, 433; acute amyotrophic, of adults, 561; ascending, 586; due to compression, 579; of adults, 561; of children, 558; spastic, 553; degenerative processes in, 533; gait of patients in, 533; types of, 533, 534.
total, 466.
unilateral, 466; in facial paralysis, 475.
vaso-motor, in unilateral lesion of the spinal cord, 582.
Paralytic symptoms in acute articular rheumatism, 974.
in anæmia, 783.
in apoplectic stroke, 664, 665, 666, 667, 668, 669; in cerebral abscess, 693; in cerebral tumour, 683.

Paralytic symptoms in compression myelitis, 579.
in conus affection, 585.
in facialis paralysis (central), 475, 476.
in focal affections of the central convolutions, 641; of the internal capsule, 645; of the pons medulla, 607; of the optic thalami, 644.
in meningeal hæmorrhages, 719; in meningitis, 706; epidemic cerebro-spinal, 950; spinal, 530, 533; in myelitis acuta, 533, 574; in neuralgias, 448; in neuritis, 504; multiple, 506; in pachymeningitis hypertrophica, 534; in paralysis agitans, 752; in poliomyelitis anterior, 558; in progressive paralysis, 697; in speech disturbances, 659; in syringomyelia, 572; in tabes dorsalis, 539, 544; in traumatic neuroses, 734; in vagus paralysis, 479; subsequent to scarlatina, 876.
Paramyoclonus multiplex, 341.
Paranephritis, 372.
diagnostic symptoms of, 372.
differential diagnosis of, from fæcal tumours, paratyphlitic abscess, 373; from renal abscess, 373; from psoas abscess, 373.
Paraphasia, 653.
Paraplegia of the lower extremity in renal abscess, 371; in renal cancer, 386.
Paraplegic symptoms in Graves's disease, 762.
in hysteria, 724.
in myelitis, 574; multiplex, 589.
in spinal meningitis, 533.
in tumours of the cauda equina, 584.
in typhoid fever, 918.
Parasites in the bile-passages, causing jaundice, 221.
in the intestine, 345.
in the spleen, 232.
Paratyphlitis, 314.
Paratyphoid, 927.
Pareses in amyotrophic lateral sclerosis, 555.
in æmia, 783.
in syringomyelia, 572.
in tabes, 544.
in traumatic neuroses, 734.
spastic, in multiple sclerosis of the spinal cord, 590.
Paresis of the heart, ætiological significance in cardiac asthma, 60; in pulmonary œdema, 119.
of the musculature of the stomach, 307.
of the vocal cord, 84.
Parietal cortex, foci of, 649.
Parkinson's disease, 751.
Parotitis, bilateral, 938.
complications of, 939.
contagiosity of, 938.
differential diagnosis of, 939; from metastatic parotitis, 939.
epidemic, 938.
facial expression in, 938.
immunity after, 938.
in diphtheria, 944.
in enteric fever, 916.
in typhus fever, 898.
incubation period, 938.
prodromes of, 938.
sequelæ, 940.
swelling of the submaxillary and sublingual glands, 938.
tumour in, 938.
Parturition paralysis, 560 (see Birth Paralysis).
Patellar tendon reflex in capsule affections, 647.
in diabetes mellitus, 831; in hereditary ataxia, 550; in Landry's paralysis, 587; in tabes, 542; in tetany, 749; in trichinosis, 999.
significance of, for the localization of myelitis, 574.
Pectoral fremitus in bronchiectasis, 100.
in bronchiostenosis, 96; in capillary bronchitis, 93; in exudative pleurisy, 156; in mediastinal tumours, 147; in pneumonia, 123, 127, 129; in pneumothorax, 163; in pulmonary atelectasis, 106; in pulmonary emphysema, 112; in pulmonary hypostasis, 109; in pulmonary œdema, 117; in pulmonary tuberculosis, 134, 138.
Pectus carinatum due to rhachitis, 853.
Peduncles of the cerebellum, 619.
of the cerebrum, 626.
Peliosis rheumatica, 808.
differential diagnosis from acute articular rheumatism, 975.
Pelvis, cellular tissue of the, purulent inflammation of the, cause of acute spinal meningitis, 531.
rhachitic deformity of the, 853.
Pemphigus, differentiation from chickenpox, 890.
in cryptogenetic septicopyæmia, 966.
in neuralgia of the cervical nerves, 454.
in neuritis, 504.
in pachymeningitis cervicalis hypertrophica, 534.
in tabes dorsalis, 545.
(of the fingers) in medianus paralysis, 491.
Pentosuria, chronic, 826.
Pepsin digestion, 257.
Peptonuria, 347.
Percussion, variation of the height of the note in bronchiectatic cavities, 100.
in fibrinous pneumonia, 122.
in pneumothorax, 163.
in tuberculosis, 132.
Peribronchitis, tuberculous, 134.
Pericardial synechiæ, 45.
condition of the pulse and veins of the neck in, 46.
retraction of the anterior thoracic wall in, 46.
Pericarditis, 39.
auscultation of the heart in, 40.
cause of œsophageal stenosis, 249.
circulatory disturbance in, 41.
condition of the apex beat in, 39.
covering of the exudate by pulmonary emphysema, 43.
differential diagnosis of, 42; from endocarditis, 9.
diffusion of cardiac dulness in, 39.
due to septic infection, 967.
exudate in, 45.
friction sound in, 40.
heart action diffused and weakened in, 40.
hydropericardium in, 42.
in acute articular rheumatism, 974.
in diphtheria, 944.
in erysipelas, 894.
in mumps, 940.
in pertussis, 949.
in scarlatina, 875.
inspection and palpation in, 40.
mediastinal tumours in, 151.
percussion in, 39.
secondary symptoms in, 41.
sequelæ, 45.
subsequent phenomena of the pressure upon the neighbourhood in, 41.
tuberculous, in acute miliary tuberculosis, 957.
Pericardium, diseases of the, 39.
Perichondritis, laryngeal, diagnosis of, 72; differential, from laryngitis submucosa, 70.
seat of, 73.
Perihepatitis, 205.
friction sound in, 205.
jaundice in, 212.
syphilitic, 188.
Perineal neuralgias, 462.
Perineuritis, 504.
Periostitis after enteric fever, 918.
metastatic, due to septic infection, 966.
purulent in diphtheria, 944.
Peripheral nerves, centres of the motor, 641.
conduction of the, 427.
connection of motor-root fibres with, 519.
degeneration of, 520, 555, 565.
diseases of, 426; ætiological points for the diagnosis of, 434.
inflammation of, 504.

Peripheral nerves, in tabes, 543.
interruption of continuity, and consequences, 429.
irritations for, 430.
reflex activity of, 428.
sclerotic changes of, 589.
terminal distribution of, in the extremities, 444, 445, 458; in the head, 452.
Peripleuritis, 160.
Peripylephlebitis, syphilitic, 188.
Perisplenitis, 232.
Peristalsis of the intestine, decreased, 334; increased, 331.
stomach, 258; peristaltic unrest of, diagnosis of, 304.
Peritonæum, affections of the, 408.
neoplasms of the, 419.
tuberculosis of the, 417.
Peritoneal cancer, 419; intestinal friction sounds in, 419; primary, 420; secondary, 419.
exudate, condition, 408; determination of, 409; differentiation of, from hydronephrosis, 383; from intestinal cancer, 328.
Peritonitis, 408.
acute, 408; body temperature in, 408, 410; circumscribed, 412; colic and gastralgia due to, 412; complication with intestinal occlusion, 332; complication with intestinal ulcers, 321; course of examination to find cause of, 411; deviations from the usual picture, 410; differential diagnosis of, from gastritis, 266; from intestinal stenosis, 332; from pleurisy, 158; exploratory puncture in, 408; generators of, 409; high position of the diaphragm in, 409; hyperæsthesia of the belly walls in, 410; morbid picture of, 408; perforation in, 413; physical examination in, 408, 413; primary, 411; puerperal and traumatic, 409; relation of a general affection to the genesis of, 412; septic, 410.
Peritonitis, chronic, 414; circumscribed adhesive, 414; circumscribed purulent, 415; differential diagnosis of serous, from ascites, 416; diffuse, 415; in hepatic cirrhosis, 185; puncture fluid of serous, 416; serous, 416; tuberculous, 417; differential diagnosis, 418.
differentiation of, from intercostal neuralgia, 455.
due to septic infection, 967.
in acute articular rheumatism, 974.
in diphtheria, 944.
in erysipelas, 894.
in scarlatina, 876.
purulent, due to typhoid intestinal ulcers, 915.
tuberculous, in acute miliary tuberculosis, 957.
Perityphlitis, 311.
course of, 313.
differentiation of perityphlitic tumour from other ileo-cæcal tumours, 313.
symptom-complex of, 312.
ulceration of the vermiform process in, 313.
Peroneus paralysis, 494.
Pertussis, 946.
Perverse instincts of epileptics, 74.
Pes equinus due to paralysis of the peroneus nerve, 503.
in hereditary ataxia, 550.
in poliomyelitis anterior acuta, 560.
Pest, 1004.
Petechial typhus, exanthem of, 897.
Phagocytosis of Metschnikoff in bacteria infection, 860.
Pharyngeal diphtheria (see Diphtheria).
Pharyngitis, 239.
chronic granulosa, 240.
diffuse chronic superficial, 240.
diphtheritic, 942.
erysipelatous, 894; in chronic laryngitis, 70; in enteric fever, 916; in epidemic cerebro-spinal fever, 949; in poliomyositis, 767; in small-pox, 881; in typhus fever, 897; in whooping-cough, 946; syphilitic, 4; tuberculous, 244.
Pharynx, diseases of the, 239; and diphtheria, 242.
examination of the, 239.
infectious phlegmons of the, 241.
syphilis of the, 244.
tuberculosis of the, 244.
Phlegmons in syringomyelia, 572.
differentiation from erysipelas, 895.
Phosphate stones in the urine in nephrolithiasis, 389.
Phosphaturia in meningitis, 705.
Phosphorus poisoning, fatty heart in, 53; fatty liver in, 194.
liver atrophy and, 180.
Phrenic nerve, compression of the, by mediastinal tumours, 149.
neuralgia of the, 454.
paralysis of the, 483; bilateral, 484; unilateral, 484.
spasm of the, 500.
Phthisis pulmonum, 130.
differentiation of, from pneumonia (croupous), 126.
florida, 134.
mixed forms of syphilitic and tuberculous, 145.
Pia mater of the spinal cord, inflammation of the, 529.
due to the virus of cerebro-spinal meningitis, 531.
in its cervical portion, 531.
in its lumbo-dorsal portion, 531.
Pigmentation of the skin in Addison's disease, 405.
in Graves's disease, 761.
in unilateral progressive atrophy of the face, 754.
Pineal gland, tumours of the, 681.
Plague, 1004; bubonic, 1004.
ætiology of, 1004.
clinical picture of, 1005.
diagnosis of, 1005.
pathology of, 1004.
Plaques muqueuses of the pharynx, 244.
Plethora, serous, in renal affections, 353, 357.
Pleura, affections of the, 153.
cancer of the, 168; and mediastinal tumours, 151.
tumours of the, 168.
Pleural exudate, bacteriological findings of, 162.
condition of, 161.
diagnosis of, 158.
echinococcus of the liver and, 205.
pulmonary atelectasis due to, 108.
symptoms of, 156, 157.
Pleurisy, aortic aneurysm, differentiation from, 160.
auscultatory results of, exudative, 157; sicca, 154.
dry, 153; and intercostal neuralgia, 158, 455.
due to anthrax infection, 996; due to septic infection, 967.
exudative, 155.
friction sound in, 154; differentiation from crepitation, 154; from dry râles, 154.
hæmothorax and, 160.
hepatic and splenic tumours and, 160.
hydrothorax and, 161.
idiopathic, 162.
in acute articular rheumatism, 974.
in measles, 867.
in mediastinal tumours, 151.
in pulmonary tuberculosis, 133.
in scarlatina, 876.
in small-pox, 886.
in typhus fever, 898.
mediastinal and pleural tumours and, 159.
muscular rheumatism and, 158.
pectoral fremitus in, 156.
percussory results in exudative, 155; in sicca, 153.
peripleuritic abscess and, 160.
pneumonia and, 159.
pulsating, 155.
purulent, in erysipelas, 894; in influenza,

987; in typhoid fever, 917; producing acute spinal meningitis, 531.
Pleurisy, tuberculous, in acute miliary tuberculosis, 956.
Pleuropericarditis, 46.
Plexus brachialis, neuralgia of the, 454; paralysis of the, 484, 485, 486, 487, 488, 489, 490, 491; spasms in the region of the, 501.
cardiacus and cœliacus, affections of the, in multiple neuritis, 508.
coccygeus, neuralgia of the, 462.
lumbalis, neuralgia in the region of the, 456; spasms in the region of the, 502.
pudendal, neuralgia of, 462.
sciatic (of the sciatic plexus) neuralgias, 458; paralysis, 494.
Pneumatosis of the stomach, 305.
Pneumococci, 125; epidemic cerebro-spinal meningitis due to infection of the meninges with, 708, 952.
in pleuritic exudates, 162; mixed infection in diphtheria, 941; in influenza, 985; in septicopyæmia, 964.
Pneumogastric nerve (see Vagus).
Pneumonia, caseous, 130.
catarrhal, 126; ætiological factors, 126; and capillary bronchitis combined, 93; and croupous, 127; differential diagnosis, 127; from atelectasis of the lungs, 128; from miliary tuberculosis, 127; in measles, 866; in mumps, 940; in small-pox, 886; in trichinosis, 999; in typhus fever, 898; predisposition of children and of the aged, 127; results of physical examination in, 127.
central, differentiation from enteric fever, 925.
common seat of, 122; infiltration of the lungs in, 122; lobular, 127; tubercular and, 126; inspection in, 123; intermittent fever in, 124; micro-organisms in the sputum in, 121; percussion results in, 122; pleurisy and, 125, 159; pulmonary œdema and, 126; rusty sputum in, 121; symptoms of, 123; typhoid and meningitic, 125; voice examination in, 123; in slightly developed infiltration, 122.
Pneumonia, croupous (fibrinous), 121; abortive forms of, 125; auscultatory symptoms, 123; circumference of pneumonically infiltrated lungs, 123; complications of, with acute articular rheumatism, 974; with gout, 847; with influenza, 987; with meningitis, epidemic, 951, 955; erysipelatous, 894; purulent, 710; with spinal, 531; differential diagnosis, 125; hæmorrhagic infarct of the lungs in, 126; hypostatic, 109; of typhoid fever, 917.
Pneumonia, interstitial, 128; ætiological factors, 128; auscultatory conditions in, 129; circulatory disturbances in, 129; contraction of the lungs in, 128; percussion in, 129.
lobular, due to septic infection, 967; in enteric fever, 917.
masked, 125.
serous, 126.
wandering, and erysipelas, 894.
Pneumopericardium, 47.
Bacillus aerogenes capsulatus in, 48.
auscultatory symptoms in, 47.
causative factors of, 47.
differential diagnosis, 48.
percussory phenomena, 47.
Pneumoperitonitis, 413.
condition of the percussion sound in the axillary line in, 414.
disappearance of liver dulness in, 413.
Pneumothorax, 102.
ætiology, 167.
auscultation in, 163.
bilateral, 162.
closed, 166.
free, 164.
gastrectasis and, 164.
in whooping-cough, 948.
large cavities and, 164.
Pneumothorax, open, 165.
percussion in, 163.
pneumopericardium and, 164.
pulmonary cavities and, 138; emphysema and, 115.
pyopneumothorax hypophrenicus, 164.
sacculated, 166.
valvular, 166.
Pneumotyphus, 917.
diagnosis of, 925.
Podagra (see Gout).
Poikilocytosis of the blood of anæmics, 779.
Points douloureux of the nerves, 446.
Polioatrophia, anterior chronica progressiva, 563.
anatomical findings in, 563.
course of the atrophy of the muscles in, 564.
differential diagnosis of, 566.
symptoms of, 564, 565.
Polioencephalitis, chronic superior, 616, 696.
Poliomyelitis anterior, 558.
acute infantile, 558; acute, of adults, 561.
bulbar symptoms of, 614.
chronic (subacute), 558, 562; chronic progressive, 563.
differential diagnosis of acute, 560; of chronic, 562; from multiple neuritis, 509, 561, 563; of chronic progressive, 566.
pathological picture of acute, in adults, 561; in children, 559.
posterior, symptoms of, 570.
Polyarthritis, acute, 970.
complications with endocarditis and pericarditis, acute, 974.
differential diagnosis of, 975.
epidemic occurrence of, 970.
heart defects due to, 974.
in diphtheria, 944.
infectious nature of, 970.
in scarlatina, 874.
symptoms of nervous, 971; on the part of the skin, 974.
Polychromatophilia in pernicious anæmia, 779.
Polydipsia, primary and symptomatic, 938.
Polymyositis, acute, 766.
diagnosis of, 766; differential, from polyneuritis, 770; from trichinosis, 769, 999.
subacute, 767.
Polyneuritis, 503, 504.
ætiological factors, 505.
differential diagnosis of, 509; from acute myelitis, 578; from anterior poliomyelitis, 561, 563; from polymyositis, 770.
symptoms of, 505, 506; rare, 508.
Polypi, mucous, of the larynx, 79.
of the œsophagus, 246.
of the rectum, 330.
Polyuria, chronic symptomatic, 839.
differentiation of symptomatic from diabetes insipidus, 837, 838.
epicritic, 838.
in acromegaly, 757; in contracted kidney, 362; in diabetes insipidus, 837; in diabetes mellitus, 827.
in epidemic cerebro-spinal meningitis, 950.
in Graves's disease, 761.
in hysteria, 730.
secondary, in polydipsia, 837; in syringomyelia, 572.
subsequent to epileptic attacks, 740.
Pons, diseases of the, 606.
degeneration of the motor conduction tract after, 551, 554.
diagnosis of, 606; differential, from basal tumours of the brain, 609; from oblongata affections, 608, 614; facialis paralysis due to, 476, 479, 606, 609; form of paralysis in, 609; hypoglossus paralysis due to, 482; symptoms of, 608, 609.
Pons Varolii, anatomical structure of, 602.
blood-vessels of, 605.
compression of, 617.
hæmorrhages of, 608, 610.
softening process due to emboli and thrombi, 610.

Porta hepatis, cancer of, and cancer of the pancreas, 224.
tumours of, and their action upon the bile ducts, 211.
Portal-vein circulation in the liver, 170.
stasis in amyloid liver, 196; in cirrhosis of the liver, 182; in pancreas affections, 223; in pylephlebitis, 219; in retention icterus, 212; in syphilis of the liver, 188.
Posterior columns of the spinal cord, degeneration of, 520; in hereditary ataxia, 550; in tabes dorsalis, 537, 540; diseases of, and symptoms, 526; fibre systems of, 514, 521; nuclei of, 596, 621; structural conditions of, in the medulla, 596.
horns, affections of, and their symptoms, 526, 571.
Posticus, paralysis of the laryngeal muscles, 88.
bilateral, 88.
differential diagnosis of, from laryngospasm, 82.
unilateral, 88.
Præsystolic murmur in mitral stenosis, 17.
Pregnancy, ætiological relation to multiple neuritis, 86.
Pressure pain at the infra-orbital foramen in supramaxillary neuralgia, 452; at the mental foramen in ophthalmic neuralgia, 451; in lumbo-abdominal neuralgia, 456; of the inflamed nerve trunks, 503; of the muscles in myositis, 766; of the skin and muscles in spinal meningitis, 530; of the spinal cord in compression myelitis, 580.
paralysis, spinal, 579; ætiological diagnosis of, 581; differentiation from tabes, 547; inflammatory, 580; symptoms of, motor, 579; sensory, 579.
points of the nerves, 435; points to relax spasms, 497.
sensation, tracts of, in the spinal cord, 521.
ulcer of the intestine, 325; of the larynx, 74.
Priapism of leucæmics, 794.
of tabetics, 538.
Proctitis, 315.
stools in chronic, 317.
tenesmus in, 315.
Proctospasm, 341.
Projection centres of the cerebral cortex, 639.
Prolapsus ani due to pertussis, 948.
Propulsion and retropulsion of paralytics, 752.
Prosopalgia, 451.
complications of, 451.
Proteins, phlogistic and pathogenic, 963.
Proteus fluorescens in Weil's disease, 214.
Protozoa, ætiological significance of, in small-pox, 881.
Pseudo-apoplexy in fatty heart, 52.
arthrosis in spinal infantile paralysis, 560.
bulbar paralysis, 615; cerebellar form, 615; cerebro-bulbar form, 615.
croup, 69; and laryngeal croup, 70.
diphtheria bacilli, pseudo-, pathogenic action of, 941.
hypertrophy of the muscles in dystrophia muscularis progressiva, 568.
leucæmia, 802; anæmia and hæmorrhagic diathesis in, 802; blood picture in, 802; diagnosis, 802; relation of, to leucæmia, 803.
ligaments, pseudo-, cause of intestinal obstruction, 337.
sclerosis of the spinal cord, 594.
Pseudo rhachitis senilis, 854.
Pseudo-tabes diabetica, 831; diagnosis of, 547; of the hæmorrhoidal, 547; of the peripheral (potatorum), 507, 546.
Psilosis, 315.
Psoas abscess, differentiation of, from paranephritic abscess, 373.
spasms, 502.
Psychical disturbances after epileptic attacks, 740.
in anæmia, 782; in brain tumours, 682; in chorea minor, 744; in cryptogenetic septicopyæmia, 967; in diabetes mellitus, 831; in diphtheria, 945; in enteric fever, 918; in focal diseases of the centrum ovale, 663; in Graves's disease, 760; in hydrocephalus, 720; in hysteria, 726; in influenza, 987; in miliary tuberculosis, acute, 957; in multiple sclerosis, 591; in myxœdema, 758; in neurasthenia, 732; in paralyses, central, 434; progressive, 697, 698; in rabies, 993; in rheumatism, acute, 972; in small-pox, 884; in tetany, 749; in traumatic neurosis, 734; in typhus fever, 899; in uræmia, 359.
Psycho-reflexes in central facialis paralysis, 474.
Psychoses in the course of polyneuritis, 508.
relation of hysteria to, 722, 724; pachymeningitis hæmorrhagica, 534; traumatic neurosis, 734.
Pterygoidei, paralysis, 467.
spasm, 496.
Ptosis in oculomotor paralysis, 465.
Puerperal fever, generator of the infection in, 894.
purulent meningitis in, 710.
Pulmo-excessivus and pulmonary emphysema, 115.
Pulmonary affections and their genetic relations to osteoarthropathy, 757; due to anthrax, 996; due to measles, 866; following scarlatina, 876; following typhus fever, 898; in cryptogenetic septicopyæmia, 967; in diabetes, 829; in influenza, 987; in leucæmia, 794; in whooping-cough, 949; tuberculous, in acute miliary tuberculosis, 956.
Pulmonary apoplexy, 140.
abscess due to septic infection, 967; abscess in influenza, 987; abscess of enteric fever, 917.
Pulmonary artery, dilatation of, in persistence of the ductus Botalli, 37; double sound of, in pulmonary valve insufficiency, 29; embolism of, 139; diagnostic significance for sinus thrombosis, 718; in mitral stenosis, 18; narrowing peripherally from the valves, 30.
emphysema due to cough paroxysms in pertussis, 949.
stenosis, 29; congenital, 29; cyanosis in, 29; differential diagnosis, 29; from persistence of the ductus Botalli, 29; in myocarditis, 49; symptoms of, 29.
Pulmonary tissue, diseases of the, 106; in gangrene of the lungs, 143; in the sputum in abscess of the lungs, 142.
Pulmonary valves, insufficiency of, 29; sounds, accentuated second, in endocarditis by dilatation and hypertrophy, 8; by mitral insufficiency, 14; by mitral stenosis, 18; by myocarditis, 49; by pulmonary emphysema, 113; by pulmonary atelectasis, 107; by patulous ductus Botalli, 37; in peripheral narrowing of the pulmonary arteries from the valves, 30; weakness of the second sound in pulmonary stenosis, 29.
Pulsation of aneurysm, aortic, 64.
diffuse, in mitral insufficiency, 12.
in mediastinal tumours, 152.
in mitral stenosis, 16.
systolic, in pleurisy with effusion, 155.
Pulse, Corrigan's, 21; collapsing, 21; in acute articular rheumatism, 971; in anæmia, 784; in angina pectoris, 57, 58; in aortic aneurysm, 66; in aortic insufficiency, 21; in acute stenosis, 27; in apoplectic insult, 664; in arteriosclerosis, 62; in asthma, cardiac, 60; in atheroma of the coronary arteries, 63; in bilateral paralysis of the accessorius, 481; in brain abscess, 692; in brain tumour, 682; in cholera Asiatica, 931; in dysentery, 929; in enteric fever, 910; in examination of the, vii; in fatty heart, 52; in gastric catarrh, 265; in Graves's disease, 759; in heart disease, 4; in hypoglossus paralysis, 483; in jaundice, 206; in

liver atrophy, 177; in meningeal hæmorrhage, 719; in meningitis, 703; in epidemic cerebro-spinal, 951; in spinal, 951; in spinal, 530; in miliary tuberculosis, acute, 966; in mitral insufficiency, 14; in mitral stenosis, 18; in multiple neuritis, 506; in muscular activity, 54; in myocarditis, 48; in nephritis, 354, 358; in pericarditis, 43; in peritonitis, 408; in persistence of aortic isthmus, 38; in pharyngeal diphtheria, 942; in pleurisy, 157; in pneumonia, 124; in progressive bulbar paralysis, 614; in pulmonary hypostasis, 109; in pulmonary œdema, 119; in septicopyæmia, 965; in small-pox, 881; in tetanus, 991; in vagus paralysis, 480; in venous, 2.
Pulse. Quincke's, 23.
water-hammer, 21.
Pulsions diverticulum, 252; seat of, 252.
sound for diagnosis, 253.
Pulsus altus et celer, in aortic insufficiency, 21.
differens, in aortic aneurysm, 66.
paradoxus, in mediastino-pericarditis, 46.
tardus, in aortic stenosis, 27.
Pupillary reflex, centre for, 524, 605.
Pupils in apoplectic insult, 664; in chorea, 745; in epileptic attacks, 739; in lesions of the corpora quadrigemina, 633; in meningeal hæmorrhages, 719; in meningitis, 704; epidemic cerebro-spinal, 951; spinal, 530; in multiple sclerosis, 590; in oculomotorius paralysis, 465, 466; in tabes dorsalis, 537, 549.
rigidity, reflex, in progressive paralysis, 697; in tabes dorsalis, 537, 549.
Purpura, 807.
diagnosis of, 809.
epidemic occurrence of, 810.
essential (idiopathic), 810; predisposing factors, 811.
fulminans, 808.
hæmorrhagic, 807; course of, 808; prodromes, 811.
infectiousness of, 810.
origin of, 810.
rheumatic, 810, 975.
simplex, 808.
symptomatic, 808.
urticans, 808.
variolosa, 886; diagnostic symptoms, 886; differential diagnosis, 889.
Pus cocci in the sputum in pulmonary abscess, 142.
in the renal pelvis, 355.
in the stools in intestinal cancer, 326; in intestinal ulcers, 320.
in the urine in cystitis, 394; in nephrophthisis, 388; in suppurative nephritis, 367, 370.
Pustule, malignant, 994.
Putrefactive processes in the stomach, effect of HCl upon, 257.
Pyæmia, character of, 964.
in the course of enteric fever, 916.
metastatic suppurations in, 964.
origin of, due to pyæmic micro-organisms, 964.
Pyelitis and cystitis, 396.
suppurative, and renal abscess, 372.
Pyelonephritis, 367.
urine in, 367.
Pylephlebitis, 219.
differentiation from hepatic abscess, 220.
in hepatic cirrhosis, 219.
symptoms, 219.
Pylethrombosis, 219.
diagnosis, 219.
hepatic cirrhosis and, 219.
symptoms, 219.
Pylorus, cancer of the, 281; and carcinoma of the gall-bladder, 284; of the liver, 201, 284; of the pancreas, 224; wandering spleen, 233.
insufficiency of the, nervous, 307.
spasmodic closure of, 307.
tumours of, and wandering kidney, 392.
Pyopneumothorax and pneumothorax, 166.
sacculated and bronchiectasis, 101.
subphrenic and pleural exudate, 164.
Pyramidal tracts, affections of, 518, 525.
course of, in the brain, 518, 626, 635, 648; in the spinal cord, 514, 515.
decussation of, in the medulla oblongata, 518, 596.
degeneration of, 518, 525.
motor conduction of, 518.
nerve units of, 518, 552.
Pyuria, examination of the urine in, 347.

Q

Quincke's pulse, 23.
Quintus (see Trigeminus) (fifth nerve).

R

Rabies, 993 (also see Hydrophobia).
attacks of rage in, 994.
atypical, 994.
beginning of, 993.
diagnosis of, 993; differential, from pseudo rabies, 994.
hydrophobic stage in, 993.
incubation period, 993; in tetanus, 993.
individual predisposition to, 993.
origin of, 993.
Pasteur's treatment of, 994.
picture of, 993.
psychical excitation in, 994.
Radial nerve paralysis, 486.
cerebral, 487.
diagnosis of, 488; ætiological factors for the, 486; details, 488.
position of the hand and fingers in, 488.
saturnine, 487; localization of, 487.
spinal, 487.
symptoms of, 488.
Radiation, strio-thalamic, 635.
Rag-picker's disease, diagnosis of, 997.
origin of, due to inhalation of anthrax bacilli, 996, 997.
Railway brain, 734.
spine, 579, 734.
Râles and friction sounds, 154.
cracked-pot sound in pleurisy, 156; in pneumonia, 122; in pneumopericardium, 47; in pneumothorax, 163; over bronchial cavities, 100.
falling, ringing drop, in pneumopericardium, 47; in pneumothorax, 163; over pulmonary cavities, 137.
"forcing through sound" in œsophageal stenosis, 248.
gurgling, in intestinal catarrh, 308.
in bronchial asthma, 103; in bronchiectasis, 100; in bronchitis, 90; in pneumonia, 123, 127; in pneumothorax, 163; in pulmonary abscess, 142; in pulmonary œdema, 118; in pulmonary tuberculosis, 132, 137.
sibilant and sonorous, in bronchitis, 90.
sibilant, in bronchial asthma, 103.
splashing, in acute colitis, 315; in gastrectasis, 288.
Rash in small-pox, 881.
Raspberry tongue (cat's tongue) in scarlatina, 874.
Raynaud's disease, 756.
nervous character of, 765.
Reaction of degeneration, 429.
complete, 429.
course of, 429.
diagnostic significance of, 431; of myelitis, 574, 575; of neuritis, 504; multiple, 506; partial, 429; of paralyses by pressure, 580; central, 432; peripheral, 430, 432, 433.
in amyotrophic lateral sclerosis, 556.
in disease of the anterior horns of the spinal cord, 525, 572.
in poliomyelitis, anterior, 558; in progressive bulbar paralysis, 614; in progressive muscular atrophy, 565, 570; in saturnine radialis paralysis, 487.
symptoms of, 429, 430.

Reaction of degeneration, typical, 430.
Reading, analysis of, 655.
diagram presentation of the central tract of, 656.
disturbances of, in aphasia, 655; in progressive paralysis, 697.
Rectum, anæsthesia of, 345.
cancer of, 329; combination with hepatic cancer, 200; differential diagnosis, 330.
catarrh, chronic, 315.
examination of, with finger and speculum in proctitis, 315.
fistulæ of, tubercular, in phthisis, 139.
function of, disturbances in compression myelitis, 580; in diphtheria, 944; in lesion of the cerebral peduncles, 630; unilateral, of the spinal cord, 583; in meningitis, 703; in myelitis, 162; in neuritis, 508; in syringomyelia, 572; in tabes, 538.
hæmorrhoids in, 330.
nerves of the, paralytic phenomena in, 342, 344; spasm of, 341.
polypi of, 330.
spasm of, 341.
syphilis of, 324.
Recurrent nerve, differentiation of, from cephalalgia, 452.
paralysis of, bilateral, 84; incomplete bilateral, 84; isolated in muscles, 85; unilateral, 84.
pressure on, in aortic aneurysm, 68; in mediastinal tumours, 149.
Reflex arcs, cerebello-cerebral, 619; short and long, 428, 523, 524.
epilepsy, 742.
irritability in affections of the posterior horns and posterior nerve roots, 526; in anæmic conditions, 782; in anterior horn affections of the spinal cord, 525; in apoplectic attacks, 665; in epileptic attacks, 739; in affections of the cerebello-lateral column tract, 527; in focal affections of the internal capsule, 647; in hydrophobia, 993; in muscular atrophy, 565; in poliomyelitis anterior, 558; posterior, 571; in progressive bulbar paralysis, 614; in pyramidal anterior lateral column affections, 525; in spinal meningitis, 530, 533; in tabes dorsalis, 542; in tetany, 749; in tetanus, 990; of the facial nerve in central paralysis, 474.
movements of expression, sensation, centres of, 643.
neuralgia, 450.
spasms, 496; in hydrophobia, 993.
spino-bulbar-cerebral, 621.
Reflexes, action of certain poisons, 524.
centres of, in the medulla oblongata, 605.
decrease of, 523.
diagnostic significance of, 428.
distributed, 523.
in Brown-Séquard's paralysis, 582; in cerebral (central) paralysis, 433, 434; in conus affection, 585; in facialis paralysis (central), 474; (peripheral), 477; crossed, 523; in meningitis, 704; in myelitis, 574; in neuritis, 504; multiple, 506; in pressure of the spinal cord, 580; in spinal (peripheral) paralysis, 433, 434, 559, 462.
increase of, 428, 523, 552.
inhibition of, 428, 523, 540.
process of reflex activity, 427, 523, 524.
production of, 523, 527.
tracts of, in the spinal cord, 427, 523, 525.
Regurgitation of blood into right auricle and cava in tricuspid insufficiency, 30.
of food in œsophageal spasm, 245; stenosis, 247.
Relapsing fever, and Weil's disease, 213.
Relapsing fever, 900.
bedbugs carrying the disease, 901.
contagion of, 900.
course of, 902.
defervescence in, 903.
differential diagnosis, 906, 924.
paroxysms in, 902; number and duration of, 904.
Relapsing fever, formation and perishing of the pathogenic organism in the various periods of, 901.
incubation period of, 902.
infection in, 900; by the blood of relapsing-fever patients, 900.
paroxysms in, 902; number and duration of, 904.
prodromes of, 902.
septic-bilious, 905.
spirilli of, 901.
symptoms of, 904.
Renal tissue, particles in the urine in renal abscess, 367; in renal tuberculosis, 374.
Ren mobilis, 321.
Reno typhoid, 919.
Resorption by stomach, 258.
test of, 262.
Respiration, centres of, 605, 738.
in acute miliary tuberculosis, 956; in anæmia, 781; in apoplectic insult, 664, 665; in Asiatic cholera, 933; in diabetes, 829, 832; in diaphragmatic paralysis, 483; spasm, 500; in epileptic attacks, 738, 739; in Landry's paralysis, 587; in laryngeal diphtheria, 943; in leucæmia, 794; in meningitis, 703; epidemic cerebro-spinal, 951; spinal, 530.
in multiple sclerosis of the spinal cord, 590.
in osteomalacia, 850.
in progressive bulbar paralysis, 612, 613.
in whooping-cough, 946.
vesicular, diminished in bronchial asthma, 103; in bronchiostenosis, 96; in fibrinous bronchitis, 80; in pleurisy with effusion, 157.
Respiratory centre, relaxation of, 52.
disturbance in bronchiostenosis, 100; in capillary bronchitis, 92; in fatty heart, 52; in hæmorrhagic infarct of the lungs, 140; in laryngeal stenosis, 77; in myocarditis, 50; in pericarditis, 43; in peritonitis, 409; in pneumonia, 123, 127; in pulmonary emphysema, 110, 111; in tumours, mediastinal, 146, 147.
movements, abnormal, 500; effect of diaphragm paralysis, 483.
murmur in bronchial catarrh, 90, 95; in mediastinal tumours, 148; in pneumothorax, 163; in pulmonary tuberculosis, 132, 137.
muscles, acute myositis of, 767; paralysis after diphtheria, 943; spasm of, in tetanus, 990.
organs, diseases of, 68; inflammation of, in diphtheritic, 943; in anthrax, 996; in gout, 847; in influenza, 987; in measles, 866; in scarlatina, 876; in small-pox, 886; in typhoid fever, 917.
Retention jaundice, 175; causes of, 209, 210; differential diagnosis from hepatic cancer, 199; enlargement of liver from, 199.
of stomach contents in chronic gastritis, 268; in gastrectasis, 287.
of urinary constituents in uræmia, 359.
Reticular substance in the medulla oblongata, 603.
Retina, anæsthesia and hypersæsthesia of, in hysteria, 729.
œdema of, in thrombosis of the cavernous sinus, 717.
Retinal hæmorrhages in cryptogenetic septicopyæmia, 967.
Retinitis albuminurica, 786; in contracted kidney, 363.
diabetica, 831.
leucæmia, 794.
Retroperitoneal glandular tumours and mesenteric cysts, 421; and renal tumours, 379.
compression of biliary passages by, 212.
Retropharyngeal abscess, 241.
in enteric fever, 916.
Retroversion of the liver, 206.
Rhachitic rosary, bone changes in, 853.
Rhachitis, 852.
anatomical changes of the bones in, 852.

Rhachitis, characteristics, 852, 853.
complications of, 853.
deficient calcification of the osteoid tissue in, 852.
differential diagnosis of, from, 854; changes of the epiphyses due to hereditary syphilis, from, 854; hydrocephalus from, 854; kyphosis due to tubercular processes, 854.
infectious nature of, 853.
of the pelvis, 853.
of the skull, 852.
of the thorax, 853.
prodromes of, 852.
relation of, to tetany, 750.
sensitiveness to pressure of rhachitic bones, 852.
symptoms of, 852, 853.
teeth perforation in rhachitic children, 853.
Rheumatism, acute articular, 970; cerebro-spinal meningitis in, 972; complications of, 974; differential diagnosis from gonorrhœal inflammation, 975; from gout, 975; from hysteric articular neurosis, 975; from peliosis rheumatica, 975; from septicopyæmia, 968, 975; from spinal meningitis, 976; in the joints of the foot and knee, 971; infectious nature of, 970; masked, 976; meningitis spinalis in, 532; of the vertebral articulations, 976; prodromes of, 971; sequelæ of, 974; symptoms of, 971, 972; cerebral, 972; virus of, 970.
chronic articular, 849; differentiation from arthritis deformans, 849; from osteomalacia, 851; changes of the joint articulations and articular ankylosis in, 849; origin and diagnosis of, 849.
muscular, 765; and pleurisy, 158; chronic, 765; clinical picture of, 765; diagnosis, 766; differential, from intercostal neuralgia, 455, 765; from sciatica, 450; from tetanus, 992; infectious nature of, 766; nature of, 766; of the muscles of the chest, 765; of the loins, 765; of the neck, 765; and back, 765; differentiation from spinal meningitis, 532.
nodosus, 975.
Rheumatoid pains, differential diagnosis from neuralgias, 447.
of the lower extremities, differentiation from tabes, 546.
of the neck and back of the neck, differentiation from spinal meningitis, 532.
Rhomboidei, spasm of, 499.
Ribs, disease of, and pleurisy, 158.
Rice-water stools in Asiatic cholera, 934.
comma bacillus in, 935.
Risus sardonicus of tetanus patients, 990.
Rock fever, 1004.
Romberg's symptom in polyneuritis, 507.
in tabes dorsalis, 539.
Rose, diagnosis of, 891.
differential, 895.
Rötheln, 879.
contagiosity of, 879.
diagnosis of, 880; differential diagnosis from measles, 870, 880; from scarlatina, 879, 880.
exanthem of, 879.
immunity after, 879.
incubation period, 879.
symptoms of, 879, 880.
Rubella (rötheln), 865, 879.
Rubeola, diagnosis of, 863.
Rumination, 307.
Rupture of œsophagus, 253.
of spleen, 231.
spontaneous, of heart, 53.

S

Sacral nerves, neuralgias in the region of, 458; paralyses in the region of, 492; spasms in the region of, 502.
pains in the prodromal stage of smallpox, 881, 887.
Salaam spasm, 497.
Saliva, reaction and secretion in diseases of the internal organs, 235.
secretion of, centre for, 604; disturbances of, in facialis paralysis, 470, 472; in hysteria, 732.
Salivation in facialis spasm, 497.
in Graves's disease, 761.
in pancreatic stone colic, 226.
in progressive bulbar paralysis, 614.
in stomatitis mercurialis, 236.
in trigeminus neuralgia, 451, 453.
paroxysms of, in place of epileptic attacks, 740.
Sarcomata of the kidney, 387.
of the larynx, 80.
of the liver, and carcinoma, 200.
of the mediastinum, 153.
of the peritonæum, 419.
of the pleura, 168.
of the spleen, 232.
of the stomach, 286.
Sarcomata in the spinal-cord canal, 584.
of the intestines, 330.
of the skull bones, pressure effect upon the brain, 690.
Scarlet fever, 870.
ætiological factors of, 870.
anomalies of the course of, 876.
complications of, with diphtheria, 875; with diseases of the eyes and ears, 876; with endocarditis, 875; with gastritides, 876.
contagiousness of the toxine of, 870.
defervescence in, 874.
desquamation in, 874.
diagnosis of, 874; differential from erysipelas, 878; from erythema, 878; from measles, 878; from rötheln, 879; from septicopyæmia, 970; from typhoid fever, 925.
diphtheria and, 268.
dropsy in, 354.
eruption in, 872; varieties of, 873.
eruptive stage, 872.
hæmorrhagic, 873.
immunity to scarlatinal infection, 871; due to inoculation with the toxine, 871.
incubation period of, 870.
lævigata, 873.
miliaris, 873.
nephritis in, 353, 874.
papulosa, 873.
predisposition to, 870.
prodromal stage, 871.
relapses, 871.
sequelæ, 876.
sine angina, 876; exanthemate, 877.
symptoms of, 871, 872, 873; unusual, 875.
variegata, 873.
Sciatic nerve, paralysis of, 492.
diagnosis and symptoms, 492.
Sciatica, 458.
central, 461.
diagnosis of, 458; ætiological, 460; differential, 459; from tabes dorsalis, 461, 546.
distribution of, in the leg, 458.
exploration of the rectum in examination for, 461.
involvement of the contralateral sciatic nerve in, 459.
peripheral, 458.
spasms of the calves of the leg in, 503.
symptoms of, 458, 459.
urinary examination for sugar, 461.
Sclerema of the skin in Graves's disease, 761.
Scleritis migrans in gout, 846.
Sclero-keratitis of gouty patients, 846.
Sclerose en plaques, cerebral hæmorrhage in, 674.
with bulbar symptoms, 614.
Sclerosis, multiple cerebro-spinal, 589, 697.
anatomical changes in, 589.
atypical, 594.
cases with negative findings, 594.
course of, 593.
diagnostic symptoms of, 589, 590; cerebral, 591; motor, 589; on the part of the eyes,

590; of the respiration, 590; of the speech, 590; sensory, 591.
Sclerosis, differentiation from amyotrophic lateral sclerosis, 557, 592; from cerebral tumours, 689; from hereditary ataxia, 551; from hysteria, 593; from paralysis agitans, 593, 752; from tabes dorsalis, 548, 592.
in children, 593.
insular, 589.
occurrence of sclerotic areas, 589.
propagation of the process, 593.
Scoliosis due to poliomyelitis anterior, 560.
due to sciatica, 459.
in hereditary ataxia, 550.
Scotoma, central, in multiple sclerosis, 590.
Scurvy, 808.
diagnostic symptoms, 808.
of the intestines, 325.
of the oral cavity, 327.
Secretion, anomalies of, in anæmia, 785.
in facialis paralysis, 471, 473.
in Graves's disease, 761.
in hysteria, 729.
in neuralgia, 446.
in neurasthenia, 733.
in progressive bulbar paralysis, 614.
in syringomyelia, 572.
in tetany, 749.
in trigeminus neuralgia, 451, 452.
Sedimentum lateritium in the urine of pneumonic patients, 124.
Senses, special, centres for, 639, 642, 643.
irritable weakness of, in hysteria, 723.
Sensibility, cortical centres of, and their association, 642; conduction of the various qualities in the spinal cord, 521.
disturbances of, 436; after diphtheria, 945; in Brown-Séquard's paralysis, 583; in capsule disease, 646; in cerebral hæmorrhage, 667, 675; in cerebral tumours, 684; in cruralis paralysis, 493; in disease of the posterior horns and posterior roots of the spinal cord, 526; in facialis paralysis, 471, 474; in focal disease of the central convolutions, 650; of the centrum ovale, 662; of the pons medulla, 607, 609; in hysteria, 725, 727; in Landry's paralysis, 587; in meningeal hæmorrhages, 719; in multiple sclerosis, 591; in myelitis, 574; in neuralgias, 446; in neurasthenia, 732; in neuritis, 503; in multiple neuritis, 505, 509; in obturatorius paralysis, 493; in pressure paralysis (spinal), 579, 580; in radialis paralysis, 489; in sciatic nerve, 494, 495; in syringomyelia, 571; in tabes dorsalis, 537, 538, 545; in tetany, 749; in traumatic neurosis, 734; in ulnaris paralysis, 489.
paralyses, partial, 436, 442; in central anæsthesia, 442; in myelitis, 574; in neuritis, 503; in syringomyelia, 571; in tabes dorsalis, 545.
Sensory disturbances, hysteric, 727; in anæmic conditions, 782; in brain tumours, 682; in capsule affections, 646; in cerebral anæmia, 700; in relapsing fever, 904; in traumatic neurosis, 734.
neuroses of the bladder, 403; of the intestine, 342; of the larynx, 81; of the rectum, 344; of the stomach, 207.
Septicæmia, differentiation from cerebral hæmorrhage, 674.
generators of infection in, 963, 964.
in anthrax, 995.
Septicopyæmia, ætiological points in, 963; and uræmia, 361; and Weil's disease, 213.
cause of suppurative nephritis, 372.
cerebral suppuration due to, 695.
cryptogenetic, 963; diagnosis, 965; differential, from acute articular rheumatism, 968; from acute miliary tuberculosis, 969; from enteric fever, 969; from malaria, 968; from meningitis, 970; from scarlatina, 970; from uræmia, 969; from variola, 970; following measles, 867; mode of infection in, 963; nature of, 963; pathogenesis, 963; spinal meningitis due to, 531; symptoms of, 965, 966.
Sero-diagnosis of enteric fever, 920.
Serratus paralysis, ætiological factors, 485.
diagnosis of, 485.
Sexual disturbances, causing hysteria, 724.
function, disturbances of, in diabetes mellitus, 831.
in multiple neuritis, 508; in multiple sclerosis of the spinal cord, 591.
in myelitis, 576.
in neurasthenia, 733.
in tabes dorsalis, 546.
Shoulder-blade, muscles of the, paralysis of, 485.
spasm of, 499.
Shoulder pain in abscess of the liver, 189.
Simulation of epileptic attacks, 743.
traumatic neurosis, 735; singultus, 500; in dysentery, 929; in hysteria, 726; in liver affections, 500; in mediastinal tumours, 149; in pericarditis, 41; in peritonitis, 408; in pons-medulla affections, 608.
Sinus thrombosis, 714.
focal symptoms of, 715.
marantic, 714, 716.
of the sinus cavernosus, 717; of the sinus longitudinalis, 717; of the sinus transversalis, 716.
origin of, 691, 714.
suppurative, 716.
Skeleton, changes of the, in osteomalacia, 850.
in rhachitis, 852, 853.
Skin, affections of the, erysipelatous, 892; in acute articular rheumatism, 974; in acute miliary tuberculosis, 957; in anthrax, 996; in cholera, 933; in cryptogenetic septicopyæmia, 966; in diabetes mellitus, 829; in glanders, 998; in Graves's disease, 761; in hæmorrhagic diathesis, 807; in hysteria, 729; in intermittent fever, 900; in measles, 865: in meningitis, 704; epidemic cerebro-spinal, 950; spinal, 530; in myelitis, 575; in myositis acuta, 767; in neuritis, 503; in pachymeningitis hypertrophica, 534; in poliomyelitis anterior, 558; chronica progressiva, 565; in relapsing fever, 904; in syringomyelia, 571; in typhoid fever, 918; in typhus, 897; in unilateral progressive facial atrophy, 755; phlegmonous, differentiation from erysipelas, 895; subsequent to scarlatina, 873.
Skin, burns of the, causing intestinal ulcers, 322; causing nephritis, 355.
colour of the, in Addison's disease, 405; icteric, 207; in fibrinous pneumonia, 124.
conduction resistance, electric, in Graves's disease, 760.
emphysema of, in whooping-cough, 948.
examination of the, in taking the history of a case, vii.
itching of the, in contracted kidney, 363; in jaundice, 207; in nephritis acuta, 354.
reflexes in anæsthesia (peripheral), 442; in apoplectic insult, 665; in capsular disease, 647; in myelitis, 575; by pressure, 580; in progressive muscular atrophy, 565; in spinal meningitis, 530; in syringomyelia, 571; in tabes, 542; in traumatic neurosis, 735.
sense perception of the, decrease of, 442; examination of the, 444; in compression of the spinal cord, 575; unilateral, 577; in lesion of the optic thalamus, 644; in neuritis, 503; in pachymeningitis hypertrophica, 534; in tabes dorsalis, 542, 545; increased, 445, 446; nerve districts of the head, 451, 452; in the extremities, 442, 443, 456, 457.
Skolikoiditis (Nothnagel), 311 (see Appendicitis).
Small-pox, 880.
abortive forms of, 885.
ætiological factors, 880.
complications of, 886.
confluent, 885.
course of, 884.
diagnosis of, 880, 887; abortive variola, 885.
differential diagnosis of, 887; from enteric

fever, 887, 925; from glanders, 888; from measles, 869, 888; from scarlatina, 887; from septicopyæmia, 888, 970; from typhus fever, 888; from varicella, 890.
Small-pox, eruptive stage, 882.
exanthem in, 882.
exsiccation stage, 883.
incubation stage, 880.
nature of the, virus, 880; of the mucous membranes, 882.
prodromal stage, 880.
sequelæ, 886.
source of contagion in, 880.
suppuration stage, 882.
symptoms of, 881, 882, 883.
umbilication of the pustule of, 882.
Smegma bacilli in urine, and tubercle bacilli, 375.
Smell, anæsthesia of, diagnosis of, 437; of central, 437; of peripheral, 437; with tabes dorsalis, 545.
hyperæsthesia of, 445.
Sneezing, spasms of, 501.
hysteric, 726.
Somnolence in hepatic atrophy, 176.
in laryngeal stenosis, 90.
in myxœdema, 758.
Sound, gastric, description, and application of, in diagnosis of diseases of the stomach, 259, 288, 289.
Spasm of the extremities in amyotrophic lateral sclerosis, 555.
of the glottis, 82.
of the muscles of the arm in cervicobrachial neuralgia, 454.
of the œsophagus, 249, 255.
of the sternocleidomastoideus, 497.
of the trachea and bronchi, 98.
of the trapezius, 498.
origin of, 496.
reflex, 496; in hydrophobia, 993.
seat of, 496.
tonic, 496; intermittent in tetany, 749.
Spasms, 495.
anatomical position of the centre for, 605.
cataleptic, 496.
clonic, 496.
convulsive, 496, 572.
co-ordinate, 496, 502.
diagnosis of, 496; of co-ordinate, 502.
epileptic, 739; experimental production of, 737; in embolism, 677; in tetany, 749.
functional respiratory, 83.
hysteric, 726; differentiation from spinal meningitis, 530; from tetanus, 990.
in angina pectoris, 57; in Asiatic cholera, 933; in atrophy of the liver, 176; in cerebral anæmia, 700; in compression of the spinal cord, 580; in meningitis, 703, 949; spinal, 530; in myoclonia, 748; in pontine affections, 608, 610; in syringomyelia, 572; in tetanus, 990.
in the regions of the cerebral nerves, 496; of the cervical nerves, 499; of the lumbar and sacral nerves, 502; of the plexus brachialis, 501.
Speculum, rectal, application of, in chronic proctitis, 315.
Speech, control of, tract of, 653, 654.
disturbances of, 651; forms of, 653; in abscess of the brain, 693; in facialis paralysis, 471, 473; in hereditary ataxia, 550; in hypoglossus paralysis, 481; in insular affections, 661; in lesions of the cortex, 651; in multiple sclerosis of the spinal cord, 590; in progressive bulbar paralysis, 612; in traumatic neurosis, 735; localization of, 659.
faculty of, development of, 652; localization of, 652, 653.
process of, 652, 653; acts of the, 654; centres (cortical), 659, 663; phases of the, 652, 653; tracts of the, 653, 654; subcortical, 660.
Sphincter, paralysis of the bladder, 401.
combined with detrusor paralysis, 402.
paralysis of the rectum, 342.
Sphincter, paralysis of voluntary sphincter innervation, 401.
spasm of the bladder, 402; combined with detrusor spasm, 402; dysuria and ischuria in, 402.
rectum, 344.
urinary discharge in paralysis of the bladder, 401.
Sphygmography of the pulse in aortic insufficiency, 21.
in aortic stenosis, 27; in arteriosclerosis, 62; in cardiac disease, 3; in mitral insufficiency, 14; in mitral stenosis, 18.
Spinal apoplexy, diagnosis of, 594; of the seat, 594; symptoms of, 594.
cord, abnormal smallness in hereditary ataxia, 550; anæmia of the, 594; canal, tumours of the, 583; cavity formation in the, 571; centres of the, 524; cerebellolateral column tracts in, 521, 542, 543; circulatory disturbances in the, 594; compression of the, 579 (see also Compression Myelitis and Pressure Paralysis); secondary, and tabes, 547; degeneration of the, 537; ascending secondary, of the posterior columns, 520; descending, 518; due to diphtheria, 944, 945; in the pyramidal lateral-column tracts, 551, 552; gray, 537; of the cerebello-lateral-column tracts, 521, 542, 550, 554; of the posterior columns, 537, 538, 550; of the anterior horn ganglion cells, 554; of the pyramidal tracts, 552, 555; primary and secondary, of the lateral columns, 552; clinical signs, 552; secondary, of the anterior column lateral tracts, 552; diseases of the, 512; consideration of anatomical structure and functions of the cord, 513; localization of, from symptoms, 525; of the spinal cord substance, 536; rules for the diagnosis, 513; system, 514, 515; the reflexes in, 522; fibre-column systems of the, 514, 516, 517; hæmorrhages, 594; causing degeneration, 552; paraplegia symptoms, 577; hyperæmia of the, 594; interruption of conduction, 579; lesions of the, degeneration symptoms, 552; meninges of the, diseases of the, 529; tumours of the, 583, 586; microscopical structure of the, 515; motor tract of the, 518; multiple focal affections of the, 521, 522; reflex tracts, 522; segments of the, functions of the, 527, 528, 585; position of, 528; in the vertebral column, 528; sensory tracts, 520; substance of the, 513, 514; system, diseases of the, 536, 553; combined, 550, 552; types of, 553, 554; tumours of the, 583, 586; anæsthesia due to, 583, 586; degeneration processes due to, 552; differential diagnosis of, from amyotrophic lateral sclerosis, 557; from syringomyelia, 573; intramedullary, 579, 582, 586; irritation symptoms in, 573; meningeal, 583; of the cauda equina, 584; pressure phenomena, 579, 582, 584; unilateral lesion of the, 582; due to pressure of spinal meningeal tumours, 582, 584; symptoms of, 582, 583.
Spinal ganglia, degenerative changes of, in tabes, 540, 542; point of origin of sensory nerve fibres, 520.
irritation, 547, 687.
Spirals in the sputum in asthma, 104.
in capillary bronchitis, 94.
Spleen, abscess of the, diagnosis of, 230.
cancer of the, 232.
changes of form and position of, 233.
diagnosis of diseases of the, 227.
dislocation of, in emphysema of the lungs, 112; in mediastinal tumours, 150; in pleurisy, 160.
embolism of the, origin of, 1, 230.
enlargement of the, in acute miliary tuberculosis, 956; in anæmic conditions, 786; in enteric fever, 911, 912, 917; in epidemic cerebro-spinal meningitis, 951; in parotitis, 938; in hæmoglobinuria, 814; in influenza, 986; in leucæmia, 793; in ma-

laria, 999; in pseudoleucæmia, 962; in relapsing fever, 902; in scarlatina, 974; in septicopyæmia, 967; in typhus fever, 897.
Spleen, examination of the, vii, 227.
hyperæmia of the, 228; hyperplasia of the, 228; in abscess of the liver, 230; in atrophy of the liver, 177; in hyperæmia of the liver, 152; in neoplasms of the liver, 186; in nervous dyspepsia, 296; in syphilis of the liver, 157; in Weil's disease, 213.
infarct of the, hæmorrhagic, 230; symptoms of, 230.
parasites of the, 232.
rupture of the, 231; in enteric fever, 917; in relapsing fever, 902.
syphilitic sclerosis of the, 232.
syphilomata of the, 232.
tuberculosis of the, 232.
tumour of the ætiological factors in the diagnosis of, 232; and abdominal tumour, 228; and gastric cancer, 228, 284; and pleural exudate, 160; and renal tumour, 378; chronic, due to malarial infection, 229; due to amyloid degeneration, 231; due to circulatory disturbances in the portal-vein system, 229; due to stasis in the portal-vein system, 229; due to unknown reasons, 230; infectious, 229; postcritical, in pneumonia, 124; respiratory displacement, 227; syphilitic and tuberculous, 232.
tumour from anomalies of metabolism, 230.
Splenic infarcts of typhoid patients, 917.
Splenitis, 231.
Splenius muscle spasm, attitude of the head in, 499.
Spondylitis cervicalis, differentiation from hypertrophic cervical pachymeningitis, 534.
gouty, 847.
rheumatic, 971, 976.
tuberculous, with secondary compression of the cord, 579; differentiation from tabes, 547.
Sprue, 315.
Sputum, blood in the same, in hæmorrhagic infarct, 140.
coctum et crudum in bronchitis, 90.
in bronchial asthma, 104.
in bronchiectasis, 99.
in capillary bronchitis, 93.
in laryngeal tuberculosis, 75.
in lung abscess, 142.
in mitral stenosis, 19.
in pulmonary gangrene, 143; œdema, 118; tuberculosis, 130.
micro-organisms in pneumonic, 121.
rusty, 121.
Staphylococci, occurrence and effect of, upon diphtheritic mucous membranes, 941.
septic infection due to, 963.
Staphylococcus infection in fibrinous bronchitis, 95.
in pneumonia, 122.
Stasis conditions, cerebral anæmia due to, 700; hyperæmia due to, 701; ecchymoses of the skin due to, 810; in sinus thrombosis, 716, 717.
œdema of the lungs, 119; differential diagnosis, 120.
phenomena in aortic insufficiency, 25; stenosis, congenital, 28; in bronchiectasis, 100; in bronchitis, 91; in cardiac disease, 1; in fatty heart, 51; in liver cirrhosis, 181; in mediastinal tumours, 148, 149; in myocarditis, 48; in pericarditis, 41; in pneumonia, interstitial, 129; in pulmonary atelectasis, 107; in emphysema, 112; in hypostasis, 109; in tuberculosis, 135; in stasis kidney, 348; in tricuspid insufficiency, 31; in upper half of the body in rupture of an aortic aneurysm into the vena cava, 66.
stus epilepticus, nature of, 740.
phosus, 897, 911.
Stellwag's sign, 760.
Stenocardia, 52.
ætiology, 58.
cause of attack, 58.
Stenosis, aortic, 28.
of the bronchi, 95.
of the intestines, 330.
of the larynx, 72.
of the mitral valve, 18.
of the œsophagus, 247.
of the pulmonary artery, 28.
of the tricuspid valve, 33.
Stenotic murmurs in bronchiostenosis, 97.
in mediastinal tumours, 148.
in pseudo-croup, 69.
Stercoral ulcers, 325.
Sternocleidomastoideus, paralysis of, bilateral, 488.
spasm of, clonic, 497; spasm of, bilateral, 496; spasm of, tonic, 496.
Stigmata, hysteric, 734.
Stitches in the side, in catarrhal pneumonia, 127.
in croupous pneumonia, 123.
Stomacace, 236.
differential diagnosis, 236.
Stomach, abscess of, symptoms, 288.
alteration in form of the, 291.
anæmia of the, 271.
anatomical structure and physiological activity of, 291.
artificial dilatation of the, with carbonic-acid gas or air, 289; congenital predisposition, 307; genuine nervous, 307; of anæmics, 271, 785; of diabetics, 836.
Stomach, cancer of the, 280; absence of free HCl in the stomach, 281; and aneurysms, intestinal cancer, lymph-gland tumours, 285; atony of, 290; atrophy of the mucous membrane, 271; duodenal cancer, 285; pancreatic cancer, 284; tumours of the omentum, 285; of the spleen, 284; characteristic vomiting in, 280; diagnostic signs, 280; differential diagnosis regarding origin of the tumour, 283; nature of, 286; perforation of the stomach in, 283; retention of, to gastric ulcer, 280; stagnation of stomach contents in, 282; tumour in, 281; with reference to abscesses of the abdominal wall, gastric sarcoma, myoma, fibroma, 286.
Stomach, catarrh of the, acute, 264; ætiological points, 265; differential diagnosis, 265; from gall-stones, 266; from infectious diseases, 265; from peritonitis, 266; pulse in, 265; stomach contents in, 264; symptoms of, 264; temperature of the body in, 266; chronic, 268; ætiological points, 270; atrophy of the gastric mucous membrane, 271; clinical picture in, 268; diagnostic rules, 270; excessive acid secretion, 269; production of mucus, 268; in gastric cancer and dilatation, 270; in hepatic cirrhosis, 181; in measles, 867; in pulmonary emphysema, 114; in scarlatina, 976; in typhus fever, 898; mucous, 269; reduction of HCl in, 269; retention of food in the stomach in, 269.
changes of form and position of the, 291.
contents, chemical examination of, for albumoses, syntonin and peptones, 257; for free HCl, 261; for lactic acid, 262; in atrophy of the gastric mucous membrane, 272; in cancer of the stomach, 281, 282; in dilatation of the stomach, 287; in gastric catarrh, acute, 264; chronic, 269; ulcer, 276; nervous dyspepsia, 294.
dilatation of the, 287; chronic gastric catarrh in, 290; determination of the capacity of the stomach in, 289; differential diagnosis of, from ascites, 290; mechanical and dynamic, 290; pneumothorax, 164; insufficiency of the stomach in, 290; physical examination in, 288; retention of the stomach contents, 287; vomiting in, 287.
diseases of the, diagnosis of, 255; with the aid of Ewald's test-breakfast, 260; ex-

pression of stomach contents after, 259; examination, chemical, of the stomach contents, 260; of the capability of the stomach wall to resorb in, 258; in leucæmia, 794; lavage of the stomach in, 259; proof of HCl in the stomach in, qualitative, 261; quantitative, 261; sounding, diagnostic, in, 259.
Stomach, disturbances of motility, sensibility, and secretion, 292.
examination of the, in taking the history of a case, vii.
hæmorrhage of the, in hepatic cirrhosis, 181.
hour-glass shape of the, 291.
inflammation of the, interstitial purulent, 266.
juice secretion, diminished, in anæmia, 785.
lavage of the, 259.
mucous membrane of the, anatomical structure and function of the, 255; atrophy of the, 271; consequences of, 272; differentiation of, from amyloid degeneration of, 273; from cancer of the stomach, 273; from nervous achylia, 272; pathogenesis and symptoms, 271; hyperæsthesia of, 299.
neuroses of the, 292; differential diagnosis of, from chronic gastric catarrh, 270; functional disturbances caused by, 293; motor, 304; secretory, 300; sensory, 297.
resonance of the heart sounds in the, and differentiation from pneumopericardium, 48.
spasm of the, 297; of the cardia, 305; of the pylorus, 306; differentiation from gastric ulcer, 278 (see also Gastralgia).
tube, Nélaton's, in affections of the stomach, 259; in dilatation of the stomach, 289.
Stomach, ulcer of the, peptic, 273; absence of pain after complete cicatrization, 275; and cholelithiasis, 279; and duodenal ulcer, 277; and gastric cancer, 277; and neuralgias, 278; causes of, 276; diagnostic symptoms, 273; differential diagnosis, 277; excessive formation of acid in, 276; in enteric fever, 916; pains in, 274; seat of, 275; sensation of oppression in the gastric region, 274; syphilitic and tubercular, 279; vomiting in, 274.
vertical position of the, 292.
wall of the, abnormal tension of the, 291; diffuse purulent infiltration of, 266; examination of the resorbability of, 262; paresis of the, 307; structure of the, 255.
" weak," 307.
Stomatitis, aphthosa, 236.
catarrhal, 236.
mercurial, 236.
ulcerative, 236.
Stones of the bladder, causing cystitis, 397.
and kidneys, differentiation, 390.
sounding of, in the bladder, 397.
Stools, dysenteric, appearance and condition, 929.
rice-water-like, in Asiatic cholera, 934.
typhoid, 913.
Strabismus, in affections of the pons, 609.
in paralysis of the abducens, 466; of the oculomotorius, 465; of the trochlearis, 466.
spastic, in tetany, 749.
Strangulation of the intestine, symptoms of, 333, 334.
Streptococci, ætiological significance of, in acute articular rheumatism, 970; in epidemic cerebro-spinal meningitis, 952; in erysipelas, 891; in septicæmia, 891; in septicopyæmia, 964; in suppurations, 891.
causing peritonitis, 409; causing pneumonia, 122.
in diphtheritic membranes, 242.
in pleuritic exudates, 162.
in the tonsillar membranes of scarlatinal patients, 875.
mixed infection of, in diphtheria, 941; in influenza, 985; in typhoid fever, 916.
Streptococci, transmission of, 895.
Strychnine poisoning, spasms due to, and tetanus, 992.
Subacidity, nervous, of the stomach, in nervous dyspepsia, 294, 296.
Submaxillary and sublingual glands, swelling of the, in mumps, 938.
Subsultus tendinum of typhoid-fever patients, 911.
Succussion sound in pneumopericardium, 47; in pneumothorax, 164.
Sudamina in acute articular rheumatism, 974.
Suffocation, acute, in laryngeal stenosis, 78.
in mediastinal tumours, 146, 147.
in spasm of the glottis, 82.
Sugar in the urine in acute yellow atrophy of the liver, 178; in hepatic cirrhosis, 183; in pancreatic diseases, 222.
reaction of the urine in diabetes mellitus, 824.
resorption by the gastric wall, 258.
Sunstroke, 771.
pathology, 771.
symptoms, 771.
Supersecretion of gastric juice, nervous, 300.
Supraorbital neuralgia, diagnosis of, 451; differential, from migraine and cephalalgia, 453.
due to refrigeration, 451.
in malaria, 451, 981.
Suprascapularis paralysis, 484.
Sweat secretion, centres for, 525, 605.
disturbances of, in acute articular rheumatism, 974; in diabetes, 829; in hysteria, 730; in syringomyelia, 572.
increased, after epileptic attacks, 740; in Graves's disease, 761; in malaria, 979; in myositis, 767; in tetanus, 991; in typhoid fever, 911.
Sympathetic nerve, paralysis of the cervical part, in capsular disease of the brain, 647.
symptoms in Klumpke's paralysis, 492.
Syncope in anæmia, 783.
Syphilis causing brain softening, 680; brain tumours, 690; chronic anterior poliomyelitis, 562; chronic spinal meningitis, 534; spastic paralysis, 554; tabes dorsalis, 546.
neuralgic pains due to, 450, 461.
of the bronchi and trachea, 98; of the larynx, 75, 76; of the liver, 187; of the lungs, 145; of the mouth, 236; of the palate and pharynx, 244.
varicella-like eruption in, 890.
Syphilomata of the brain, 710.
of the spinal cord, 584.
of the spleen, 232.
Syringomyelia, 571.
differential diagnosis of, 573; from acromegaly, 757; from amyotrophic lateral sclerosis, 557; from polyneuritis, 511; from progressive muscular atrophy, 567; from spinal-cord tumours, 573.
pathological findings in, 571.
symptoms of, bulbar, 572; motor, 572; sensory, 571; vaso-motor, 572.

T

Tabes dorsalis, 537.
ætiological points for diagnosis, 546.
Brach-Romberg's symptom of, 539.
complications of, motor, 544; sensory, 545.
condition of the reflexes in, 537.
co-ordination disturbances of muscular action in, 539; cause of, 540.
crises, 538.
degenerative processes in the spinal cord in, 542.
diagnosis of, in the first stage, 537; in the second stage, 539; in the third stage, 539; differential, 546; from arthropathies of the vertebræ, 547; from Friedreich's ataxia, 549; from multiple neuritis, 548; sclerosis, 548; from neuralgia, 546; from

neurasthenia spinalis, 547; from rheumatic affections, 546.
Tabes, eyes, condition of, in, 537.
initial symptoms in, 538; occurrence of, 542.
neuritic symptoms of, 540.
paralytic symptoms of, 539.
rarer symptoms of, 544.
sciatic pains at the onset, 546.
Tachycardia in Graves's disease, 759.
in influenza, 986.
Tactile sense, centre for, 642, 649.
conduction tracts of, in the spinal cord, 522.
disturbances of, 442; in affections of the posterior horns of the cord, 526; in posterior poliomyelitis, 570; in syringomyelia, 571; in tabes dorsalis, 545; in unilateral lesion of the spinal cord, 582; of the tongue in chordal paralysis, 474; terminal apparatus of, 435.
Tangential fibres of the cortex of the brain, 638.
Taste, anæsthesia of, diagnosis of, 438.
hyperæsthesia of, occurrence of, 445.
perception of, examination for, 440; in facialis paralyses, 469, 471, 473; in tabes dorsalis, 545; of the tongue, 478; nerve tract of, 438; within the Fallopian canal, 478; without the Fallopian canal, 478.
Tears, secretion of, in facialis paralysis, 469, 471.
in Graves's disease, 761.
in hysteria, 730.
in trigeminus neuralgia, 451.
Teeth, anomalies of, of rhachitic children, 853.
caries, cause of trigeminus neuralgia, 452; in diabetics, 830.
Temperature curve in enteric fever, 908; in malaria, 978; in measles, 863; in relapsing fever, 902; in scarlatina, 872; in typhus fever, 898; in variola, 884.
of the body after epileptic attacks, 740; in anæmia, 785; in apoplectic insult, 664; in embolic insult, 677; in gastric catarrh, 265; in meningeal hæmorrhages, 719; in meningitis, 703; in peritonitis, 410; in pharyngeal diphtheria, 942; in rabies, 994; in rötheln, 879; in tetanus, 940; in tetany, 750; in unilateral lesion of the spinal cord, 582.
sense, cortical centre of, 643; disturbances of, in affections of the posterior horns of the cord, 526; in posterior poliomyelitis, 570; in syringomyelia, 570, 573; peripheral terminal apparatus of, 435; tracts of, 521.
Temporal cortex, foci of, symptoms of value in diagnosis, 651, 660, 693.
reflexes, diagnostic significance of, 429; in amyotrophic lateral sclerosis, 555; in anæsthesia, peripheral, 442; in apoplectic attack, 664; in Brown-Séquard's paralysis, 582; in capsular affections, 647; in compression of the spinal cord, 580; in degeneration of the motor conduction tract, 552; in diabetes mellitus, 831; in dystrophia muscularis, progressive, 569; in facialis paralysis (central), 475; in hysteric paralysis, 725; in multiple sclerosis of the spinal cord, 590; in poliomyelitis anterior, 558; chronic progressive, 565; in polyneuritis, 506; in progressive paralysis, 697; in spinal meningitis, 530; in tabes dorsalis, 537; in traumatic neurosis, 735.
sheaths, inflammations, rheumatic, 974.
Tenesmus in dysentery, 929.
in proctitis, 315.
in rectal ulcers, 321.
Teres major, paralysis of, 484.
Testicles, atrophy of, following orchitis parotidea (infectiosa), 939.
Tetanus, 988.
bacilli, diagnostic importance of, 989; importation of, into the body, 989; morphology of, 989.
Tetanus, cryptogenetic (idiopathic), 989.
diagnostic value of the symptoms of, 990.
differential diagnosis of, from hydrophobia, 993, 994; from hysteric spasms, 992; from masticatory spasm, 992; from meningitis, spinal and cerebro-spinal, 991; from muscular rheumatism, 992; from strychnine poisoning, 992.
due to vaccination, 9.
immunization against, 989.
local, 989.
mode of infection in, 989.
morbid picture of, 990.
neonatorum, 989.
of the head, differential diagnosis, 991.
puerperal, 989.
rheumatic, 989.
traumatic, 989.
Tetany, 748.
ætiological factors of, 750.
and myxœdema, 758.
Chvostek's sign of, 749.
conduct of the nerves in, 749.
differential diagnosis of, from arthrogryphosis infantum, hysteria and tetanus, 750.
epidemic occurrence of, 750.
in gastrectasis, 288.
of rhachitic children, 854.
secondary phenomena in, 749.
spastic condition in, 749.
Trousseau's sign of, 749.
Thomsen's disease, 753.
myotonic reaction (Erb) in, 753.
Thoracic glands, neuralgia of, 456.
Thoracicus longus, paralysis of, 485.
Thorax, barrel shape, in emphysema, 112.
deformities of, rhachitic, 853.
examination of, vii.
in mediastinal tumours, 147.
in pneumonia, fibrinous, 123.
phthisical, in tuberculosis, 134.
systolic retraction of apex of heart, 45.
Thought, capability of, central station of, 651.
Thrill, præsystolic, in mitral stenosis, 16.
systolic in aortic aneurysm, 64; in aortic stenosis, 27; persistence of aortic isthmus, 38; pulmonary stenosis, 29.
Thrombus formation in right heart and veins cause of embolism in pulmonary artery, 139.
Thumb, musculature of the, atrophy of, in paralysis of the median nerve, 491; in poliomyelitis anterior, chronic progressive, 564; in syringomyelia, 572.
paralysis of the, 489, 490, 491.
Thyreoarytænoideus internus, functions of, 86.
Thyreoid gland, the, and exophthalmic goitre, 759, 763; and myxœdema, 758.
Thyreoiditis in acute articular rheumatism, 974.
Tibialis paralysis, 495.
Tic convulsif, 496, 735, 748.
general, 748.
rotatoric, 499.
Tissue shreds in the sputum in pulmonary abscess, 142; in pulmonary gangrene, 144.
stools in chronic intestinal catarrh, 317; in intestinal ulcer, 319.
urine in nephrophthisis, 374.
Toes, position of, in hereditary ataxia, 550.
in paralysis of the peroneus, 494; tibialis, 495.
Tongue, atrophy of, in hemiatrophia progressive facialis, 755; hypoglossus paralysis, 481; in progressive bulbar paralysis, 612; muscular atrophy, 566; oblongata disease, 608.
coating of, unilateral neuralgia of the lingualis, 425; in dysentery, 930; in erysipelas, 894; in measles, 864; in scarlatina, 874; in typhoid fever, 911; in typhus, 897.
deviations of, upon putting forth, in unilateral paralysis of the hypoglossal nerve, 481, 645.

Tongue, diagnostic utilization of the coat of the, 235.
disturbances of taste of, 439, 440, 473.
musculature of fibrillary twitchings, in bulbar paralysis, 612; myositis in, 767.
paralysis of, in pons-medulla affections, 608, 612.
spasms of, diagnosis and symptoms, 498; in tetanus, 990.
the, in diabetics, 830; in enteric fever, 911; in relapsing fever, 904; in scarlatina, 874.
tubercular ulcers of the, 237.
Tormina intestinorum, 340.
Torticollis, 497.
rheumatic, 765.
Toxines, ætiological significance in infectious diseases, 858; immunity of organism to bacterial, 861; in cholera, 935; in Landry's paralysis, 588.
effect of, upon the body, 858; in anthrax, 995; in diphtheria, 941; in tetanus, 989.
nature of, 858.
Trachea, compression of, by mediastinal tumours, 98.
diseases of, 90.
Oliver's symptom in aneurysm of aorta, 65.
Tracheostenosis, diagnosis of, 77.
causes of, 99.
due to foreign bodies, 99.
in tracheotomy, 99.
Tractions diverticulum, 252.
Transfer symptoms in hysteria, 729.
Transudates and exudates of the thoracic cavity, differential diagnosis of, 161.
Trapezius muscle, paralysis, 480; bilateral, 480.
spasm of, 498.
Trauma causing abscess of the brain, 695.
functional disturbances of the nervous system, 734.
meningeal spasms, 718.
neuralgias, 449.
Tremor, and athetosis, 747.
epileptic, 740.
hysteric, 726.
in cerebellar tumours, 624.
in dementia paralytica, 697.
in Graves's disease, 760.
in multiple sclerosis of the cord, 589.
in paralysis agitans, 751; varieties of, 752, 753.
in traumatic neuroses, 734.
in writer's cramp, 501.
Trichinosis, 999.
blood condition in, 1000.
diagnosis, 999; differential, from cholera nostras, 1000; from poliomyelitis, 1000.
microscopic findings of the fæces and muscles, 1000.
mode of infection, 999.
pathogenesis of, 999.
symptoms of, 999.
Tricuspid insufficiency, 30; combination with aortic insufficiency, 31; differential diagnosis, 32; in myocarditis, 50; in pulmonary emphysema, 113; symptoms, 30; in venous pulse, 31, 32; relative, 32; stenosis, 33; congenital, 33; symptoms, 33.
Trigeminus, anæsthesia in the region of, 441; in tabes dorsalis, 545.
conduction of taste perception by, 438, 439, 441.
neuralgia of, 451; in the first branch, 451; in the second branch, 452; in the third branch, 452; differential diagnosis of, 453; in sinus thrombosis, 717; in tabes dorsalis, 539, 545.
paræsthesia of, in tabes, 545.
paralysis of the motor portions of, 467; alternating in pontine disease, 607, 608; seat of, 467.
Trismus, diagnostic criteria of, 496; in meningitis, 704; in tetanus, 991; in trichinosis, 999.
due to invasion of tetanus bacilli, 989.
Trochlearis, nucleus of the, in the middle brain, 630.
paralysis of, 466; double picture in, 466; eye movements in, 466; in tabes dorsalis, 537; nuclear, 616; seat of the cause of paralysis of, 467.
Trophic disturbances after apoplectic insult, 668.
in anæmics, 782; in compression myelitis, 580; in medianus paralysis, 491; in meningitis, 704; in multiple sclerosis, 591; in myelitis, 573, 574, 575; in neuralgias, 447; in neuritis, 508; in syringomyelia, 572; in tabes dorsalis, 545; in tetany, 749.
origin of, 525.
Trophoneuroses, 754.
Tropical fever, 984.
parasites of, 977.
Trunk, musculature of the, paralysis after apoplectic attacks, 667.
Tubercle bacilli, differentiation from leprosy bacilli and smegma bacilli, 130.
in pleuritic exudates, 162.
in pulmonary tuberculosis, 130.
in sputum in laryngeal tuberculosis, 75.
in the blood and urine in acute miliary tuberculosis, 957.
in tuberculosis of the palate and pharynx, 237.
methods of staining, 130.
proof of, in urine in nephrophthisis, 375.
Tubercles in the spinal-cord canal, 584.
of the brain, occurrence of, in childhood, and their diagnostic significance, 690.
Tuberculin, use for diagnostic purposes, 131, 418.
Tuberculosis, general, diffuse, 955 (see also Acute Miliary Tuberculosis).
giving rise to meningitis, 705, 707, 709, 969; spinal, 531.
local, 569; of the brain (solitary and multiple tubercles), 690; of the lungs after measles, 867; of the vertebral column, 854.
of the bladder, 398; of the intestines, 323; of the kidney, 374; of the larynx, 74, 75; of the lungs, 130; chronic (see Lungs); of the palate and pharynx, 244; of the peritonæum, 417; of the spleen, 232; of the stomach, 279; of the tongue and oral cavity, 237; of the vertebræ (see Spondylitis).
pulmonary, after measles, 867; after pertussis, 949; after typhoid fever, 922; in diabetes, 829.
Tugging, tracheal, in aortic aneurysm, 65.
Tumour, in carcinoma of the intestine, 326; of the stomach, 281.
in perityphlitis, 312.
in stenosis of the intestine, 335.
of the brain, 680; in the central cranial fossa, 685; in the posterior cranial fossa, 685; of the lobes of the brain, 686.
of the spinal cord and its meninges, 582, 583.
pulsating, in aortic aneurysm, 64.
Twitchings, epileptic, in cortical lesions of the central convolution, 650, 671.
Typhlitis, 311.
and intestinal stenosis, 332.
constipation in, 312.
differential diagnosis, 313; from cæcal tumours, 314.
examination of, 314.
fever in, 312.
stercoralis, 312.
ulcer formation in, 312.
Typhoid bacilli, distribution outside of the body and transmission, 906.
effect of, in the intestinal canal of infected individuals, 906, 907.
vomiting in, 312.
Typhoid bacilli in pleuritic exudates, 162.
bilious, differential diagnosis from Weil's disease, 213.
fever, 906; abortive forms of, 919; abscess formation due to, 916; ætiology of, 906;

afebrile, 919; ambulatory, 919; causing spinal meningitis, 531; complications of, 914, 915, 916, 917, 918; on the part of the circulatory organs, 917; of the digestive organs, 915, 916; of the nervous system; 918; of the respiratory organs, 917; of the skin, muscles, bones, urinary, and sexual organs, 918; diagnosis of, 906, 919, 920; differential, 922; from acute miliary tuberculosis, 922, 959, 960; from anthrax internus, 925; from central pneumonia, 925; from cryptogenetic septicopyæmia, 924; from gastric fever, 926; from influenza, 988; from measles, 925; from meningitis, 926; from relapsing fever, 924; from scarlatina, 925; from small-pox, 925; from typhus fever, 924; with the Gruber-Widal reaction, 920; duration of, 908; eruption of, 912; incubation period, 908; infection, 907; course of, 907; influence of the state of ground-water upon infection with, 907; jaundice in, 917; meningitis in, purulent, 918; mixed infection, 916; morbid picture of, first and second weeks, 911; third week, 912; prodromal stage of, 908; pulse in, 910; radialis paralysis due to, 487; relapses of, 919; secondary suppuration in, 916; status typhosus, 911; stupidus, 911; symptoms of, 908, 909, 910, 911, 912, 913; cardinal, 921; varieties of, 918; versatilis, 911.

Typhus fever, 896.
complications of, 898.
contagiosity of, 896.
diagnosis of, 899; differential, from enteric fever, 899, 924; from measles, 899.
eruption of, 897.
eruption and florition stage of, 897.
incubation period, 897.
nature of the contagious principle, 896.
precritical fall, 898.
prodromes, 897.
status typhosus, 897.
symptoms of, 897, 898.
temperature curve in, 898.

Tyrosin in urine in acute yellow atrophy of the liver, 177.

U

Ulcer, gastric, 273; differentiation from intercostal neuralgia, 278.
intestinal, 319; infectious, 323; luetic and tuberculosis of the wall of the stomach, 279; peptic, 322; tuberculosis, 323.
of the larynx, 74.
of the œsophagus, 245.
of the palate and pharynx, 244.
peptic, of stomach, 273; and gall-stone colic, 216; and pleurisy, 158; complicated with gastric catarrh, 270.
perforating, of the foot, 546.
tuberculous, of the larynx, 74, 75; of the oral cavity, 237; of the pharynx, 244.

Ulnaris paralysis, 489.
causes of, 489.
paralysis of the cutaneous sensory branches of, 491.

Undulant fever, 1004.

Uræmia, chronic, 354, 358, 359, 363.
diagnosis, 359; differential, from cerebral affections, 360; from infectious diseases, 360.
in acute nephritis, 354; in amyloid kidney, 366; in chronic nephritis, 358; in contracted kidney, 363; in origin of, 259; in psychical disturbances, 363.

Uræmia, differential diagnosis of, from acute miliary tuberculosis, 959.
from coma diabeticum, 833; due to cerebral hæmorrhage, 674.
from epileptic attacks, 743.
from meningitis, 711.
from septicopyæmia, 969.

Urates, marked excretion of, in gastric catarrh, 265.

Urates, in affections of the liver, 172, 177, 183.
in pneumonia, 124.

Urea, excretion in anæmic conditions, 777, 786.
in Asiatic cholera, 933; in contracted kidney, 362; in diabetes mellitus, 827; in leucæmia, 795; in nephritis, 352, 357.

Ureters, concrements in the, 388.

Urethral crises in tabes dorsalis, 538.

Urethritis rheumatica, 975.

Uric-acid excretion in affections of the liver, 173.
in contracted kidney, 362.
in diabetes mellitus, 827.
in gout, 845.
in leucæmia, 795.
in nephritis, acute, 352; chronic, 362.
in stones in the bladder, 397; in the kidneys and ureters, 389, 390.

Urina spastica in angina pectoris, 57.
in gastralgia, 298.
in hysteria, 730.

Urinary bladder, affections, 393; nervous, 400; mucous membrane of, 403; anæsthesia of, 404; hyperæsthesia, 403; as cause of spasm of the bladder, 403; musculature of, 401; paralysis, 401; spasm, 402; tuberculosis of, 398; due to involvement of the bladder in the tuberculous process, and inflammation of the urinary passages, 398; origin of, 398; veins of the, 400; varicose dilatation of, 400.
casts in the urine in contracted kidney, 362; in engorged kidney, 348; in glomerulo-nephritis, 356; in jaundice, 208; in nephritis, acute, 352; in nephrolithiasis, 388; in pyelonephritis, 367.
organs, affections of, 346; condition of the urine in, 346, 347.
sediment, in nephritis, acute, 352; chronic, 357; in renal tuberculosis, 374.

Urinate, desire to, in cystoplegia, 401.
in nephritis, acute, 353.
in nephrolithiasis, 388.

Urine, condition of, after apoplectic stroke, 665, 666; in acute articular rheumatism, 974; in acute miliary tuberculosis, 967; in adipositas universalis, 841; in anæmic conditions, 786; in chorea, 745; in cholera Asiatica, 934; in diabetes insipidus, 837; in diabetes mellitus, 823, 824, 825, 826, 827; in diphtheria, 944; in dysentery, 929; in exophthalmic goitre, 761; in gouty attacks, 845, 847; in hæmoglobinuria, 812; in hydrophobia, 993; in influenza, 986; in leucæmia, 795; in malaria, 980; in meningitis, 704; in osteomalacia, 851; in relapsing fever, 904; in rhachitis, 853; in scarlatina, 874; in tetanus, 991; in trichinosis, 999; in typhoid fever, 914, 918.
discharge of, in hydronephrosis, 381; in peritonitis, 408; in spasm of the bladder, 402, 403; of the stomach, 297; involuntary, in floating kidney, 393; in paralysis of the musculature of the bladder, 401.
dripping of, 401.
fermentation, ammoniacal-alkaline, in cystitis, 394, 395.
in albuminuria, 346; in amyloid spleen, 196; in aortic insufficiency, 25; in cancer of the bladder, 399; in concrements, 389; examination of, 389; in contracted kidney, 362; in cystitis, 395; in engorged kidney, 348; in fatty kidney, 364; in fibrinous pneumonia, 124; in gastric catarrh, 265; in intestinal catarrh, 308; stenosis, 331; in jaundice, 208; in liver atrophy, 177; carcinoma, 198; cirrhosis, 183; in myocarditis, 49; in nephritis, acute, 351; chronic, 357, 361, 362; suppurative, 367; in nephrolithiasis, 388; in nephrophthisis, 374; in nervous dyspepsia, 296; in pancreatic disease, 222; in pleurisy, 157; in pulmonary tuberculosis, 139; in pyuria, 347; in renal echinococcus, 384; infarct, 374; tumours, 379, 381, 386; in uræmic intoxication, 359; in Weil's disease, 213.

Urine reaction in jaundice, 208; in nephritis, chronic, 357, 361, 362; suppurative, 367; in nephrolithiasis, 374; in pulmonary tuberculosis, 139; in tuberculosis of the bladder, 399.
retention of, 393; causing cystitis, 396.
secretion of, centre for the, in the brain, 643; in the spinal cord, 524; in Asiatic cholera, 934; in diabetes insipidus, 837; in diabetes mellitus, 825; in hysteria, 730; in malaria, 980; in meningitis, 704; acute cerebro-spinal, 951; spinal, 530; in myelitis, 576; in spinal-cord lesion, 526, 527; in tetanus, 991; in unilateral lesion of the spinal cord, 582.
segregator in renal tuberculosis, 376.
Urobilin icterus, 174.
origin of, 174.
Urogenital tuberculosis, 374.
implication of bladder in, 398.
miliary tuberculosis, 957.
Urticaria in cryptogenetic septicopyæmia, 966.
in influenza, 987.
in neuralgia, 447, 454.
in neuritis, 508.
in paroxysmal hæmoglobinuria, 814.
Uvula, oblique position of, in facialis paralysis, 468, 473.

V

Vagus, compression of, by aortic aneurysms, and symptoms, 65.
action of, upon gastric-juice products, 258.
by mediastinal tumours, 149.
lesions of, as cause of asthmatic attacks, 106.
neuritic affections of, 506.
nucleus and central distribution, 599.
paralytic symptoms, 479.
Valve in pneumothorax, 166.
organically closed and mechanically closed, 167.
symptoms, 167.
Valvular defects of the heart, 11.
combined, 33.
of the right heart, 28.
Varicella, 889.
and variola, 889.
character of, 889.
differential diagnosis of, from artificial exanthems, 890, 891; from herpes, 890; from miliaria vesicles, 890; from pemphigus, 890; from varioloid, 890.
eruption of, 889; successive crops of, 889.
incubation period, 889.
infection in, 889.
of the mucous membrane, 890.
symptoms of, 889, 890.
Varices of the bladder, 400.
Variola, 880.
Varioloid, 885.
confluens, 885; development of the eruption, 885; morbid picture of, 885.
non-febrilis, 885.
pemphigosa, 885.
pustula hæmorrhagica, 886; morbid picture, 886.
sine exanthemata, 885; diagnosis, 885.
varieties, 885, 886.
vera, 885.
verrucosa, 885.
Vascular murmurs in mediastinal tumours, 149.
Vascular nerve centres in the spinal cord, anatomical position of, 525.
Vaso-motor disturbances after apoplectic stroke, 665.
in acromegaly, 757; in affections of the cerebral peduncles, 630, 631; in anæmic conditions, 782; in capsular affections, 647; in cerebral anæmias, 700; in disease of the posterior horns of the cord, 526; in facialis paralysis, 471, 478; in lesion of the central convolutions, 650; in meningitis, 704; in myelitis, 575; in neuralgia, 447; of the trigeminus, 451; in neurasthenia, 732; in neuritis, 504; multiple, 508; in poliomyelitis posterior, 570; in spinal progressive muscular atrophy, 566; in syringomyelia, 572; in tabes dorsalis, 545; in unilateral lesion of the cord, 582; origin of, 525.
Vaso-motors, centres for, 525, 605, 700.
toxic paralysis of, in enteric fever, 918.
Veins of the throat, collapse of the, in pericardial synechiæ, 47.
in inspiratory swelling of, in mediastinal pericarditis, 47.
in pulsation of, in heart diseases, 2; in pulmonary emphysema, 113.
Veins, pressure in, in cardiac diseases, 2.
Velum medullare anticum and posticum, 597.
Venous compression in aortic aneurysm, 66.
in mediastinal tumours, 149.
Venous murmurs in anæmic conditions, 784.
thrombosis and phlebitis in gout, 847; in multiple myositis, 767.
Venous murmurs in hepatic region, from dilatation of the portal vein, 182.
Venous pulse in cardiac disease, 2.
diastolic-præsystolic, 2.
in tricuspid stenosis, 33.
Ventricle of the heart, atrophy of (concentric of the left in pure mitral stenosis), 16.
dilatation and hypertrophy of the left, 16; in aortic insufficiency, 20; in nephritis, 363; in persistence of the aortic isthmus, 38.
dilatation and hypertrophy of the right, 16; in bronchiostenosis, 96; in congenital aortic stenosis, 28; in emphysema, 113; in interstitial pneumonia, 129; in mitral stenosis, 18; in pulmonary atelectasis, 107; in pulmonary insufficiency, 29; stenosis, 29; in tricuspid stenosis, 33; in valvular disease of the right, 28.
Ventricles of the brain, anlage of the third, 634.
blood extravasations into the same, and symptoms, 664.
dropsy of, and its symptoms, 719, 720; in cerebellar tumours, 625.
Ventricular sæptum of the heart, defects of, 37.
Vertebræ, caries and cancer of the, causing pressure paralysis of the cord, 579, 581.
Vertebral column, curvature of, the cause of pulmonary atelectasis, 107; stenosis of the œsophagus, 249.
Vertebral column, curvatures of, due to rhachitis, 852, 853.
due to sciatica, 461.
due to spinal-cord compression, 579.
in hereditary ataxia, 550.
painfulness and stiffness of, in compression myelitis, 579; in hysterics, 728; in spinal meningitis, 530, 533.
Vertical position of the stomach, 292.
Vertigo, cerebellar, 622.
epileptic, 740.
in intestinal disease, 344.
of tabetics, 546.
of traumatic neurosis, 735.
Vesical crises in tabes, 538.
Vessels, large, malformations, 36.
disease of, 62.
Villous cancer of the urinary bladder, 399.
Visceral crises in multiple neuritis and tabes dorsalis, 538.
Vision, disturbances of, in central facialis paralysis, 475.
in foci of the corpora quadrigemina, 633; of the occipital cortex, 649; in hæmorrhages of the brain, 668; in lesion of the optic thalami, 644; in tabes dorsalis, 537, 538; in traumatic neurosis, 735; in tumour of the central cranial fossa, 685; in unilateral, in abscess of the brain, 693.

Vision, field of, narrowing of the, concentric, in hysteria, 729; in traumatic neurosis, 735.
Vocal cord, paralysis of, hysteric, 725.
Vocal cords, "cadaveric position," 84.
membranous adhesions of, 77.
tuberculous ulcers of, 75.
Vocal cords, paralysis of, 83; bilateral, 84.
cause of, 84.
due to disturbances of laryngeal, inferior (recurrent), 85.
due to disturbances of laryngeal, superior (recurrent), 84.
in aneurysm of aorta, 65.
in mediastinal tumours, 149.
in pericarditis, 41.
in pulmonary tuberculosis, 135.
unilateral, 84.
Vola manus, anæsthesia of, 490.
Volume, diminution of liver in acute yellow atrophy, 178.
in cirrhosis, 180.
in enlargement of, in prolonged jaundice, 209.
Vomiting in cerebellar diseases, 624.
in cholera Asiatica, 634.
Vomiting, in gastrectasis, 287.
in gastric cancer, 280; ulcer, 274; in intestinal stenosis, 331; in nephritic acuta, 354; chronica, 359; in nervous, 297, 302, 306; in pericarditis, 41; in peritonitis, 408; in perityphlitis, 312; in Weil's disease, 213.
Vox cholerica, 931.

W

Wandering kidney, 391; and carcinoma of the intestine, 327; and corset-lobe liver, 392; and intestinal stenosis, 335; and tumours of gall-bladder, 392; and pyloric tumours, 392; and wandering spleen, 392.
differential diagnosis, 392.
hydronephrosis in torsion, 382.
incarceration symptoms, 393.
Wandering liver, 206.
Wandering spleen, 233; and carcinoma of intestine, 233; and movable pyloric tumour and fæcal tumours in transverse colon, 234; and thickened corset lobe, 206; and wandering kidney, 392.
Warmth, perception of, centre of, in the brain, 525.
conduction of, in the spinal cord, 521, 525, 571.
disturbance of, in affections of the posterior horns of the cord, 526; in Graves's disease, 761; in paralysis agitans, 752.
terminal organs of, in the skin, 435.
Water retention in nephritis, 353.
Water-wheel murmur in pneumopericardium, 47.
Water-whistle murmur in open pneumothorax, 165.
Weil's disease, 213.
ætiology, 214.
clinical picture, 213.
differential diagnosis, 213.
hæmorrhage of the skin in, 213.
Whooping-cough, 946.
character of, 947.
complications of, 948.
contagiosity of, 947.
diagnosis of, 946; differential, 949.
following measles, 867.
paroxysms of cough in, 946; cause, 947; subsequent condition of, 948.
sequelæ, 949.
symptoms of, in the catarrhal stage, 946; in the cortical stage, 949; in the convulsive stage, 946.
Will, paralysis of, in functional brain disease, 723.
in hysteria, 723.
Williams's tracheal sound in pleurisy, 156.
in pneumonic infiltrated lungs, 123.
Wintrich's sign over pulmonary cavities, 136.
Word deafness, 651.
sounds, central station of, 651.
Worm, injuries of the, in the cerebellum, 623.
Writer's cramp, 501.
electric irritability of the muscles in, 502.
Writing, analysis of, 656.
diagrammatic description of the process of, 657.
disturbances of, in aphasia, 656, 657; in progressive paralysis, 697.

X

Xanthopsi in jaundice, 208.
X-rays in aortic aneurysm, 67.
X-rays in cancer of the bowel, 330.
in nephrolithiasis, 389.
X-rays in renal calculi, 389.

Y

Yawning, spasmodic, origin of, 501.
Yellow fever, 1001.
ætiology of, 1001.
clinical picture of, 1001.
diagnosis of, 1002.
mode of transmission of, 1001.
pathology of, 1001.
types of the disease of, 1002.

THE END

LANE MEDICAL LIBRARY

To avoid fine, this book should be returned on
or before the date last stamped below.

FEB 3 '21

L73 Leube, W.O.v. 44048
L652s Medical diagnosis: Spe-
1904 cial diagnosis of inter-
-nal medicine; fr. the 6th
German ed. Ed. by Salin-
ger
E. McHall
FEB 3 '27